VOL. 6 NO. 1 JANUARY-MARCH 2001 QUARTERLY

JOURNAL OF SURGERY PAKISTAN INTERNATIONAL

Approved by the Pakistan Medical & Dental Council & Index Medicus for the Eastern Mediterranean Region

Published by: Prof.Abdul Aziz for Prof.F.U.Baqai, Baqai Postgraduate Medical Institute, IIIC, 1/12, Nazimabad, Karachi
Address for correspondence: Prof. Abdul Aziz, Editor JSP, National Institute of Child Health Karachi OR C/o Treasurer, College of Physicians and Surgeons Pakistan, 7th Central Street, DHA, Karachi-75500
Subscription rates: Per copy-in Pakistan Rs.100/= in SAARC countries U.S. $20, in other countries U.S.$30, Annual- in Pakistan Rs.300/= in SARRC countries US $ 60, in other countries U.S.$90.
Layout by : Aleemuddin Siddiqui.

EDITOR'S NOTE

Medical Information on Websites: How Reliabile it is?	Jamshed Akhtar	1

ARTICLES

Inguinal Hernia Repair Under Local Anaesthesia	G.M. Khan Baluch	2
Management of Laryngeal Cancer	Tariq Rafi	4
Breech Presentation (An Experience with 137 Cases)	Naila Ehsan	7
Amoebic Liver Abscess: Aspiration Versus Conservative Treatment	Suhail Ahmed Almani	11
Vesicoureteric Reflux in Children	Noor Ahmad Channa	15
An Experience with Primary Closure in Pilonidal Sinus	Anis Subhan	17
Coagulation Disorders in Patients with Carcinoma of the Hepatobiliary and Pancreatic System	Aisha Y. Malik	19
Pattern of Disease and Treatment of Allergic Rhinitis in Karachi	Ehsan-ul-Haq	24
Can Diagnosis of Acute Appendicitis be Simplified?	Tariq Mahmood Rehan	27
Ileal Typhoid Perforation - An Experience with 42 Cases	Mohammad Haleem Taj	30
Anxiety as a Psychological Risk Factor in the Disorders of Cardiovascular System	Seema Munaf	33
A Validation Study of Besson Score for Differential Diagnosis of Stroke Subtypes	Saadat H. Zaidi	38

CASE REPORTS

Haemoperitoneum by Taenia Infestation	Nizam Ud Din Danwer	41
Obturator Hernia	Kulsoom Junejo	42

Editorial :

MEDICAL INFORMATION ON WEBSITES: HOW RELIABLE IT IS?

The internet has revolutionized the learning environment in the field of medicine. This mode of delivering knowledge is becoming increasingly popular. It provides an access to latest advancement in the field of medicine at one's own desktop and at the same time provides unparalleled communication between the colleagues via email. With increasing access to web sites related to health there is growing concern how reliable these sites are in imparting correct information to end users. In a study by Chen LE on the topic of paediatric surgery on internet it was concluded that quality of information related to the field varied according to the owner of the site. The sites owned by lay people contained information that was incomplete and did not reflect the opinion of most of the paediatric surgeons[1]. This observation needs serious consideration because of its possible grave impact on the public.

The present format of internet is such that there is no legal frame work as to the quality of information provided. To monitor quality of medical information a Code of Conduct for medical websites is used by the Health on Net Foundation. The logo of this foundation is displayed on the websites that register with them. The foundation reviews the sites on regular basis to assess the quality of information[2].

The internet at present is in infancy in our country and it is expected that there is no immediate threat to the health of lay people but with advancement in information technology the day is not far when many would have an access to this facility and then it would become a real issue. There is growing need in this regard to inform people as to which of the sites are safe and reliable. In this regard Ministry of Health and private hospitals can do a lot by developing their own websites for providing information on various ailments and facilities available at their institutes in dealing with them. In addition On Net facility can be provided by specialists on hospital panel list. Use of Urdu version would be great help in this regard.

References

1. Chen LE, Minkes RK, Lanager JC. Pediatric surgery on the internet: Is the truth out there? J Pediatr Surg 2000 35, 1179-82.

2. http://www.hon.ch/Conduct.html: HON Code of Conduct for Medical and Health Websites.

JAMSHED AKHTAR

INGUINAL HERNIA REPAIR
UNDER LOCAL ANAESTHESIA

G.M. KHAN BALUCH

ABSTRACT:
Inguinal hernia is a common surgical problem throughout the world and is seen at all ages and in both sexes. Surgery is the only successful form of treatment. Hernia repair under general or spinal anaesthesia is not free from complications and needs long hospital stay. This study was conducted to evaluate inguinal hernia repair under local anaesthesia regarding intraoperative analgesia, post operative ambulation, cost, hospital stay and complications. Forty patients with inguinal hernias, 20 to 70 years of age were operated under 0.5% xylocaine with 1:200000 adrenaline infiltration. In all cases repair by darning was done. Adequate analgesia was obtained during operation and early post-operative period. Average hospital stay was 40 hours and average cost of the operation was rupees one thousand only (ours being a government hospital). Three cases developed wound infection and one haematoma. It is concluded that inguinal hernia repair under local anaesthesia is safe and economical.

KEY WORDS: Inguinal Hernia Repair, Local Anaesthesia

INTRODUCTION

Hernia is a common surgical problem throughout the world and is seen at all ages and in both the sexes.[1] This accounts for about 10-15% of all surgical procedures and majority of hernia repair operations (80%) are performed for inguinal hernia, though this figure is even higher in male population.[2] Surgery is the only successful mode of treatment. If hernia repair is performed under general anaesthesia, it entails in prolonged convalescence and extended hospital stay, which is associated with complication of general anesthesia. Cost is another prohibiting factor. With spinal anaesthesia also, convalescence is prolonged and headache is troublesome in some patients. There is a risk of meningitis as well. Repair of inguinal hernia using local anaesthesia is becoming increasingly popular as it avoids many of the systemic side effects associated with general anaesthesia, spinal anaesthesia and epidural anesthesia. It provides excellent and early post operation pain relief.[3]

PATIENTS AND METHODS

This prospective study on the inguinal hernia repair under local anaesthesia was conducted in surgical unit III of Nishtar Hospital Multan over a period of two years from January 1998 to December 1999. Forty patients with inguinal hernia were managed by repair under local anaesthesia. Only those patients were included in the study who had funicular and bubonocele type of hernia. Patients with bladder outlet obstruction were excluded and those with chronic cough were first treated for the chest problem. No discrimination was made between direct and indirect inguinal hernia. Age range was 20 to 70 years and all the patients were male. Xylocain 0.5% with adrenaline 1:200000, maximum dose being 7 mg/kg body weight was used. A 10 ml of local anaesthetic solution was injected by a series of intracutaneous injections along the proposed line of incision. Subcutaneous infiltration was carried out next and a total of 10-15 ml of solution was injected. The ilioinguinal and iliohypogastric nerves were infiltrated with 3 ml of solution each. After analgesia, incision was made in the skin, down to the aponeurosis of external oblique. 10 ml of solution was infiltrated in the external oblique aponeurosis, along the line of incision. Five to ten ml of local anaesthetic solution was injected at the level of the deep inguinal ring to block the genito-femoral nerve and sensory filaments of spermatic plexus. Posterior wall draining was carried out in all the patients with proline No 1. Wound was closed in layers after proper hemostasis.

RESULTS

Forty patients were operated by this method and all of

Correspondence:
Dr. G .M. Khan Baluch, Asstt. Prof. Surgical Unit III,
Nishtar Medical College & Hospital, Multan.

them were male with age distribution shown in Table - I.

TABLE-I AGE DISTRIBUTION

S.NO.	AGE	NO. OF PATIENTS
1	20 - 30	15
2	31 - 40	10
3	41 - 50	7
4	51 - 60	3
5	61 - 70	5

Thirty (75%) hernias were on the right side, eight (20%) were on the left and two (5%) were bilateral. In cases of bilateral hernia, only one side was operated upon at a time. Thirty two (80%) hernias were indirect while eight (20%) were direct. In all cases darning method of repair was used.

Average hospital stay was forty hours, ranging from 24 to 72 hours. The main complications were wound infection seen in three (7.5%) cases and haematoma in one (2.5%). No recurrence was reported during the study. Average cost of operation was rupees one thousand (being a government hospital).

DISCUSSION

Hernia is a common surgical problem being 73% of all cases and surgery is the only successful form of treatment.[4] No patient under the age of twenty years was included in this study. Some authors have reported the use of local anaesthetic agents for repairing inguinal hernia even at the age of one year.[5]

According to our study, the modified mesh hernia repair with stapling device is feasible, inexpensive and safe procedure that is well tolerated under local anaesthesia.[6] Another study shows that repair of inguinal hernia under local anaesthesia with tension-free polypropylene mesh has good results.[7]

In the present study darning method of repair with Proline No. 1 was undertaken with excellent results. Both indirect, as well as direct hernias, were repaired under local anaesthesia. Adequate analgesia was achieved in all the cases. Other authors have reported their experience with hernia under local anaesthesia and have found that there is also less need for early post operative analgesic medication as it avoids many of the complications associated with general or a spinal anaesthesia.[3]

In our study the only complications recorded were wound infection in 3 patients and wound haematoma in one patient which is the same as reported by Ahmad G. et al.[8] In one study it has been reported that two patients developed transient quadericeps paresis after local inguinal nerve block.[9] But no such complication was observed in our study.

A comparison of complication rate is shown in Table-II.

TABLE-II COMPARISON OF COMPLICATION RATE

COMPLICATION	PRESENT STUDY	OTHER STUDIES
Wound Infection	3 (7.5%)	4 (7%)
Haematoma	1 (2.5%)	2 (2.5%)
Quadriceps Paresis	0 (0.0%)	2 (2.5%)

Average hospital stay in our patients was forty hours which is quite short in our circumstances, where most of the patients come from rural areas and belong to low socio-economic group. The same results have been observed in other studies.[10]

Cost of hernia repair under local anaesthesia was significantly low, that is Rs.1000/- per patient, as compared to spinal or general anaesthesia and our cost is almost equal to those mentioned in other studies.[8] In our society, where majority of patients coming to government hospitals are from poor class, this factor is very important.

This study shows that inguinal hernia repair under local anaesthesia is safe and economical with short hospital stay. Secondly it avoids complications of general or spinal anaesthesia. We recommend that all adult patients with uncomplicated inguinal hernias should be treated for repair under local anaesthesia.

REFERENCES

1. Devlin H.B. Groin hernia. Surgery (Quarterly ed.) 1993; 385-96.
2. Giles G.R. and Halasz N.A. The abdominal wall and hernia In: Cuschieri A, Giles G.R. and Moosa A.R. eds. Essential surgical practice. 3rd ed. Oxford Butterworth Heinmann: 1995; 1433-50.
3. Karatersas A; Morris R.G.; Walsh D.; Hung P.; Slavotinek A.H. Evaluation of safety of inguinal hernia repair in elderly using lignocain infiltration anesthesia. Aust-N-Z-J-Surg 1993 Apr; 63(4): 266-9.
4. Mann C.V. Hernias. Umbilicus Abdominal wall. In: Mann C.V.; Russell R.C.G; Williams N.S. (eds.) Bailey & Love's short practice of Surgery 22nd ed. London ELBS 1995; 885-903.
5. Splinter W.M.; Bass J.; Komocar L. Regional anaesthesia repair in children: Local Vs Caudal Anaesthesia. Can-J-Anaesth 1995 Mar; 42(3): 197-200.
6. Ng-Ek; Chung S.C.; Lan Y.H.; Lee D.W.; Chan A.C.; Li A.K. Mesh hernioplasty using a stapler. Aust-N-Z-J Surg 1996 Nov; 66(11): 751-2.
7. Amid P.K.; Shulman A.G.; Lichtenstein I.L. Open "Tension-free" repair of inguinal hernia: The lichtenstein technique. Eur-J-Surg 1996 Jun; 162(6): 447-53.
8. Ahmad G; Ahmad F.; Aziz A.; Haq M.E. Inguinal hernia repair under local anaesthesia. The Professional. Jul-Aug-Sep 1996; 3(3): 193-96.
9. Szell K. Local anaesthesia and inguinal hernia repair: A cautionary tale. Ann-R-Coll-Surg-Engl 1994 Mar; 76(2): 139-40.
10. Agbakwuru E.; Arigbabu A.O.; Akinola O.D. Local anaesthesia in inguinal herniorrhaphy. Our experience in Ib-Ife, Nigeria Cent-Afr-J-Med 1995 Dec; 41(12): 405-9.

MANAGEMENT OF LARYNGEAL CANCER

TARIQ RAFI, KAMALUDDIN KHAN

ABSTRACT:
This is a retrospective study of 625 cases of laryngeal cancer seen over a period of ten years from 1989 to 1998 at the Department of ENT, Jinnah Postgraduate Medical Centre, Karachi. Most of the patients belonged to low socio-economic group and were in advanced stages of the disease. Nodal involvement was surprisingly low as compared to available international data. Different treatment options were used, that is laser surgery, total laryngectomy, near total laryngectomy, radio-therapy and chemo-therapy.
The prognosis was not bad for the laryngeal cancer and the 5 year survival rate was around 80%.

KEY WORDS: Laryngeal Cancer, Survival, Management.

INTRODUCTION

Laryngeal cancer is not very common and the incidence varies from place to place,[1] the areas of high incidence are Brazil, black population of the USA, Hong Kong and India, whereas the areas of low incidence are Japan, Norway, Sweden and New Guinea. Pakistan has an intermediate incidence, but as no cancer registry is maintained, it is very difficult to obtain correct statistical data. It represents approximately one third of all malignancies in the UK[2] and in Pakistan is the third most common malignancy after bronchogenic and oral cancer. The disease most commonly occur after the fifth decade of life and males are more commonly affected. The disease is closely associated with socio-economic, geographical, ethnic, environmental, habitual and dietary habits.

The commonest etiological factor in Pakistan is smoking[3] and to a lesser extent, pan chewing (tobacco). Alcohol is not an important etiological factor in this part of the world, but when this habit is present alongwith pan chewing and smoking, the incidence is very high.[3] A rise in incidence of laryngeal cancer was observed in England and Wales during 1960-1968 and 1972-1978.[4]

French being the highest alcohol consuming nation, there is a higher incidence of laryngeal cancer in France, as compared to rest of the Europe which has led to the suggestion[5] that alcohol plays a promoting role, particularly in the supraglottis cancer.

The disease usually starts in the fifth or sixth decade of life especially in males, either as a leukoplakia or fungating growth. The most common site in the subcontinent is supraglottis, followed by glottis. This is not reflected by the international studies, where glottis is the most common site. The disease usually presents late in Stage II or III, thus limiting the treatment options. There has not been much improvement in the treatment of laryngeal cancer due to late presentation, but now near total laryngectomy and other newer techniques offer better results, especially for conserving speech.

PATIENTS AND METHODS

This retrospective study was carried out on 625 patients of cancer larynx in the Department of ENT and Head & Neck Surgery, Jinnah Postgraduate Medical Centre Karachi, over a period of ten years from 1989 to 1998. A detailed history was obtained, a prepared proforma was filled and examinations including Indirect Laryngoscopy (IDL) and flexible endoscopy. Biopsy was taken with the flexible bronchoscope. Those patients in whom biopsy was not possible, were admitted for direct laryngoscopy under general anaesthesia. After confirmation of malignancy on biopsy, CT scan was done in those cases where spread of disease was suspected.

The TNM staging was done after which patient was either re-admitted for surgery or sent for radiothereapy. A regular

Correspondence:
Dr. Tariq Rafi Associate Professor of ENT,
Jinnah Postgraduate Medical Centre, Karachi

follow-up was carried out for a minimum period of two years. The data collected was compared with available local and international data to see the difference in the pattern of the disease, its presentation, treatment and prognosis.

RESULTS

The ages of our patients varied from 26 to 78 years.

TABLE-I SITE OF DISEASE N = 625

Stage	No. of Patients	Percentage
Supraglottis	329	52.6%
Glottis	226	36.2%
Sub-glottis	70	11.2%

TABLE-II STAGE OF DISEASE

Stage	No. of Patients	Percentage
T1	79	12.6%
T2	236	37.7%
T3	198	31.6%
T4	112	18%

TABLE-III TREATMENT OPTIONS

Treatment Option	No. of Patients	Percentage
Laser	38	6%
TL + Radical neck dissection	50	8%
T.L +Partial Pharyngectomy	112	18%
TL	319	51%
Laryngectomy/Radiotherapy	481	77%
Radiotherapy alone	66	17%

TL (total laryngectomy)

TABLE-IV PROGNOSIS

Site	3 Year Survival	5 Years Survival
Supraglottis	82.4%	73.6%
Glottis	93.1%	86.7%
Sub-glottis	72.3%	67.8%

Supraglottis was the commonest site in our study (Table I). It predominantly affected males in 609 cases (97.4%). The most common etiological factor was smoking in 623 cases (99.7%) or a combination of smoking and pan chewing. Five patients were using alcohol.

The most common histopathological feature was squamous cell carcinoma in 562 (89.9%) cases, followed by verrucous carcinoma in 44 (7.3%). The Majority of patients were either in T_2 or T_3 (Table II). The incidence of lymph node metastasis was around 11.1% (n=69) in our study.

The treatment options were laser, surgery and radiotherapy. Laser excision was done in 6% cases (n=38), where the tumor was in very early stage involving the glottis. Total laryngectomy (TL) was combined with radical neck dissection or total laryngectomy alone. In all cases of surgery adjuvant radiothereapy was given, radiotherapy alone was given in 17% (n=66) cases, which were in early stage of the disease T_1 & T_2, as this gives equally as good results as surgery, with voice preservation. Radiotherapy was also given in late stages of the disease as a palliative therapy, when surgery was not possible (Table III).

Stomal recurrence was seen in 2.3% (n=15) cases. The overall survival in glottis cancer over a five years period was 86% (Table IV).

DISCUSSION

Laryngeal cancer is one of the most common fatal disease affecting the male population. It accounts for approximately 1% of all malignancies[3] and is the third most common after bronchogenic and oral carcinoma. Undoubtedly the habit of smoking and pan chewing are the most common etiological factors, made worse by the malnourished status of the patient. The disease usually starts in the fifth decade of life as enythroplasia and leukoplakia and gradually progresses to become squamous cell carcinoma as seen in 89% cases,[6,7] followed by the verrucous variety, which is the second most common type seen in 7.3% cases. Its incidence in Pakistan is much higher than reported in the international literature.

The incidence of lymph node metastasis in our study is much less than reported in the UK and USA.[8,9] The regional lymph node metastasis depends upon age, stage, histology and site of the tumor. The younger the age, the more likelihood of regional lymph node metastasis and so is the case with tumors in advanced stages of disease. The supraglottis tumors have higher tendency for metastasis due to rich lymph supply, whereas the glottis has the least incidence. Advanced disease involves surrounding skin, with the result that after excision large defects are left behind which require reconstruction with flaps. Pectoralis major myocutaneous flaps were used in 3 cases to cover the skin defect.

The stomal recurrence in our study was low, 2.3% (n=15), though majority of patients needed tracheostomy several days before surgery; a factor which may be the cause high incidence of stomal recurrence. During surgery 0.6% (n=4) patients died due to various reasons like anaesthetic and surgical complications. The most common site was supraglottis, which is not in conformity with findings in the UK, where glottis is the commonest site. Supraglottis is also reported to be the commonest site in India and Bangladesh. A possible reason could be the dietary habit of chewing pan and tobacco.

The treatment options were limited to laser, surgery and radiotherapy or a combination of surgery and radiotherapy. Laser was used in very early lesions for scrapping or excision with a very strict postoperative followup.

Radiotherapy was used in early lesions as T_1 and T_2, where it gives equally good results as compared to surgery, but with the advantage of preserving the function. Total laryngectomy was carried out in majority of the cases where it was combined with modified neck dissection where lymph nodes were palpable. All cases of laryngectomy were followed by adjuvant radiothereapy. Laryngectomy was mostly performed for T_3 and T_4 cases. Total laryngectomy was done in some cases as a palliative treatment to relieve symptoms. Voice rehabilitation in majority of cases was through oesophageal voice; affording patients were given electro larynx. Our overall 5 year survival rate is in conformity with international data.

REFERENCES

1. Waterhouse, J., Muir, C. Cancer incidence in five continents, Vol. IV. IARC Scientific Publication No.42. 1982 Lyon: International Agency for Research on Cancer.
2. Powel, J. and Robin, P.E., Cancer of the head and neck the present state in head & neck cancer, edited by P. R. Evans & P.E. Robins. Castle House Publication 1983 P 3-16, Tunbridge Wells.
3. New house, M.L., Gregory, M.M; & Shannom, H. Eteology of Carcinoma of larynx. International Agency for research on cancer (IARC) Scientific publication 1980 30, 687-695.
4. Michael M., Increase in laryngeal cancer in Britain and Australia in relation to tobacco and alcohol trends. The Lancet '1' 1978 1244-1247.
5. Hinds M.W; Thomas, D.B., and O' Reily, H.P. Asbestos, Dental X-ray, Tobacco and Alcohol in the epidemiology of laryngeal cancer, cancer 1979 44 1114 - 1120.
6. Van Nostrand; Olofson, J. Verrucous Cancer of the Larynx. A chemical and pathologic study of 10cases. Cancer 1972 30, 691-702.
7. Ferlito, A and Recher, G., Ackerman's Tumor of the Larynx, A clinico pathologic study of 77 cases Cancer, 1980 46, 1617-1630.
8. Som, M.L. Conservation Surgery for Carcinoma of the Supraglottis. Laryngol Otol 1970 84, 655 - 678.
9. SAND, H, H. Neoplasma Malignum Laryngis thesis, Copenhagen; Polyteknisk Forlag 1975.

BREECH PRESENTATION
(AN EXPERIENCE WITH 137 CASES)

NAILA EHSAN

ABSTRACT:
This is a prospective study conducted at the Department of Obstetrics and Gynecology, Sandeman Provincial Hospital, Quetta over a period of 18 months from Jan: 1998 to Jun: 1999. A total of 4006 cases were delivered during this period, out of which 137 had breech delivery, making an incidence of 3.41%. Among them 43 (31.38%) were booked cases and 94 (68.61%) were nonbooked. Fifty-eight (42.3%) patients were primigravidas and 79 (57.6%) multigravidas. Nineteen (13.8%) patients had preterm, 113 (82.4%) had term and 5 (3.6%) had post term deliveries. Thirty-two (24.16%) patients had cesarean sections and 105 (75.84%) had different type of vaginal delivery. (i.e. assisted breech delivery 75, spontaneous vaginal delivery 21, and breech extraction 9). Minor degree of maternal complications in the form of genital tract infection, urinary tract infection, wound infection, postoperative abdominal distention and vomiting were observed in 50% cases undergoing cesarean section. Minor complications like extended episiotomy, vaginal lacerations, cervical tears and postpartum hemorrhage were seen in 7.6% patients undergoing vaginal deliveries. No maternal death occurred in this study. Twenty-two (16.05%) of fetuses showed different type of congenital malformations. Thirty-eight perinatal deaths occurred, most of them due to congenital malformations. Fetal morbidity and mortality was not different by either mode of delivery.

KEY WORDS: Breech Delivery. Complications.

INTRODUCTION

The presentation of caudal pole of the fetus over pelvic inlet or lower in the birth canal is called breech presentation.[1] Breech presentation is the most frequent malpresentation and is a clinical challenge to obstetricians due to association of increased risk to the mother and fetus. Perinatal mortality of all breech fetuses is approximately 25 per 1000 live births, which is 2-3 times higher than nonbreech fetuses.[2] This high incidence is because of association of breech presentation with prematurity and congenital malformations in addition to the risk of delivering a breech fetus. There has been a lot of controversy regarding the best mode of delivery of breech fetuses. Many centers adopt a policy to deliver all women with breech presentation by cesarean section without any benefit to the mother and fetus, while 40% of breech presentation could be delivered vaginally without endangering the neonatal outcome. Also in terms of maternal outcome vaginal delivery is the better option.[3] External cephalic version is gaining popularity in reducing the incidence of cesarean section for breech presentation.[4] It is the need of the day that patients with breech presentation should be given a chance to deliver vaginally provided they are carefully assessed and meet the set criteria.[5] Moreover experienced obstetrician in a fully equipped unit should manage breech delivery with all facilities of good neonatal care.

Correspondence:
Dr. Naila Ehsan Associate Professor, Department of Obstetrics and Gynaecology, Bolan Medical College, Quetta.

PATIENTS AND METHODS

This prospective study was conducted at the Department of Obstetrics and Gynecology, Sandeman Provincial Hospital, Quetta (attached teaching hospital of Bolan Medical College). The study period was from January 1998 to June 1999, over a period of 18 months. Most patients were nonbooked and admitted in labour ward with no antenatal record during pregnancy. Booked patients were carefully assessed in out-patient department for determination of mode of delivery. Detailed history of every patient was obtained regarding age, parity and previous obstetrical history, to include previous record of labour, perpeurium, weight of babies, histories of congenitally malformed babies, still births and any instrumental delivery including cesarean section.

General physical, abdominal and vaginal examination was carried out to assess the type of breech, its engagement, dilatation and effacement of cervix, presence or absence of membranes, color of liquor and pelvic adequacy in addition to maternal and fetal well being. Routine investigations like blood group, RH factor, hemoglobin estimation, blood sugar and urine analysis was done for every patient in labour. Ultrasound was done on all booked cases to confirm gestational age, size of fetus, to exclude multiple pregnancies, placenta previa and congenital abnormality. Mode of delivery was decided on the basis of clinical history, examination, ultrasound, assessment of the size of fetus and clinical pelvimetry. Patients who had any medical or obstetrical complication with breech presentation were selected for cesarean section and those who met the set criteria for vaginal delivery were given trial of labour. Patients with breech presentation undergoing labour were nursed in left lateral position and strictly forbidden for bearing down until full dilatation of cervix. Partogram was maintained and injection Neubain was given intramuscularly in first stage of labour. When cervix was fully dilated patient was put in lithotomy position and advised to bear down with uterine contractions. When perineum was distended and breech was climbing the perineum mediolateral, episiotomy was made after infiltrating the area with 1% Xylocain. Assisted breech delivery was employed.

RESULTS

The total number of deliveries during the study period were 4006. Out of these 137 were with breech presentation making frequency of 3.41%. Among 137 breech presentations 58 (42.3%) were primigravidas and 79 (57.6%) were multigravidas. According to gestational age 19 (13.8%) were pre-term, 113 (82.4%) were term deliveries and 5 (3.6%) were post term. Among 137 cases 43 (31.38%) patients were booked and had antenatal checkups more than thrice, while 94 (68.61%) were nonbooked patients. Out of 43 booked cases 11 cases were admitted through out- patient department for other associated complications like previous cesarean sections, pregnancy induced hypertension, gestational diabetes, contracted pelvis and postmaturity,while 32 booked cases were admitted in emergency in labour ward. The total who had vaginal delivery were 105(75.84%). the different types of vaginal deliveries were assisted breech delivery 75(71.4%), spontaneous breech delivery 21(20%) and breech extraction 9(8.56%). In 68 patients after coming head was delivered by Smelli-veit method and out-let forceps were applied to 7 patients. Patients undergoing cesarean section were 32 (24.16%), among them 13 (40.62%) were elective and 19 (59.37%) were emergency cesarean sections. Indications of cesarean section in patients are shown in Table-I.

TABLE-I INDICATION FOR CESAREAN SECTION IN BREECH PRESENTATION (N=32)

Indication for cesarean section	No. of Patients
Previous cesarean Section	8 (25%)
Weight of baby> 3.8 Kg	12 (37.5%)
Pregnancy induced Hypertension	2 (6.25%)
Gestational diabetes	3 (9.3%)
Contracted pelvis	2 (6.25%)
Placenta previa–III	1 (3.1%)
Precious Pregnancy	1 (3.1%)
Postmaturity	3 (9.3%)

Incidence of cesarean section was more in booked cases (65.62% n=21) compared to nonbooked cases (34.38% n=11). Eight (7.6%) cases undergoing vaginal delivery had maternal complications like extended episiotomy, vaginal laceration, cervical tears and post partum hemorrhage. Sixteen (50%) patients undergoing cesarean sections had maternal complications like genital tract infection, urinary tract infection, wound infection, vomiting and abdominal distention in post operative period. No maternal death was observed in this study.

Out of total 137 breech deliveries ,22 (16.05%) fetuses showed congenital malformations. All the congenitally malformed fetuses were delivered vaginally.

Apart from congenital malformations, no evident fetal morbidity was observed by either mode of delivery. The perinatal deaths noticed in this study were 38, giving an incidence of 277 per 1000 births. From these 38 perinatal deaths 35 fetuses were those who delivered by cesarean sections. The different causes of perinatal mortality in these 38 cases are depicted in Table II.

TABLE-II DIFFERENT CAUSES OF PERINATAL DEATH. (N=38)

Perinatal Death	No. of Patients
Congenital abnormality	18 (47.3%)
Prematurity	6 (6%)
Birth anoxia	
• struck head	2 (5.2%)
• Cord prolapse	2 (5.2%)
• Placenta previa	2 (5.2%)
• Abruptio placentae	3 (7.8%)
• Obstructed labour	2 (5.2%)
• Maternal hypertension	2 (5.2%)
• Undiagnosed	1 (2.6%)

DISCUSSION

The reported incidence of breech delivery at term is 3-4%, at 29-32 week is 14% and at 28 week is more than 20%.[6,7] In the present study the incidence of breech presentation is 3.41% of total deliveries. Among these breech deliveries only 13.8% were premature, which is somewhat lower than the reported incidence. This is due to the fact

that in our setup majority of patients do not maintain antenatal checkups and deliver at home by Dais; only a minority of patients specially with complications come for hospital delivery. Incidence of breech presentation increases with increasing parity and maternal age.[8] This trend is also observed in this study as 57.6% were multigravidas while 42.3% were primigravidas.

In modern practice two standard management strategies are accepted, one vaginal breech delivery for selected low risk patients and second cesarean section. The frequency of second approach is increasing day by day.[9] This is generating a need for increased level of experience in delivering the breeches via vaginal route, as younger obstetricians are increasingly becoming unskilled in this art.[10] To curtail the rate of cesarean section for breech delivery, external cephalic version is good option with a success rate of 30-42 % in United Kingdom and 65% in USA.[11-12] In this study external cephalic version was attempted successfully in six patients during their antenatal visits followed by immediate heart monitoring at 36 weeks of gestation, thus reducing the incidence of breech presentation and their associated problems. In 43 booked cases of breech presentation, external cephalic version was not attempted due to associated complications like placenta previa ,pregnancy induced hypertension, diabetes and multigravidas, because no significant benefit of external version has been reported in such patients.[13]

Our 76.6% patients with breech presentation were delivered vaginally and 23.3% underwent cesarean section. Most authors recommend a criterion for trial of vaginal delivery in breech presentation (as given in Table-III).[7]

TABLE-III CRITERIA FOR TRIAL OF LABOUR

- Estimated fetal weight 1.5- 3.8 Kg
- Extended breech
- Exclusion of footling breech or hyper extended neck
- Structurally normal fetus
- Normal liquor
- Normal placental site
- Exclusion of significant fibroid
- Normal clinical assessment of maternal pelvis
- Exclusion of significant maternal disease

This criteria was strictly adhered while patients with breech presentation were selected for vaginal delivery. During labour all such patients were monitored carefully by partogram. In those patients where the progress of labour was not satisfactory, the decision of cesarean section was taken.

It is an established fact that vaginal delivery in breech presentation is more successful in patients who are diagnosed during labour or received in labour with more than three centimeter of cervical dilatation, than in those who are diagnosed antenatally and allowed to deliver vaginally.[14] Similar finding have been observed in our study that 88.3% nonbooked and only 51.16% booked cases were delivered vaginally, beside the reasons contained in above quoted reference.[14] The high incidence of cesarean section among booked patients in our study was due to associated complications in these patients which compelled for their elective cesarean section.

Mode of delivery in breech presentation remained a point of controversy regarding the outcome of fetus. Cochrane systemic review is accepted as current gold standard of obstetrics. Analysis of its prospective study found no difference between two approaches in terms of perinatal morbidity. (malformations having been excluded).This study also mentioned that elective cesarean section was associated with high rates of maternal morbidity.[5] While another study showed a significantly high rate of fetal morbidity by vaginal delivery than by cesarean section: 12.2% versus 3.2% [15] It is widely perceived that perinatal morbidity rate in vaginal breech delivery is dependent on the quality of birth management, skill of professionals and criteria for admission to trial of labour.[16] Similarly in the present study perinatal morbidity was not different in either mode of delivery, whereas maternal morbidity was more in patients undergoing cesarean section.

Delivery of premature breech is also a debatable issue . Most of obstetricians use cesarean section for uncomplicated breech less than 32 weeks gestation,17 while others believe that poor outcome of premature breeches are mainly related to complication of prematurity and not to the mode of delivery.18 We are in favour of second group and found no difference in fetal outcome by either mode of delivery.

Six perinatal deaths occurred in premature breeches due to complication of prematurity and not by the complication of delivery. A total 38 perinatal deaths occurred in the present series. Most of them (47.35%) were due to congenital malformations and others were due to complications of pregnancy not related to the mode of delivery. In our study the incidence of congenital malformation in breech is quite high than the reported incidence of 6%.[19] This is due to the fact that majority of patients in our setup do not carry out a proper antenatal checkup so that selected termination of abnormal pregnancies would have been done in first trimester. Furthermore injudicial use of drugs in pregnancy is a common practice in our society.

REFERENCES

1. Alan. H, Decherney and Martin. L. Pernoll. Current book of gynaecology and obstetrics, 8th edition. Malpresentation and cord prolapse, 1994; 410-20.

2. Ayesha khan. Vaginal delivery in primigravida with breech presentation. Medical channel, 1997; 3(1): 18-21.
3. Bowes WA, Taylor ES, O'Brien M and Bowes C. Breech delivery: evaluation of the method of delivery on perinatal results and maternal morbidity. Am J Obstet Gynecol, 1979;135: 965-73.
4. Cantu. Esquivel. MG et al. External version in breech presentation. Gynecol obstet. Mex, Oct, 1996; 64: 474-6.
5. John Studd. Progress in gynaecology and obstetrics. Volume 13. Management of breech presentation at term, 1998;87-97.
6. Geoffrey Chamberlain. Obstetrics by ten teachers. 16th edition. Abnormal presentation, 1995; 206-14.
7. Haughey MJ. Fetal position during pregnancy. Am J Obstet Gynecol 1985; 153: 885-886.
8. F. Gery Cunningham et al. Williams obstetrics, 19th edition. Dystocia due to abnormalities in presentation, position and development of fetus, 1993; 493-501.
9. Cheng M & Hannah ME. Breech delivery at term-a critical review of the literature. Obstetrics Gynecol 1993; 82: 605-18.
10. Charles. R. Whitfield. Dewhurst textbook of obstetrics and gynaecology, 6th edition. Malposition , malpresentation and cephalopelvic disproportion, 1999; 277-90.
11. Bewley S, Robson SC, Smith M, Glover A and Spencer JAD. The introduction of external cephalic version at term into routine clinical practice. Eur J Obstet Gynecol Reprod Biol, 1993; 52: 89-93.
12. Zhang J, Bowes WA & Fortney JA. Efficacy of external cephalic version. Obstet Gynecol, 1993; 82: 307-12.
13. Healy M, Porter R, Galimberti A. Introducing external cephalic version at 36 or more in a district general hospital; a review and audit. Br J Obstet Gynaecol 1997; 104: 1073-9.
14. Nwosu EC, Walkinshaw S, Chia P, Manasse Pr, Atlay RD. Undiagnosed breech. Br J Obstet Gynaecol. 1993; 100: 531-35.
15. Collea JV, Chein C, Quilligan EL. The randomized trial of term frank breech presentation: a study of 208 cases. Am J Obstet Gynaecol. 1998; 137: 235-44.
16. Krebs L, Langhoff-Roos J & Weber T. Breech at term- mode of delivery? Acta Obstet Gynecol Scand. 1995; 74: 702-6.
17. Penn ZK & Steer PJ. How obstetricians manage the problem of preterm delivery with special reference to preterm breech. Br J Obstet Gynaecol. 1991; 98: 531-4.
18. Cibils LA, Karrison T & Brown L. Factors influencing neonatal outcome in the very low birthweight fetus(<1500g) with a breech presentation. Am J Obstet Gynecol 1994; 171: 35-42.
19. Neville F, Hacker J & George Moore. Essentials of obstetrics and gynaecology, 2nd edition. Fetal malpresentation. 1992; 230-35.

AMOEBIC LIVER ABSCESS: ASPIRATION VERSUS CONSERVATIVE TREATMENT

SUHAIL AHMED ALMANI*, ABDUL SATTAR MEMON**, NOOR MUHAMMAD MEMON*.

ABSTRACT:

A study was conducted at Liaquat Medical College Hospital Jamshoro, Hyderabad for comparing the results of therapeutic needle aspiration versus drug treatment alone in amoebic liver abscess. Fifty patients were enrolled, out of which 27 were considered for both ultrasound guided needle aspiration (s) and drugs, and 23 were treated with drugs alone. Results showed that abscess cavities of 26 patients (96.3%) of the aspirated group and 13 patients (56.5%) of the non-aspirated group resolved at the end of third week. It was observed that therapeutic needle aspiration (s) under ultrasound guidance was more effective in achieving rapid clinical response and it accelerates resolution and has additional advantages of being inexpensive and simple with short hospital stay.

KEY WORDS: Amoebic liver abscess, Treatment.

INTRODUCTION

Amoebic liver abscess is a common extra-intestinal manifestation of Entamoeba histolytica.[1,2] Hepatic amoebiasis is very common in warm climates of the tropical and sub-tropical countries, but rarely occurs in temperate zone where it is imported by tourists, immigrants and expatriates.[3] It is common in Mexico, Africa and South East Asia[4] and very common in Pakistan.[5,6] Due to recurrent amoebic intestinal infection, the prevalence of amoebic liver abscess is also very high. The low socio-economic class is particularly affected with this disease. Malnutrition, poor personal hygiene, poor sanitary conditions and ignorance are important factors contributing to high incidence of disease in this population.[7] Amoebic liver abscess with secondary bacterial infection behaves like pyogenic liver abscess, an early recognition of which is necessary to avoid high morbidity and mortality in these cases.[8] Late diagnosis increases the incidence of complicated disease and about 5% patients with amoebic liver abscess die.[9] There is a need for increased awareness of possible amoebic abscesses in those who have been in endemic areas, because with prompt use of effective treatment, complications can be avoided.[10]

PATIENTS AND METHODS

This study included 50 cases of amoebic liver abscess, admitted at Liaquat Medical College Hospital Jamshoro / Hyderabad, over a period of three years from July 1997 to June 2000. A diagnosis of amoebic liver abscess was made on ultrasonographic examination and serological test based on IHA.

Patients were interrogated in detail and examined thoroughly. They were subjected to complete blood picture, liver function tests. HBs Ag, stool test for entamoeba histolytica and x-ray chest for any pleuropulmonary complication. Aspiration of the hepatic pus was done in 27 patients and tested for bacteriology and active trophozoites. On ultrasonography the number, site and size were determined.

These 50 patients were divided into two groups. Group-I: 27 patients (54%), treated with both drugs and ultrasound guided therapeutic needle aspiration. Group-II: 23 patients (46%), treated with drugs alone. In all cases amoebicidal drug therapy, (Metronidazole and Chloroquine infusion / oral-dose according to age) was started and in cases of secondary infection anti-bacterial therapy was instituted.

The percutaneous needle aspiration was attempted in the operation theatre under local anaesthesia. The depth and centre of abscess was established under ultrasound guidance. Disposable spinal needle (size 16) was introduced into the liver and the abscess aspirated. The aspirated material was sent for culture, sensitivity and cytology. The ultrasound examination was repeated for

Correspondence:
Dr. Suhail Ahmed Almani
*Department of Medicine **Department of Surgery
Liaquat Medical College Hospital,
Jamshoro/Hyderabad

the confirmation of complete evacuation of abscess contents after 24 to 48 hours. Whenever the residual material was reported, aspiration was repeated with the same procedure. Both groups of patients were monitored clinically and sonographically.

RESULTS

In a total of 50 patients of amoebic liver abscess 41 (82%) were males and 9 (18%) females, age ranging from 12 to 70 years. Patients' history and physical examination revealed that fever (96%), right hypochondrium pain (94%) and anorexia (60%) were the commonest symptoms, while hepatic tenderness (100%) and hepatomegaly (100%) were the cardinal signs of amoebic liver abscess. Temperature ranged from 38.5°C to 40°C. It was remittent in 70% and intermittent in 26% cases. It was accompanied by chills in 36% cases. Splenomegaly was present in only 4% cases (Table-I).

TABLE-I CLINICAL FEATURES IN CASES WITH AMOEBIC LIVER ABSCESS

Clinical Features	Number of Patients	Percentage
Fever	48	96%
Right hypochondriac pain	47	94%
Chills	18	36%
Anorexia	30	60%
Nausea and vomiting	28	56%
Loose motions (6-8 day)	10	20%
Generalized weakness	50	100%
Weight loss	03	06%
Dry cough with chest pain	04	08%
Anaemia	36	72%
Jaundice	09	18%
Hepatomegaly	50	100%
Hepatic tenderness		
• Mild	21	42%
• Moderate	16	32%
• Severe	13	26%
Splenomegaly	02	04%

Stool examination showed cysts in 80% cases and vegetative forms in 20%. Laboratory investigations showed a raised serum bilirubin in 46% and raised SGPT in 58% cases. More significant was the level of serum alkaline phosphatase which was elevated in 90% cases with a mean rise of two fold above the normal. Leucocytosis was present in all the patients (Mean TLC=19,807/cu mm). ESR was raised in 100% cases and Hb was <10 gm/dl in 72% cases.

Serological test (Indirect haemagglutination test) revealed anti-amoebic antibodies present in 100% cases. Liver abscess aspirates examination showed brownish colour in 22 cases and yellowish brown in 5 cases. Trophozoites were present in 19 cases. Pus for C/S showed E-coli in 2, klebsiella, staphylococus aureus and streptococus pyogene in one case each (Table-II).

TABLE-II LABORATORY FINDINGS IN AMOEBIC LIVER ABSCESS

Investigation	No. of Patients	Percentage
Hb < 10gm/dl	36	72%
ESR raised	50	100%
Hyperbilirubinaemia	23	46%
SGPT raised	29	58%
Alkaline phosphatase raised	45	90%
Hypoalbuminaemia	19	38%
Stool		
• Cysts of Entamoeba histolytica	40	80%
• Trophozoites of Entamoeba histolytica	10	20%
Serological test (Indirect Haemagglutination)		
Anti-amoebic antibodies - Present	50	100%
Liver abscess aspirates examination	27	100%
• Colour		
Brownish (Anchovy Sauce)	22	82%
Yellowish brown	05	19%
• Trophozoites of E. histolytica	19	72%
• Growth on aerobic culture	05	19%
E. Coli	02	7.6%
Klebsiella	01	3.8%
Staphylococcus aureus	01	3.8%
Streptococcus Pyogenes	01	3.8%

Radiology of the chest was normal in 33% cases. Two major radiological abnormalties noted were raised right hemidiaphragm 48% and right sided pleural effusion (8%) while 12% had both abnormalities.

Ultrasonographic findings showed that the size of the abscesses ranged from 3.8 x 3 cms to 14 x 13.3 cms. Abscesses in the right lobe were 90%, in the left 8% and in both lobes 2%. Cases having a single abscess were 76%, with two abscess 18% and multiple abscess 6%.

In the aspirated group of 27 patients, 14 patients (51.8%) had one aspiration, 8 patients (30%) had two aspirations and 5 patients (19%) had more than two aspirations.

The results show that abscess cavities size less than 8 x 7 cms (15 out of 15 patients), abscess cavities size more than 8 x 7 cms (11 out of 12 patients) of the aspirated group and in the non-aspirated group abscess cavities less than 8 x 7 cms (8 out of 13 patients) and abscess cavities more than 8 x 7 cms (5 out of 10 patients) (total 13 out of 23 patients i.e. 56.5%), resolved at the end of third week.

DISCUSSION

In the present study majority of patients presented with typical clinical features of amoebic liver abscess. Mahdi et al[11] reported fever (100%), right upper abdominal pain (66.7%), hepatomegaly (57.1%) and jaundice in 19% cases. Memon AS et al[12] observed jaundice in 7 cases.

Schwartz et al[13] have reported that fever accompanied by chills and sweating was present in over 75% patients; hepatomegaly was observed in 30-60% cases. While the predominant symptoms and signs recorded by Rubin et al[14], were fever (87%), hepatomegaly (51%) and right upper quadrant pain (47%).

Patients were followed-up daily for improvement. The initial complaints and general condition of patients slowly improved. There was resolution of fever, pain, tenderness in the liver area and hepatomegaly. It was observed that between 7-10 days, patients started tolerating an oral diet.

Follow-up ultrasonography was done in every patient on the day of admission and then weekly for subsequent weeks showed resolution in size of abscess cavity. In our study ultrasonography was found to be the most useful, non invasive and accurate means of establishing a diagnosis. It helps in localizing the site for aspiration and monitoring the complete healing or resolution time of abscess cavity. Ultrasound monitoring of patients showed the initial size of the abscess ranged from 3.8x3cms to 14x13.3cms and complete healing or resolution time in both groups varying between 20 to 90 days. Sikmjee et al[15], reported that the ultrasound examination of the liver appear to be very suitable or assessing satisfactory progress of healing in amoebic liver abscess. Freeman et al[16], reported that sonographic monitoring of patients treated for amoebic liver abscess has shown the resolution time varying between 18 to 120 days.

Our result shows a significant difference between two groups and there was more rapid clinical response in the aspirated group than in the non-aspirated group and the abscess cavities of 26 out of 27 patients (96.3%) of the aspirated group and 13 out of 23 patients (56.5%) of the non-aspirated group had resolved at the end of third week. No complication occurred in any case. Memon AS et al[17], attempted aspiration under ultrasound guidance and no abscess was missed; hence it is a better and safer procedure than blind approach.

A comparison of the results of this study with similar study of Freeman et al[16], showed that the abscess cavities of 18 out of 19 patients (94.7%) of the aspirated group and 10 of 17 patients (58.8%) of the non-aspirated group had resolved at the end of third week. Widjaya et al[18] reported that shorter duration of amoebic liver abscess resolution time in the group of patients treated with the combined therapy was observed, particularly in the Ist four weeks of the treatment and concluded that percutanceous needle aspiration is a successful thereapeutic approach in the treatment. Mehnaz et al[19] reported that aspiration of pus along with appropriate medication was found more appropriate management than medical therapy alone and aspirated group hospital stay was 7 days as compared to more than 20 days for non-aspirated group. Hai et al[20], reported that two hundred and twenty cases of amoebic liver abscess were seen between 1981-1986. Over 88% responded well to conservative treatment with aspiration (s).Thamlikitkul et al[21], reported that 222 patients with amoebic liver abscess were analysed from 1978 to 1985. Majority (78.8%) of the patients were successfully treated with a combination of antimicrobials and aspiration. Sharma et al[22] reported that after confirmation of the diagnosis, the amoebic liver abscess cavity should be aspirated for quick relief and cure.

Vanijanonta et al[23] reported that thirty six patients were treated with drugs and aspiration of the abscess. All patients responded well within four weeks and there was no relapse or treatment failure. Adeyeno et al[24], reported that thirty three patients with amoebic liver abscess were seen over a five-year period. Management consisted mainly of the administration of the antiamoebic agent metronidazole and repeated needle aspiration; 96.8% recovered within four weeks. Nigam et al[25], reported that aspiration of amoebic liver abscess together with a combination of metronidazole was more effective than either metronidazole alone or with chloroquine.

Keeping in view the results of this study and the other studies conducted at different hospitals we conclude that therapeutic needle aspiration under ultrasound guidance was more effective in achieving rapid clinical response and it accelerates resolution and has the advantage of being safe, simple, less time consuming. It hastens the recovery and shortens the duration of stay in hospital.

REFERENCES

1. Siddiqui-MN; Rizvi-SB; Ahmed-MS; Rizvi-IH Amoebic liver abscess complicated by a hepatoduodenal fistula Clin-Radiol; 1992 : 46(2); 142-3.
2. Ralls; Nalini-CS; Kaur-M. Amoebiasis-Entamoeba histolytic infections a review. East-Afr-Med-J; 1987: 63(1); 81-4.
3. SheikhM. AR; al Karawi-MA; Yasawy-M et al. Modern Techniques in the diagnosis and treatment of gastrointestinal and biliary tree parasites Hepatogastroenterology; 1991; 38 (2); 180-8.
4. Subhash-R; Nail Abraham Koshy; Nebeen C Nayak. Tropical liver disease. Medicine International 3; 1990; 3449.
5. Sheikh-Z; Khan MH; Qamer-R Profile of 100 cases of liver abscess: JPMA 1989; 39; 256-258.
6. Sheikh-Z; Khan MH; Qamer-R "Profile of 100 cases of liver abscess: JPMA 1989; 39; 256-258.
7. Khan-MH; Qamar-R; Sheikh-Z. Serodiagnosis of amoebic liver abscess by IHA method JPMA 1989: 39; 262-4.
8. Katzenstein D; Rickerson-V; Bravde-A. New concepts of amoebic liver abscess derived from hepatic imaging serodiagnosis and hepatic enzymes in 67 consecutive cases in Diego. Medicine 1982; (4); 237-246.

9. Adams-EB; Maclood-IN. Invasive amoebiasis; Amoebic liver abscess and its complications Med; 1977; 59(4); 325-334.

10. Lane-HR; Nicholson-G1. Amoebic liver abscess an Auckland experience, N.Z. Med-J; 1984; 28; 97 (752); 187-90.

11. N.S. Mahdi, F. Al-Obaidi and A.Z. Benyan Hepatic abscess among Iraqui Patients. JPMS 39: 295, 1989.

12. Memon AS, Memon A Percutaneous needle aspiration of the amoebic liver abscess under ultrasound guidance. Biomedica Vol-9 (1993) 40-42.

13. Schwartz, S.I. Shires; G.T, Spencer, F.C. and Stocer, EH. Principles of Surgery 3rd ed. New York Mc Graw Hill 1979.

14. Rubin, RH. Swartz, M. N and Malt, R. Hepatic abscess; changes in clinical, bacteriologic and therapeutic aspects. Am.J. Med. 1974; 57:

15. Skimjee-AE, Patel-A; Gathiram-V; Engle-Ibrecht-HE; Singh-K; Rook.F Serial ultrasound in amoebic liver abscess. Clin-Radiol; 1985; 36(1); 61-8.

16. Freeman-O; Alkamaguna-A; Jarikre-LN. Amoebic liver abscess; the effect of aspiration on the resolution or healing time: Ann-Trop-Med-Parasilot; 1990; 84(3); 281-7.

17. Widjaya-P; Bilic-A; Babic-Z; Ljubicic-N; Amoebic liver abscess; ultrasonographic characteristics and results of different therapeutic approaches Acta-Med-Lugost; 1991; 45(1); 15-21.

18. Mehnaz; Ali-SM. :Liver abscess in children not an uncommon problem". JPMA 1991: 41(11); 273-5.

19. Hai-AA; Singh-A; Miltal-VK; Karan GC Amoebic liver abscess Review of 220 cases: Int-Surg; 1991: 72(2); 81-3.

20. Thamlikitkul V; Yamwong P. Liver abscess; a clinical study of 222 patients J-Med-Assoc-Thai; 1990; 73(5); 264-8.

21. Sharma-MP; Acharya-SK; Verma-N; Dasarathy-S Clinical profile of multiple amoebic liver abscess J-Assoc-Physicians-India; 1990: 38(11) 1 837-9.

22. Sharma-MP; Rai-PR; Acharya – RP; Ray-IG; Tando-BN. Needle aspiration of amoebic liver abscess. BMJ; 1989; 25; 299 (6711); 1 209-9.

23. Vanijanonta-S; Bunnag-D; Looareesuwan-S; Harinasuta-T. Low dose tinidazole in the treatment of amoebic liver abscess Southeast Asian-J-Trop-Med-Public-Health; 1985 ; 16(2); 253-6.

24. Adeyeno-Ao; Aderovnmu-A Intra thoracic complications of amoebic liver abscess. J-R-Soc-Med; 1984; 77(1); 17-21.

25. Nigam P; Gupta AK; Kapoor KK, Sharan GR; Goyal RM; Jashi LD. Cholestasis in amoebic liver abscess. Gut' 1985; 26(2); P 140-5.

NICT)

VESICOURETERIC REFLUX IN CHILDREN

NOOR AHMAD CHANNA, ABDUL HAMEED SHAIKH, AFROZE RAMZAN

ABSTRACT:
A prospective study was carried out on 65 patients with vesicoureteric reflux (VUR). Twenty-four patients were in primary (group A) and 41 patients in secondary VUR (group B). Male to female ratio in group A was 1.6:1 and in group B 4:1. Nine (37%) patients in group A and 17 (42%) in group B presented after 3 years of age. Main clinical presentations of group A were failure to thrive, anaemia, U.T.I. and renal failure. Main clinical presentations of group B were urinary complaints, anaemia, failure to thrive, U.T.I and renal failure. Bilateral grade IV VUR was found in 11/24 (46%) in group A and 23/41 (55%) in group B. The causes of secondary VUR were PUV (32) prune belly syndrome (6) and meningomyelocele (3). Early diagnosis and prompt management is required to prevent irreversible renal impairment.

KEY WORDS: Vesicoureteric reflux, Types of VUR, Children.

INTRODUCTION

Vesicoureteric reflux means retrograde passage of urine from bladder into the ureter. It is diagnosed on micturiting cystourethrogram (MCUG). It may be primary due to short intravesical portion of ureter resulting in incompetent vesicoureteric junction[1] and secondary VUR is due to associated urological malformation like neuoropathic bladder, posterior urethral valves and ectopic ureter etc. According to international classification[2] VUR is graded from I to V.

There are three major consequences of VUR:

- It maintains a residual urine in the urinary bladder which predispose to UTI.
- In the presence of UTI, it transmits bacteria to upper urinary tract.
- It may transmits bladder pressure to kidneys.

VUR may disappear spontaneously during long term prophylaxis of UTI depending on grade of reflux. 85% of Grade I and II and 41% of grade III, IV and V disappear in 7 to 15 year follow up.[3]

Clinical presentation of VUR can vary from almost none to severe urinary obstruction in secondary VUR. Most common clinical presentations of VUR are U.T.I and reflux nephropathy. Chronic renal failure is not only due to VUR but also due to renal hypoplasia / dysplasia with in-utero associated reflux.[4] Reflux nephropathy is the commonest cause of CRF as shown in different studies.[5,6,7] This study was planned to determine clinical profile of patients with VUR and grade and type of VUR, whether primary or secondary.

Correspondence:
Dr. Noor Ahmad Channa Associate Professor Paediatrics
Chandka Medical College, Larkana.

PATIENTS AND METHODS

This is a study of 65 patients attending Nephrology Unit of National Institute of Child Health, Karachi between January 1995 and January 1998 having VUR on MCUG. All patients who presented with urinary complaints, UTI or renal failure were evaluated. Detailed history and clinical examination were recorded. Blood CP, urine analysis, urine culture, blood urea and serum creatinine were carried out to determine UTI and renal failure. VUR was suspected initially on ultrasound revealing hydronephrosis and hydroureter. Diagnosis was confirmed on MCUG that also determined the grading of VUR and type of VUR. Primary VUR was labelled as group A and secondary VUR as group B.

RESULTS

Out of the total of 65 cases included in the study, 55 (84.6%) were males and 10 (15.4%) females. All the patients with secondary VUR were male except one (Table I). Most of VUR patients presented with fever, anaemia failure to thrive and urinary complaints. Urinary complaints were more common in group B as compared to group A (Table II). Causes of secondary VUR were PUV 32 (78%), Prune-Belly syndrome 8 (15%) and Meningomyelocele 3 (7%).

In group A most common grade of VUR were grade IV (46%) and grade II (12%) (Table III). In group B most

common grades of VUR were grade IV (73%) grade III (17%) and grade V (10%). UTI was found in 22 out of 24 (91%) cases in group A and 32 out of 41 (78%) cases in group B. CRF was found in 9 out of 24 (37%) cases and 26 out of 41 (63%) in group B.

TABLE-I AGE AND SEX DISTRIBUTION

Age	Male	Female	Total	Percentage
Group A				
1-3 years	4	5	9	37.6%
4-6 years	7	1	8	33.1%
7-12 years	4	3	7	29.3%
Total:	**15**	**9**	**24**	
Group B				
1-3 years	23	1	24	28.5%
4-6 years	6	-	6	14.7%
7-12 years	11	-	11	26.8%
Total:	**40**	**1**	**41**	

TABLE-II CLINICAL PRESENTATION

Presentation	Group A (n=24)	Group B (n=41)
Fever	**18 (75%)**	**36 (88%)**
Anaemia	**17 (71%)**	**31 (76%)**
Failure to thrive	**13(54%)**	**27 (66%)**
Urinary complaint	**9 (37%)**	**36 (88%)**

TABLE-III GRADING OF VUR

Grades	Unilateral	Bilateral	Total
Group A			
I	-	-	-
II	-	1	1 (4%)
III	2	3	5 (21%)
IV	5	11	16 (67%)
V	-	2	2 (80%)
Total:	**7**	**17**	**24**
Group B			
I	-	-	-
II	-	-	-
III	2	5	7 (17%)
IV	7	23	30 (73%)
V	-	4	4 (10%)
Total:	**9**	**32**	**41**

DISCUSSION

Prevalence of VUR in Pakistan is not known whereas in western literature Bailey reported 0.4% to 1.8% of children without U.T.I and about one third of girls evaluated for U.T.I had V.U.R.[8] In our study primary VUR in boys is 62.5% and in girls 37.5%, which is comparable to Tiballs et al study which shows 75% in boys and 35% in girls.[9]

Primary VUR is more common than secondary and 85% of primary VUR, especially of lower grade resolve[10] spontaneously. Secondary VUR is caused by bladder outlet obstruction (functional e.g. neuropathic bladder and structural e.g. PUV) which only resolves with relief and treatment of obstruction[10]. We have less number of cases of primary VUR possibly due to selective use of MCUG, as used to happen in earlier studies from the west[11] or we are getting cases late as 90% of grade I and II reflux resolve within five years.[10]

UTI reveals presence of VUR in 35-50% cases.[12,13] In our study U.T.I was present in 91% in group A and 78% in group B, which is higher because the selected cases were with U.T.I. VUR in our study was 29% unilateral and 71% bilateral, which is comparably low to Tiball's study[9], showing unilateral 58% and bilateral 42%. This may be due to age difference in studies. We have not found grade I VUR in our cases. However grade III (20%) and grade V (8.3%) are nearly comparable to Tiball's study (grade III 24.6% and grade V 12.2%). It is also observed that grade II VUR (42%) is very low in our study as compared to Tiball's (40.8%), whereas grade IV (66.4%) in our study is much higher than Tiball's (14.4%). This may be due to earlier detection in Tiball's study.

REFERENCES

1. Stephens FP. Vesicoureteric reflux in congenital malformations of the urinary tract praeger publishers, New York 1983: 233.
2. International reflux committee Medical versus surgical treatment of primary vesicoureteric reflux Paediatrics 1981: 67 : 392.
3. Edwards P. Normandas K.S prescorsd. W et al. Disappearance of VUR during long term prophylaxis of U.T.I in children Br. Med. J 1977, 285-8.
4. Hinchliffe S.A Chan Y.T, Jan. Id Chan. Renal hypoplasia and postanatally acquired cortical loss in children with VUR Pediatr. Nephrol 1992: 6: 439-44.
5. Jamro S CRF Dissertation CPSP 1994.
6. Chantler C. Holiday M. Progressive loss of renal function Paeds Nephrol 2nd; Eds: Collin N.V.W Baltimore U.S.A: 7773-89
7. Sirin. A emer et al: Etiology of CRF in Turkish children: Paeds Nephrol. 9, 95: 548-51.
8. Belman AB, Vesicoureteric reflux-clinics of N. Am: 44, 1997: 1171-1190.
9. Tiballs JM, Bruyn RD, Primary vesicoureteric reflux: How useful is postnatal ultrasound Arch. Dis.Child. 1996-75: 444-7.
10. Rana. ES Lowe LH. Blask An et al: Imaging in Paediatric urology: clin: of N. Am.44 1997: 1065-89.
11. Najamaldin A, Burge DM, Atwell JP. Fetal vesicoureteric reflux. Br. J. urol 1990; 56: 403-6.
12. Jodal U. Natural history of bacteruria in childhood: infect: dis. Clin. N. Am 1987, 1:713.
13. Semellie JM. Edwards D, Hunter N. et al vesicoureteric reflux and renal scan. Kidney. Int. 1975 8 (suppl): 65.

AN EXPERIENCE WITH PRIMARY CLOSURE IN PILONIDAL SINUS

ANIS SUBHAN, NUSRAT ANIS, ABDUL MAJEED BALOCH.

ABSTRACT:
Variety of procedures adopted for the management of pilonidal sinus attest to the fact that the choice depends on the surgeon. The advantages of successful outcome in primary closure are obvious. This procedure is carried out under general anaesthesia, the hospital stay is minimal and postoperative care restricted to the basics. The problem with procedures carried out as day cases under local anaesthesia is the long and tedious follow-up required postoperatively till the wound heals. Of the 52 cases dealt by primary suture only one(1.92%) had recurrence and 2 of the initial 7 in whom suction drain was not kept required change of dressing because of soakage, prior to removal of stitches.

KEY WORDS: Pilonidal sinus, primary closure

INTRODUCTION

The term pilonidal appertains to nest of hair and is derived from Latin.[1] Its aetiology is unknown and subject to debate. The majority disagree on the theory of congenital origin.[1,2] The pathology of the established condition is well recognized; there are one or more midline pits in the natal cleft which are connected to a deeply situated cavity lined by granulation tissue. Tracks lined by granulation tissue run cephalad from the cavity either in the midline or laterally in the buttocks. It rarely presents after the age of 40 years[3] and is rarely seen in children. Due to the shearing action of the buttocks the hair enter the follicles in the natal cleft and infection develops.[4,5]

PATIENTS AND METHODS

Fifty-two patients admitted in Surgical Ward-2, Jinnah Postgraduate Medical Centre and from private clinic of the authors from January 1992 to August 2000 are included in this study.

The age of the patients varied from 19 to 35 years, average being 22 years. There were 32 males and 20 females; male / female ratio was 1.6 : 1. Two cases (3.85%) presented with acute pilonidal disease, in which the abscess was drained under local anaesthesia in the outpatient department and elective procedure was carried out after two weeks. Three (5.77%) had been operated previously and presented with painful infected wounds. They were admitted and put on antibiotics and daily wound dressing was carried out prior to surgery. The rest presented with chronic pilonidal disease with history of recurrent bouts of inflammation, which was treated conservatively on antibiotics by general practitioners.

Primary closure was carried out with wide excision of the sinus tract and underlying cavity, to expose healthy fat on both sides and glistening sacral fascia in the depths of the wound. Deep sutures of Prolene were placed at 2.5cm intervals across the wound, picking up the sacral fascia and left untied. The skin edges were then sutured. The deep sutures were tied over a roll of gauze placed over the wound. In the first seven cases no drain was kept, but suction drain was placed in the remaining cases, which was removed on the third postoperative day. The patients were advised strict bed rest for one week. Initially they were kept on a low residue diet to restrict postoperative bowel movements and advised to rest in the prone or semi-prone position. The deep sutures were removed on the seventh postoperative day and the skin sutures on the tenth day. Adoption of high standard of cleanliness and weekly hair removal was advised postoperatively.

RESULTS

There was only one recurrence (1.92%), which occurred in a very hairy female patient who left the hospital on the third postoperative day and the protocol was not observed. In the initial seven cases where no drain was kept, the dressing had to be changed in two (28.57%) cases. The dressing remained dry in the remaining cases

Correspondence:
Dr. Anis Subhan, Assistant. Professor of Surgery, Surgical Ward 2, Jinnah Postgraduate Medical Centre, Karachi.

and was only changed on the seventh postoperative day at the time of removal of the deep sutures.

DISCUSSION

There is little controversy regarding treatment of acute pilonidal disease. The majority agree to simple drainage followed by definitive surgery two to three weeks later.[6-8]

A variety of procedures have been used for the treatment of chronic pilonidal sinus. Simple treatment technique advocated by Lord and Millar consists of excision of the midline sinus openings, picking up of hair from underlying cavities and from any lateral tracks.[9] Phenol injections are used to destroy the epithelium of the tracts and reliance is placed after care. This method has been used with varying results.[1,10,11]

Karydakis technique first described in 1977,[12] consists of asymmetric wound closure, which results in the primary sutured wound lying to one side or other of the midline. This technique has been used effectively by a number of surgeons with a recurrence rate of 0-4%.[13-15]

Bascom's technique[17,18] has its merits that it is undertaken as a day-case procedure under local anaesthesia, but needs strict weekly follow up till the wound heals. Recurrence rates of 8-10% have been reported.[4,16]

Bascom's cleft closure technique which is different from Bascom's original procedure, which is somewhat similar to Karydakis operation is advised for unhealed midline wounds. Though performed as a day case in local anaesthesia, it needs outpatient followup, as the majority of the wounds do not heal primarily.

Primary suture has been used with varying results.[6] The advantages of successful outcome after primary suture are obvious. The hospital stay is minimal, and the patient is spared the long and tedious convalescence associated with frequent and often painful dressings. The main cause of failure in primary closure is inability to secure firm apposition of the edges leading to collection of serum and blood in the cavity and ultimate disruption of the wound. Taking special care to bring the sides together and placement of a suction drain can prevent this. This is supported by adopting a high standard of cleanliness, and hair removal in the region postoperatively till the wound gains full strength.

REFERENCES

1. Edwards MH. Pilonidal sinus: a 5 – year appraisal of the Millard-Lord treatment. Br J Surg 1977; 64: 867-868.
2. Brearley R. Pilonidal sinus. A new theory of origin. Br J Surg1955; 43: 62-68.
3. Clothier PR, Haywood IR. The natural history of post anal (pilonidal) sinus. Ann R Coll Surg Engl 1984; 66: 201-203.
4. Bascom J. Pilonidal disease : origin from follicles of hairs and results of follicle removal as treatment. Surgery 1980; 87: 567-572.
5. Bascom J. Pilonidal disease : long term results of follicle removal. Dis Col Rectum 1983; 26: 800-807.
6. Goligher JC. Surgery of the anus rectum and colon, 5th Ed. London : Bailliere Tindall, 1984; 221-236.
7. Senapati A, Cripps NPJ. Pilonidal Sinus. Recent advances in surgery 23, 2000; 33-42.
8. Millar DM, Lord PH. The treatment of acute post anal pilonidal abscess. Br J Surg1967; 54: 598-599.
9. Lord PH, Millar DM. Pilonidal sinus : a simple treatment. Br J Surg 1965; 52: 298-300.
10. Maurice BA, Greenwood RK. A conservative treatment of pilonidal sinuses of the natal cleft Br. J Surg 1964; 51: 510-512.
11. Stansby G. Phenol treatment of pilonidal sinuses of the natal cleft. Br J Surg 1989; 76: 729-730.
12. Karydakis GE. New approach to the problem of pilonidal sinus Lancet 1973; ii:1414-1415.
13. Anyanwu AC, Hossain S, Williams A, Montgomery AC. Karydakis operation for sacrococcygeal pilonidal disease : experience in a district general hospital. Ann R Coll Surg Eng 1998; 80: 197-199.
14. Karydakis GE. Easy and successful treatment of pilonidal sinus after explanation of its causative process. Aust N Z J Surg 1992; 62: 385-389.
15. Kitchen PR. Pilonidal sinus : experience with the Karydakis flap. Br J Surg 1996; 83: 1452-1455.
16. Senapati A, Cripps NPJ, Thompson MR. Bascom's operation in the day-surgical management of symptomatic pilonidal sinus Br.J Surg 2000; 87(8): 1067-1070.
17. Bascom J. Pilonidal sinus In : Current Therapy in Colon and Rectal Surgery. New York : Dekker, 1990; 32-39. Bascom J. Pilonidal sinus. Curr Pract Surg 1994;6: 175-180.
18. Bascom H. Pilonidas sinus. Curr Pract Surg 1994;6: 175-180.

COAGULATION DISORDERS IN PATIENTS WITH CARCINOMA OF THE HEPATOBILIARY AND PANCREATIC SYSTEM

AISHA Y MALIK*, TAHIR H MALIK**, ABDUL HAYEE**

ABSTRACT:
Coagulopathies are common accompaniments of hepatic disease. Besides other pathologies, carcinomatosis also involves the hepatobiliary system and pancreas. Liver is at the center of coagulation and is affected by these diverse pathological conditions. An analysis of coagulation disorders was performed in patients with coagulopathies secondary to carcinomas of hepatobiliary system, with a view to study the spectrum of disorder. Twenty-five patients with coagulation disorders were studied. Of these 7(28%) had hepatocellular carcinoma, 12 (48%) had metastatic liver disease, 3 (12%) patients had carcinoma of gall bladder, 2(8%) had carcinoma head of pancreas and 1(4%) had a periampullary growth. Patients with deficient /defective Vit. K dependent factors showed a markedly prolonged PT(36%).40% had prolonged PT and APTT, whereas the TT was affected in those with deficient/defective fibrinogen. Patients with DIC fared the worst with prolonged coagulation test times, raised FDP concentration and thrombocytopenia.

KEY WORDS: Coagulation disorders, Liver disease, Disseminated intravascular coagulation.

INTRODUCTION

Coagulation involves a multitude of proteins which undergo sequential activation resulting in the production of a procoagulant enzyme, alpha thrombin, which is responsible for the formation of fibrin plug. It is a rapid response and limited both in terms of location and duration. Coagulation is classically divided into the intrinsic and the extrinsic pathways which converge upon the common pathway. There are, however, various sites of interaction between these pathways. Present evidence suggests that the intrinsic pathway plays an important role in the propagation and maintenance of fibrin formation while the extrinsic pathway is critical in the initiation of fibrin formation. Disorders of coagulation occur when there is disturbance in the process of formation of the permanent hemostatic plug. These disorders are invariably caused by deficiency or decreased activity of one or a number of coagulation proteins.

As the production and regulation of the components of haemostasis is largely dependent on normal hepatic function, liver disease is associated with alteration of coagulant / anticoagulant, fibrinolytic / antifibrinolytic balance, as well as qualitative and quantitative alteration in the cellular components of haemostasis.[1] The liver synthesizes majority of the coagulation factors including fibrinogen, factor II, VII, IX and X, as well as factors XI, XII, XIII and factor V. Similarly synthesis of plasminogen and the inhibitors-antithrombin III, antiplasmin and protein C and S occurs in the liver.[1] It is also the site of clearance of active coagulation factors and their inhibitor complexes. Liver disease with cholestasis leads to Vitamin K malabsorption. This results in the failure of carboxylation and acarboxy forms are present in the plasma. Patients with severe liver disease often demonstrate haemostatic defects characteristic of disseminated intravascular coagulation. Malignancy too, is often associated with DIC. Cytokines, especially TNF and interleukin 1, can elaborate tissue factor formation intravascularly. Cancer procoagulants directly activate factor X, as well.[2] Hepatocellular carcinoma is prevalent in Asia and West Africa due to high HBV and HCV load bile duct obstruction and invasion of the duct by tumour embolus causes jaundice with resultant sequelae.[3] In HCC formation of des-gamma carboxy forms of Vitamin K dependent factors form and des-gamma carboxy

Correspondence:
Dr. Aisha Y. Malik,
*Department of Pathology, **Shaikh Zaid Hospital, Lahore
Fatima Jinnah Medical College, Lahore.

prothrombin has been reported in 75-90% patients. This is used as a tumour marker.[4] These acarboxy forms cause coagulation abnormalities. Differential diagnosis includes, among others, metastatic liver disease. Pancreatic carcinomas, especially of the body and tail are associated with D I C as opposed to patients with carcinoma of the head of pancreas which causes ductal obstruction.[5] These arise twice as frequently in the head(70%)as in the body(20%) or tail (10%) of the gland. Coagulation tests are affected[6,7] variably depending on the severity of the disease. The purpose of this prospective research was to study the spectrum of coagulation disorders secondary to carcinomas of the hepatobiliary and pancreatic system.

PATIENTS AND METHODS

An analysis of coagulation disorders secondary to carcinoma of the hepatobiliary and pancreatic system was performed in 25 patients. These patients were admitted to the medical and surgical wards of Sheikh Zayed and Sir Ganga Ram hospitals and had been diagnosed either on ultrasound or ultrasound guided liver biopsy, for metastatic liver disease or hepatocellular carcinoma. Twenty control samples were run with every batch of test plasma. The control group were normal, healthy subjects of both sexes, who were not taking any medication and gave blood voluntarily.

Blood samples were collected by standard atroumatic procedure, mixed with anticoagulant and immediately dispatched to laboratory.

The samples were divided in three aliquotes:

- In a glass tube containing Ethylenediamine tetra acetic acid (in the concentration of 1.5±0.25 mg/ml)1 ml was delivered.
- 4.5 mls were citrated by adding the blood to 0.5 ml of 3.2% trisodium citrate (.109M) in a glass tube and gently mixed by inversion. This citrated blood was centrifuged at 2000g for 15 minutes to obtain platelet poor plasma.
- 2 mls were added to the spli-tube contained in the FDP kit. This was allowed to stay at 37ºC. After clot had formed and retracted the serum was collected directly or else after centrifugation.

Control blood samples were taken in the same manner as the patients and tests were carried out without delay. Platelet count was performed manually using the improved Neubauer chamber and 1% ammonium oxalate as diluent. Prothrombin time (PT) was done using commercially available reagent Neoplastine (Diagnostica Stago). It is a freeze dried thromboplastin prepared from fresh cerebral tissue, in calcium with a predetermined ISI. For activated partial thromboplastin time (APTT): C.K. prest (Diagnostica Stago) was used .It contains:

- Reagent 1: Cephalin (platelet subsitute) freeze dried
- Reagent 2: Activator, buffered suspension of kaolin (5mg kaolin/ml). CaCl2 - 0.025 M.

Thrombin Time (TT) was tested using commercially available reagent, thrombin-prest (Diagnostica Stago). It is titrated calcium thrombin (approximately 3 NIH units / vial) freeze dried. The coagulation tests were performed in duplicate by the manual tube tilt method as described by Dacie and Lewis.[8] For fibrin degradation product (FDP) commercially available reagent used was Spli Prest (Diagnostica Stago). Semi quantitative estimation was done by latex agglutination.

RESULTS

Of these 25 patients,7 patients had hepatocellular carcinoma, 12 patients had metastatic liver disease, 2 patients with carcinoma of the head of pancreas, (one had metastasis in the liver too), 1 had a periampullary growth with metastatic deposits in the liver, 3 patients had carcinoma of the gall bladder. Of these 3 patients, one had undergone cholecystectomy, one had metastatic deposits at the porta hepatis the third had cholelithiasis as well. The bilrubin, AST and ALT were variably raised, however patients with obstructive jaundice showed markedly raised alkaline phosphatase also.

The mean age of all patients was 51.36±11.2 years (30-70 yrs). The patients with metastatic liver disease had a mean age of 48.5±10.8 years (Mean±SD). The mean age of the patients with hepatocellular carcinoma was 47.8±21.1 years, there were 4 males and 3 females. The two patients suffering from carcinoma of the pancreas were 65 and 70 years respectively, while the patients with carcinoma of the gall bladder, the range of age was 35-44 years mean 40.3+ 4.7 years. The former were both males and the latter were females. Of the 12 patients with MLD, 6 were males and 6 females. The patient with periampullary growth was a 60 years old male.

The platelet count of all the 25 patients ranged between 30-300X10^9/L. The 12 patients who had metastasis in the liver platelet count was between 68-300X10^9/L mean 168.5±61.9X10^9/L.The 7 patients with hepatocellular carcinoma had a platelet count of 30-200X10^9/L with a mean of 131.8±(71.8SD)X10^9/L. Of these HCC patients 3 patients had a count of 30X10^9/L - 80X10^9/L mean 57.3 + 25.5 X 10^9/L and the rest 187.75 + 13.2 10^9/L.The 3 patients with carcinoma of the gall bladder had a mean platelet count of 263.0±16.0X10^9/L (250-281X10^9/L).The patient with periampullary growth had a platelet count of 179X10^9/L. The 2 patients with carcinoma of head of pancreas had a platelet count of 35X10^9/L and 120X10^9/L The platelet count of the control group was 313±56.4X10^9/L.

Prothrombin Time (P.T) is given in Table I.

TABLE-I PROTHROMBIN TIME OF PATIENTS RANGE OF P.T.

Disease	12-14 Sec	15-20 Sec	21-30 Sec	31->60 Sec
Hepatocellular Carcinoma (n=7)	—	2(28.5%) Mean(15.5±0.7) INR(mean 1.5±0.6)	3(42.8%) Mean(25.3±4.1) INR(mean 4.0±1.2)	2(28.5%) Mean(50.0±14.1) INR(mean 15.5±8.97)
Metastatic Liver Disease (n=12)	—	2(16.6%) Mean(18.5±2.1) INR 2.05±0.9)	9(75%)** Mean(24.2 ±2.6) INR 3.7± 0.9	1(8.3%) INR 10.04
Carcinoma Gall bladder (n=3)	—	—	3(100%)** Mean (24.0±1.0) Mean INR 3.96±0.23)	—
Carcinoma head of pancreas (n=2)	—	1(50%) INR 1.6	—	1(50%) INR 21.9
Periampullary growth (n=1)	—	—	1(100%) INR 33	—

* *p < .01*
** *p < .001*
Range of control 12-14 Seconds. (mean 12.5 ± 0.9)
Normal range 11-16 Seconds[8]
INR 1.05 ± 0.06

Activated Partial Thromboplastin Time (A.P.T.T) is given in Table II.

TABLE-II ACTIVATED PARTIAL THROMBOPLASTIN TIME OF PATIENTS RANGE OF A.P.T.T.

Disease	34-40 Sec.	41-50 Sec	51-60 Sec	>60 Sec
Hepatocellular Carcinoma (n=7)	2(28.5%) Mean 39.5±0.7	3(42.8%) Mean 46.6±2.0	–	2(28.5%) Mean 81.5±26.1
Metastatic Liver Disease (n=12)	5(41.6%) Mean 39.2±1.70	6(50%)** Mean 47.0±2.9	1(8.3%)	–
Carcinoma Gall bladder(n=3)	2(66.6%) Mean 34 ± 0	1(33.3%)	–	–
Carcinoma head of pancreas(n=2)	–	1(50%)	1(50%)**	–
Periampullary growth(n=1)	–	1(100%)	–	–

* *p < .01*
** *p <.001*
Range of control 34-40 Seconds. (mean 36.3 ± 2.1)
Normal range 30 - 40 Seconds[8]

DISCUSSION

Liver disease is common in developing countries because of the high prevalence of viral hepatitis, with a considerable incidence of chronic infection with progression to cirrhosis, liver failure and hepatocellular carcinoma[9]. The mean age of patients with hepatocellular carcinoma was 47.8(±21.1) years in our study. A similar age incidence has been reported by others[10-12].

Thrombin Time (T.T) is given in Table III.

TABLE-III THROMBIN TIME OF PATIENTS RANGE OF T.T

Disease	14-17 Sec	18-30 Sec	31-40 Sec	60 Sec
Hepatocellular Carcinoma (n=7)	4(57.1%) Mean 15.3±1	1(14.2%)	1(14.2%)	1(14.2%)** 81.5±26.1
Metastatic Liver Disease (n=12)	10(83.3%) Mean 15.0±0.9	1(8.3%)	–	1(8.3%)**
Carcinoma Gall bladder (n=3)	3(100%) Mean 14.6 ± 1.1	–	–	–
Carcinoma head of pancreas (n=2)	1(50%)	–	1(50%)**	–
Periampullary growth(n=1)	1(100%)	–	–	–

* *p<.01*
** *p<.001*
Range of Control 14-17 Seconds. (Mean 16.3 ±
Normal Range < 2 Seconds from control[8]

Fibrinogen Degradation Products (F.D.P) is given in Table IV.

TABLE-IV SERUM FIBRINOGEN DEGRADATION PRODUCT CONCENTRATION

Disease	<10ug/ml 40ug/ml	>10ug/ml,< ug/ml	>40ug/ml,<80	80ug/ml
Hepatocellular Carcinoma (n=7)	5(71.4%)	–	1(14.2%)	1(14.2%) 81.5±26.1
Metastatic Liver Disease (n=12)	10(83.3%)	1(8.3%)	1(8.3%)	–
Carcinoma Gall bladder (n=3)	3(100%)	–	–	–
Carcinoma head of pancreas (n=2)	–	1(50%)	–	1(50%)
Periampullary growth(n=1)	1(100%)	–	–	–

Patients with metastatic liver disease (MLD) were 48.5±10.8 years old. Riaz et al[11] report 51 years as the mean age of their patients with MLD. The two patients with carcinoma of head of pancreas were 65 and 70 years of age respectively which is similar to the age incidence reported by Beazley.[13] The mean age of patients with carcinoma of gall bladder was 40.3+/-4.7 years.Waris et al[14] also report an average age of 45 years.

In the 7 hepatocellular carcinoma patients there were 4 males and 3 females. Riaz et al[11] also report male preponderance. Taseer et al[12] report 72% males and 28% females in their study. In metastatic liver disease the male :female ratio was 1:1. Similar ratios have been reported by Riaz et al.[11] Carcinoma of the head of pancreas was in males only, a fact substantiated by Beazley.[13] He also reports that 75-80% have obstructive jaundice, as did our patients. As for carcinoma of the gall bladder our patients

were all females Waris et al[14] also report a female preponderance.

In our patients the most frequent presentation of the coagulation disorder was prolongation of the P.T, this was seen as an isolated disorder in 36% of the patients, followed by the APTT. This is because of obstruction to bile flow caused by the metastatic deposits in the liver parenchyma, at portahepatis and obstruction of the common bile duct resulting in Vitamin K deficiency therefore factors II, VII, IX and X are affected initially. Vitamin K hydroquinone acts as a co-factor to gamma glutamyl carboxylase which converts the glutamic acid residues(Glu) at NH2 terminal of coagulation factors II, VII, IX, X, protein C&S to gamma carboxyglutamic acid (Gla). These post translational gla residues enable them to bind to calcium and phospholipid membrane, events that are crucial for their activity. Decrease in factor VII and II is more pronounced in cases of neoplastic obstruction. As vitamin K supplies deplete and the production of functional, gamma carboxylated factors decrease, the plasma levels of previously produced factors fall in accordance with their biologic half lives. Factor VII has a very short half life of +/- 6 hours. Thus P T is affected the earliest.[15] At the same time there is a progressive increase in nonfunctional acarboxy II, VII, IX and X in the plasma. These proteins induced by Vitamin K absence(PIVKA) exert an inhibitory effect on coagulation.

Prolongation of both the PT & APTT was observed in 40% of our patients. Parenteral vitamin K restored the prolonged times to normal in the absence of hepatic parenchymal damage. These tests should be measured 6 to 12 hours after the injection, when any residual prolongation would reflect the degree of hepatocellular damage[1]. Similar observation has been reported by Robson et al,[16] Sigurdson and Cohen,[17] Cuschieri[18] and Sherlock.[19] Hepatic failure produced encephalopathy and DIC in 5 of our patients. Similar findings have been reported by DeConno and Polastri[20] in metastatic liver disease patients.

In cases of hepatocellular carcinoma there is also an acquired vitamin K dependent carboxylation deficiency resulting in formation of des-gamma carboxy prothrombin in these patients. This has an inhibitory effect on coagulation as stated earlier. Other vitamin K dependent factors are also affected and acarboxy/des-gamma carboxy forms occur.[21] These des-gamma carboxy forms occur because Vitamin K stores are low in tumor cells whereas they are normal in the nontumourous liver tissue. Parenteral vitamin K causes a transitory decrease in the level of des-gamma carboxy prothrombin followed by a return to the abnormal level, contrasting with the pattern observed in patients with vitamin K deficiency due to other causes.[21,22] The thrombin time was prolonged in 6 patients, either because of defective /deficient fibrinogen or abnormalities of fibrin polymerization. Hypofibrinogenaemia may be because of deficient synthesis or fibrinolysis. It has also been suggested that the fibrinogen molecule itself is functionally abnormal in patients with liver disease.[7,23] These dysfibrinogens are because of increased sialic acid content of fibrinogen and have been described in HCC, acute hepatic failure and liver cirrhosis.[1,16,24] The fibrin degradation products were significantly high in 6 patients who had set a stage for disseminated intravascular coagulation. In 4 they were markedly raised whereas in two there was low grade DIC however in hepatocellular carcinoma it may have been the underlying cirrhosis which worsened. In one patient with HCC there was prolongation of TT but the FDP's were less than 10 ug/ml either because of dysfibrinogenemia or because the tumor cells secrete an antifibrinolytic agent[19] Pancreatic carcinomas arise twice as frequently in the head (70%) as in the body (20%)or tail (10%) of the gland.[25] One, of the two patients with carcinoma of head of pancreas had liver metastasis, it is a rapidly progressive tumour and early, invasion to the local nodes and liver has been reported by Phoa et al[26]. These malignant pancreatic cells express raised levels of tissue factor which activates blood coagulation.[27] Iversen et al[28] report fulminant DIC in 10.8% patients in their study with advanced cancer, of whom one had carcinoma of pancreas. Nesvetov[29] reports that in 42% of the lethal outcome in conditions as terminal stages of cancer, sepsis, mechanical jaundice etc. DIC was the cause of death.

The overall magnitude of the disturbance of coagulation correlates fairly well with the extent and severity of the underlying disease. Deficient / Defective synthesis of Vit. K dependent coagulation factors was the prominent feature in the patients with obstructive jaundice, Vit. K injection restored the prolonged PT and APTT to normal. In patients with disseminated intravascular coagulation the global coagulation tests were deranged. Our reliance on these continues for rapid results. However, for proper management of these patients serial evaluation of these tests is essential.

REFERENCES

1. Lee LH, Baglin TP. Haemostasis associated with liver disease and liver transplantation. Annals academy of medicine 1994; 23(6): 77-83.
2. Green KB, Silverstein RL. Hypercoagulability in cancer. Hematology / oncology clinics of North America 1996; 10(2): 499-530.
3. Stone MD, Cady B. Cancer of the liver, gallbladder, and bile duct. In Cancer Surgery; Ed. McKenna RJ, Murphy GP. JP Lippincott Philadelphia 1994;87-93.
4. Khurshid H, Malik IA. Hepatocellular carcinoma: clinical features, evaluation and treatment. J Pak Med Assoc 1995; 45(5): 136-142.

5. Bick RL, Baker WF. Disseminated intravascular coagulation syndromes. Heamotology pathology 1992; 1-24.
6. Lechner K, Niessner H, Thaler E. Coagulation abnormalities in liver disease. Seminars in thrombosis and hemostasis 1977; 4(1): 40-56.
7. Brozovic M. Acquired disorders of coagulation. In Hemostasis and thrombosis Ed Bloom AL; Thomas DP: Churchil Livingstone London UK 1987; 519-531.
8. Dacie JV, Lewis SM. Investigation of acute hemostatic failure. In practical hematology 7th edition Churchill livingstone Edinburgh UK 1994.
9. Khan RU. A study of non A non B hepatitis with specific reference to hepatitis E. Pakistan J of health 1993; 30(1): 37-42.
10. Cherqui D, Piedbois P, Pierga JY, Duvoux C, Vavasseur D, Van-Nhieu JT, et al. Multimodal adjuvant treatment and liver transplantation for advanced hepatocellular carcinoma. Cancer 1994; 73(11): 2721-26.
11. Riaz S, Azhar R, Hameed S. Pattern of liver disease at Shaikh Zayed Hospital, Lahore. Pakistan J of pathology 1995; 6(1): 5-8.
12. Taseer IH, Malik IH, Mustafa G, Arshad M, Zafar MH, Shabbir I, et al. Primary hepatocellular carcinoma with high prevalence of hepatitis B virus infection in southern Punjab, Pakistan. JCPSP 1996; 6(6): 293-95.
13. Beazley RM. Pancreatic Cancer. Cancer Surgery 1994; edited by McKenna RJ and Murphy GP: JP Lippincott Philadelphia 105-108.
14. M Waris, M Ayyaz, H J Majid, H R U Islam. Presentation of Cancer of the Gall bladder at Mayo Hospital; Annals of King Edward Medical College,Vol.6 No.2,2000.183-185.
15. Parker RI. Etiology and treatment of acquired coagulopathies in the critically ill adult and child. Critical care clinics 1997; 13(3): 591-609.
16. Robson SC, Kahn D, Kruskal J, Bird AR, Kirsch RE. Disordered hemostasis in extrahepatic portal hypertension. Hepatology 1993; 18(4): 853-857.
17. Sigurdson ER, Cohen AM. Surgery for liver metastases. In Cancer Surgery. Ed. McKenna RJ, Murphy GP. JP Lippincott Philadelphia. 1994;95-97.
18. Cuschieri A. Pathophysiology of the hepato biliary pancreatic system. Essential surgical practice 1995; Ed Cuschieri A, Giles GR 3rd Edition Butterworth Heinemann 101-103.
19. Sherlock S, Dooley J. Haematology of liver disease. In Diseases of the liver and biliary system; 10th Edition, Black Well Scientific Publication 1997;45-47.
20. DeConno F, Polastri D. Clinical features and symptomatic treatment of liver metastasis in the terminally ill patient. Ann Ital Chir 1996; 67: 819-26.
21. Huisse MG, Leclercq M, Belghiti J, Flejou JF et al. Mechanism of the abnormal vitamin K-dependent carboxylation process in human hepatocellular carcinomas. Cancer 1994; 74(5): 1533-1541.
22. Dusheiko GM. Hepatocellular carcinoma associated with chronic viral hepatitis. British Medical Bulletin 1990; 46(2): 492-509.
23. Palascak JE, Martinez J. Dysfibrinogenemia associated with liver disease. J Clin invest 1977; 60: 89-95.
24. Paramo JA, Rocha E. Hemostasis in advanced liver disease. Seminars in thrombosis and hemostasis 1993; 19(3): 184-190.
25. Mayer RJ. Pancreatic cancer. In Harrison's principles of internal medicine. Ed. Fauci AS, Braunwald and Isselbacher KJ. 14th edition McGraw Hill New York 1998; 581-583.
26. Phoa SSKS, Reeders JWAJ, Rauws EAJ, Wit LD, Gouma DJ, Lameris JS. Spiral computed comography for preoperative staging of potentially respectable carcinoma of the pancreatic head. Br J of surgery 1999; 86: 789-94.
27. Kakkar AK, Chinswangwatanakul Y, Lemoine NR, Tebbutt S, Williamson RCN. Role of tissue facytor expression on tumour cell invasion and growth of experimental pancreatic adenocarcinoma. Br Journal of Surgery 1999; 86: 890-94.
28. Iversen N, Lindahl AK, Abildgaard U. Elevated TFPI in malignant disease: relation to cancer type and hypercoagulation. British Journal of haematology 1998; 102: 889-895.
29. Nesvetov AM. Morphological diagnosis of DIC syndrome. Shock liver. Anesteziol Reanimatol: 1997; 27-31.

PATTERN OF DISEASE AND TREATMENT OF ALLERGIC RHINITIS IN KARACHI

EHSAN-UL-HAQ, TARIQ RAFI, KAMALUDDIN KHAN.

ABSTRACT:
This study was conducted on 100 patients visiting Jinnah Postgraduate Medical Centre Karachi, over a period of three years (1996 to 1999) to gain an insight in the problem of nasal allergy in Karachi. It was found that perennial rhinitis is more common in Karachi and the response to treatment with antihistamines and local steroid was much better as compared to antihistamines alone.

KEY WORDS: Perennial allergic rhinitis, Treatment.

INTRODUCTION

Nasal allergy is an altered state of reactivity of body. It is a hypersensitive condition pre-disposed to an antigen-mediated reaction and is a common problem faced by the clinicians. Its presentation differs from person to person and from place to place. In the U.K. hay fever is common while in Karachi, perennial nasal allergy is more common. It has two variants, allergic rhinitis and vasomotor rhinitis, the former being true to its description. Vasomotor is non-infective, non allergic rhinitis.

The external causative substances, to which body shows exaggerated response all-round the year cause perennial allergy while factors found in specific seasons result in seasonal allergic rhinitis. Fluctuation of nasal symptoms in perennial allergic rhinitis is due to proliferation of mite or movement of patients from one place to another. House dust found in furniture, carpets and heavy curtains may be the causative agent. Its incidence is above 20% while about 40% of these patients develop bronchial asthma. In asthmatic patients nasal allergy is an associated feature in about 56% patients.

In England 20% patients with allergic rhinitis develop asthma, while 75% asthmatics have nasal allergy.[1] Seasonal allergy or hay fever is caused by pollens and the severity of symptoms vary according to the number of aeroallergens, which are more in specific seasons and in low-lying areas, as compared to hills. Vasomotor rhinitis, although resembles in presentation to allergic rhinitis, is not due to any identifiable allergen. Symptoms are due to autonomic imbalance. Predisposing factors are emotional stress, drugs, smoking, chemical fumes and changes in temperature.

An allergic reaction is IgE -mediated type I reaction. Delayed reaction starting after 4-12 hours also has its role. Histamine is the major mediator as well as leukotriens, E.C.F.-A, HMW-NCF, PAF, and VIP. Substance found in nasal secretions regulate the IL-1, IL3, IL-5, IL6, WF - ALPHA and IFN GAMMA.[2] In perennial allergic rhinitis, late phase response is important which is caused via cytokines (IL-4, IL-5, IL-6, IL-8, GM-CSF).[3] Level of TNF - alpha also play a role in allergic rhinitis; its level may increase 10 times in allergy as compared to normal people.[4] Histamine which widens the inter-endothelial cell junction causes venular endothelial contraction and its most important effect is vasodilatation.

PATIENTS AND METHODS

Patients were investigated for three major symptoms i.e. nasal blockage, sneezing and rhinnorrhea, as well as minor symptoms like itching and redness of eyes, itching of nose and irritation at palate and ears. Minor symptoms, less than 10 sneezes in a single bout, were not included in this study. Details of nasal secretion were also asked from patients regarding amount, colour, consistency and stuffiness of nose, whether constant or periodic. History of family and personal allergic problems like asthma, eczema and contact dermatitis were ruled out. Place of residence was also noted.

Patients in good health, free from associated systemic diseases were selected. Patients with allergic rhinitis

Correspondence:
Dr. Tariq Rafi, Department of ENT,
Jinnah Postgraduate Medical Centre, Karachi

symptoms of less than two years duration were not included in the study. Pregnant and lactating mothers and patients with history of drug allergy were also excluded. Meticulous clinical examination, anterior rhinoscopy, septum, size of turbinate and colour of mucosa were recorded. In selected cases flexible and rigid endoscope (Hopkin's Rods) was used to confirm the findings. Laboratory investigations were carried out at JPMC Pathological Laboratory, except IgE levels.

Blood was examined for eosinophilia and leucocoytosis. Nasal smears were sent to see the number of eosinophils and nasal scraps to see mast cells. In cases of polypectomy, specimens were sent to laboratory for histopathological examinations. In selected cases IgE levels were assessed. Nasal provocation test, skin or prick tests were not done because of lack of facilities.

RESULTS

Total number of patients were 100, 57 male and 43 female. Oldest patient's age was 59 years and youngest was 7 years. The major presenting symptoms were nasal obstruction (97%) and nasal discharge (92%). The patients with multiple bouts of sneezing, which usually started as they got up early in the morning, were 87%.

Blood examination showed eosinophilia in 45 cases. Nasal smear was carried out in all the 100 patients and in 34 cases marked eosinophils were identified. Out of 64, 21 patients had raised mast cells in the nasal scrap. X-ray PNS in 87 cases were found hazy in 26. In three patients, total IgE level was assessed and was raised in two.

Polypectomy definitely improved the symptoms but SMR had no significant effect on sneezing or rhinorrohea, though obstruction improved and was relieved.

DISCUSSION

Nasal allergy is a problem all over the world. No country, race or age is spared. Its incidence, although not confirmed, is thought to be above 20% in our country.

It varies from country to country and place to place; in the UK hay fever is a hallmark of nasal allergy. In this study only 13% cases had seasonal allergy, majority 87% had perennial nasal allergy. Perhaps the reason is the living style. Mostly people of Karachi, an industrial big city, are living in congested rooms with heavy curtains and carpets, where house dust mite, the causative agent, have favourable conditions to grow. The other common causes in Karachi are the layers of smoke and smog floating in environment, continuously released from public transport. Such scenes can be seen on any working day in the old city where the roads are enshrouded in thick smoke.

In this study 76% people were resident of Karachi; 17% had either shifted from the Punjab during the last two years or were still living there. The problem in the Punjab is relatively seasonal, the commonest cause being dust allergy, specially dust that floats during threshing of wheat crop. Other common allergens are cotton, flowers of mango/orange and animal fodder. In Islamabad, Australian fern was grown on pavements for beautification of roads, but has now become one of the largest source of allergen. Pesticides and chemicals have increased the average production of crops, but are also responsible for allergies.

In vasomotor rhinitis, autonomic imbalance is the cause, although stuffiness, sneezing and rhinorrohea resembles allergy rhinitis, but irritation, redness of eyes and itching is seldom present. Drugs, mental disturbance, personality changes and anxiety are stimulating factors. In this study one patient was found suffering from this condition. Such patients require detailed history and meticulous examination. Nasal mucosa in these patients is usually reddish to blue with excessive secretions. Inferior turbunate is usually hypertrophied. Skin and nasal provocation tests will always be negative.

A number of patients (11%) also had DNS. In 7% cases SMR was done and almost no effect was seen on sneezing or rhinorrohea, although obstruction improved initially. Polypectomy was done in 14% cases, who had nasal polyps. All the symptoms of allergy improved in these cases. In 23 cases of inferior turbinate hypertrophy, nasal cautry was performed; initially it was effective, but after a few weeks patients had to use local decongestant again. Chronic sinusitis was found in 37 cases which were treated with antibiotic. Anthistamine and local steroid were prescribed to 13 cases, but symptoms did not improve and antral washout was required, which had to be repeated two to three times.

The drug used in this study was Fexofenadine. None of the patients were prescribed Macrolides or antifungal treatment. The response of 78% patients was much better in controlling the symptoms, when they used local steroid, in addition to anti histamine. Local Nasal spray, Azelastine, is also reported highly effective to control the symptoms.[6] Azelastine local spray, when used in outdoor patients, in combination with anti histamine tablets, was found to be more effective.[7] Azelastine has better effect as compared to Loritadine.[8] Effects of Terfenadine were better at the onset inefficiency when compared with Cetrizine.[9] Topical corticosteroids are very effective for controlling symptoms.[10] Local use of Budesonide in powder form is also reported effective and well accepted.[11,12] The mechanism of steroid action is anti-inflammatory;[13] no serious side effect was noticed except hangover in the mornings in 2% patients.

This study revealed that nasal allergy can be improved by

different measures, as improvement was noted in 97% patients but the condition cannot be cured. Even using mast cell stabilizer like Ketotifen, one can decrease the severity of sneezing episodes and rhinorrhoea but cannot cure the disease. The role of desensitization is also controversial, although it is used in treatment of nasal allergy.

Allergy rhinitis is a condition and not a disease, which affects the quality of life but it is not life threatening. Pharmacotheraphy and sometimes surgery are helpful in improving the problem.

REFERENCES

1. Bowe-Jones-JM. The link between nose and allergic perennial rhinitis and Asthma. It is the same disease. Allergy 1997; 52(36 Supple) : 20-8.
2. Okamato-Y; Shirotori-Kudo-K; Ishikawa-K Cytokine expression after administration of substance-p human nasal mucosa. The role of substance-p is nasal allergy. J-Immunl; 1993, 151(8): 439-8
3. Naclerio-RM. Pathaphysiology of Perennial allergic rhinitis Allergy : 1997:52 (36 Suppl): 7-13
4. Kato-M et al Elevated soluble tumour necrosis factor receptor levels in seasonal allergic rhinitis patients. Allergy: 1999, Mar; 54 (3): 278-82.
5. Lipert - B.M; Werer-JA Co2 Laser surgery for Hypertrophied inferior turbinate Rhinology 1997.Mar 35 (1) :33-6
6. Wubber-W; Diez- Crespo-C Bahre-M Efficacy and Tolerability of azelastine nasal spray in the treatment of allergic Rhinitis Large scale experience in community practice. Curr -- Med , Res- Opin 1997; 13 (10) : 617-26
7. Weiler- JM; Meltzer- EO Azelastine nasal spray as adjunctive therapy to azelastine tablets in the management of seasonal allergic rhinitis Allergy -- Asthma -- Immunl 1997 Oct; 79 (4) : 327-32
8. AL-Muhaimmed H A, Parallel group comparison of azeleastine and loratidine for the treatment of perennial allergic rhinitis J- Int -- Med -- Res; 1997 July -- Aug; 25 (4) : 175 - 81.
9. Day JH; Briso MP; Clark RH; Ellisa AK; Gravis P. Onset of action and efficiency of terfenadine astemezole cetrizine and loratioline for relief of symptoms of allergic Rhinitis. Ann - Allergy - Asthma - Immunol, 1997 Aug 79 (2) : 163 - 72.
10. Fokkens WJ Allergic rhinitis and inflammation the effect of nasal corticosteriod therapy. Allergy 1997 ; 52 (36 Suppl); 29 - 32.
11. Kokot M Fluticasone propionate in the treatment of perennial allergic thinitis - open multicentre Trial Pol - Mekuriusz - Lek 2000 May, 8 (47) ; 330-4.
12. Soler R et al. A new method of Administration of leudesonide powder for the treatment of allergic rhinitis Acta - Otorhino allerginol. Es. 1997 June - July 48 (5) : 353 - 7.
13. Meletzer EO Pharmacological basis for the treatment of Perennial allergic rhinitis and non-allergic rhinitis with topical corticosteriod. Allergy 1997, 52 (36 Suppl) : 33 - 40.

CAN THE DIAGNOSIS OF ACUTE APPENDICITIS BE SIMPLIFIED?

MOHAMMAD AHMAD, TARIQ MAHMOOD REHAN, MUMTAZ AHMAD, *ANJUM SOHAIL ANSARI, MANSOR AQIL.*

ABSTRACT:
Appendicitis is predominantly a clinical diagnosis. It is important to minimize the number of negative appendectomies but not at the cost of perforation. Thus various scoring systems including Alvarado (which consists of eight features) are used to aid diagnosis. In this prospective study we based our diagnosis on modified Alvarado system.

This study was carried out in Surgical Unit II of Bahawal Victoria Hospital Bahawalpur from January to December, 2000 and patients of all age and sex groups, who fulfill the criteria, were included. Using modified Alvarado Scoring System, score of each patient was recorded. Out of 163 patients admitted, 146 fulfilled the inclusion criteria and were operated. The cases which had appendicular pathology were 71.2%, while only 11.6% had normal appendix. Out of these 146 patients, 121 patients had scored less than 7 with 15 cases of normal appendix. Twenty-five cases had score ≥ 7 with 2 cases of normal appendix. Overall sensitivity of Modified Alvarado Scoring System was only 17.1%. If Modified Alvarado Scoring System is to be used, the two most important features: (migration of pain to the right iliac fossa and tenderness in right iliac fossa) should be given maximum points.

KEY WORDS: Acute Appendicitis, Diagnosis

INTRODUCTION

Acute appendicitis is the most commonly diagnosed abdominal emergency requiring operation.[1] Appendicitis is predominantly a clinical diagnosis.[2] A failure of early diagnosis can lead to progression of the disease with its attendant morbidity and occasional mortality.[3] In many cases, usually during the prodromal phase, its clinical manifestations may be vague and uncertain.[4] Perforation rates range from 4%[5] to 45%[6] and death rates range from 0.17%[7] to 7.5%[8] in various studies. Mortality in children less than 2 years is as high as 20%.[6]

A negative appendectomy rate of 20-44% is not unusual and many surgeons would accept a negative appendectomy rate of up to 30% as inevitable.[9] It is also important to minimize the number of negative appendectomy but not at the cost of perforation. Once perforation occurs, the morbidity and length of hospital stay[10] is increased. Attempts to increase the diagnostic accuracy have included computer-aided diagnosis, imaging by ultrasound scanning and laparoscopy. Various scoring systems have been devised to aid diagnosis.[9,11,12]

Correspondence:
Dr. Dr. Tariq Mahmood Rehan Asstt. Professor of Surgery,
Quaid-e-Azam Medical College, Bahawalpur.

Alvarado first described the most popular diagnostic scoring system in 1988, which consists of eight features with distribution of ten points. But in our setup, the diagnosis was based on modified Alvarado scoring (system). The results were compared with that of Alvarado scoring system. As the diagnosis of acute appendicitis is often made by the junior surgeons its scoring should be made as easy as possible.

PATIENTS AND METHODS

The prospective study was conducted from January to December, 2000 on patients admitted in Surgical Unit II, Bahawal Victoria Hospital with complaints of acute abdominal pain in right iliac fossa.

Using Modified Alvarado Scoring system (Table I), the score of each patient was noted prior to operation, but the score had nothing to do with the management of patients. Left shift of neutrophils was not included (as was described in original Alvarado scoring system) due to technical laboratory errors.

All patients were operated and the condition of appendix was noted in each patient. In doubtful cases, the diagnosis was confirmed by histopathology.

TABLE-I MODIFIED ALVARADO SCORE

Scoring feature	Points (if present)	Male	Female	Total Patients
Migration of pain to right iliac fossa	1	86	60	146
Anorexia	1	39	31	70
Nausea/Vomiting	1	49	37	86
Tenderness	2	86	60	146
Rebound tenderness	1	46	33	79
Fever (≥ 37.50 C)	1	31	32	63
Leucocytosis (>10,000x10^{12}/ litre)	1	21	14	35

ACTION

<4 = exclusion; 5-6 = monitoring; > 7 = operation

RESULTS

A total of 163 patients with complaint of pain in right iliac fossa were admitted in Surgical Unit II, J.P.M.C. Out of these 146 patients, who fulfilled inclusion criteria, were operated. Eighty-six (58.6%) were male and 60 (41.1%) female with a ratio of 1.3:1, mean age of the patients was 22.9 years; range 4 to 69 years. On exploration 129 cases had appendicular pathology; 71.2% had inflamed/highly-inflamed appendix, 17.2% had advanced appendicitis with 15.8% perforated and 1.4% gangrenous appendix. Only 11.6% cases had normal appendices; revealing other pathologies (Table II).

Out of 146 patients, 121 (82.88%) had modified Alvarado

TABLE-II OPERATIVE DIAGNOSIS OF 146 PATIENTS

Diagnosis on Exploration		No. of patients	%
Acute Appendicitis	Highly Inflamed	104	71.2%
	Gangrenous	23	15.8%
	Perforated	2	1.4%
		129	**88.4%**
Mesenteric Lymphadenitis		5	3.4%
Ovarian Pathology		4	2.7%
PID		3	2.1%
UTI		1	0.7%
No Pathology		4	2.7%

score < 7 with only 15 (12.4%) cases of normal appendix (p-value 0.0001) and only 25 (17.12%) had score ≥ 7 with 2 (8%) cases of normal appendix p-value 0.0001).

Incidence of advanced appendicitis was 13% in patients with score <7 and 36% in patients with score >7. Overall sensitivity of the modified Alvarado score was only 17.1%.

The excluded patients were 17 or 10.24% who did not fulfill the criteria. The rest were investigated further (i.e. other scoring features, USG, blood picture and urinalysis) and 2(1.19%) found to have acute appendicitis and operated.

DISCUSSION

The principal objective of the clinical decision process is to make an early and correct diagnosis of acute appendicitis with maximal economy of resources.[10] The indication for operative treatment is normally based on clinical examination.[11] The diagnosis, operation and management of a patient with acute appendicitis can be made without investigations on careful history, physical examination and urinalysis.[3] Urinalysis should exclude UTI. Slight elevation of white cells in the urine could be due to the inflammatory process of acute appendicitis near the urethra or bladder.[13] Moreover, elevated temperature and leucocyte count will shift to left or remain non-specific and often absent in patients with acute appendicitis.[14]

All the 146 patients in this study had undergone appendectomy and were included on the basis of migration of pain and tenderness to right iliac fossa. The objection to the choice of this criteria is that all patients with acute appendicitis may not give history of shifting of pain (20-50%). But other diagnostic indicants (like anorexia, vomiting, temperature, pointing sign, Rovsing's sign, psoas or obturator sign and pain in RIF on coughing) do help. Complete blood picture, urine examination, USG and laparoscopy also support the diagnosis.

In this study only 1.19% of the cases were not diagnosed on simple criteria; 129 (88.4%) (78 male and 51 female) proved to have acute appendicitis. The negative appendectomy rate was 11.6%, which is comparable to the negative appendectomy in studies by Ohman[11] (21%), Gurleylike[15] and Fenyo[16] 17.5% each. Negative appendectomy rate was 5.5% in males and 6.2% in females (p-value 0.0002). Utilizing modified Alvarado score, out of 121 patients with score < 7, only 12.4% had undergone negative appendectomy. Surprisingly, incidence of advanced appendicitis was 13.2% in patients with score<7 and 36% in patients with score ≥ 7.2 (8%). Patients with score ≥ 7 did have a normal appendix. On the basis of modified Alvarado Scoring system, 90(61.64%) patients of appendicitis could be missed due to low score. So in this study it seems that there are some flaws in the scoring system and the specificity of Alvarado scoring system is doubtful. We think that it is due to unreasonable distribution of points for each feature of scoring system, e.g. shifting of pain and tenderness in right iliac fossa have been allocated one and two points respectively, in comparison to inconsistent features like anorexia, leucocytosis and raised temperature. We think that if six out of eight points are allocated to shifting of pain and tenderness in right iliac fossa, the specificity of scoring system can then be increased.

Errors in diagnosis are most common in young women and at extremes of age[3]. The main difficulty in assessment remains in women of childbearing age. It would be of interest to know if the additional use of ultrasonography

(USG) and laparoscopy could reduce the appendectomy rate in this group[17]. USG examination is interfered by the presence of bowel gas and is dependent on the experience of sonologist type and resolution of the machine. Results from randomized and non-randomized trials suggest that there is little difference between conventional open and laparoscopic appendectomy in terms of operation time and patient recovery[3]. No doubt, laparoscopy can help a lot in the diagnosis of acute appendicitis but overall expenses of laparoscopy and need of general anaesthesia make laparoscopy a less favourable examination, but if combined with laparoscopic appendectomy, it can be beneficial. Laparoscopic appendectomy becomes more costly if stapling devices are used and early appendicitis may be missed with this technique[2]. Moreover, in one study, computer-aided diagnosis to avoid negative appendectomy in suspected appendicitis offered no advantage over unaided clinical diagnosis[18].

The study demonstrates that modified Alvarado Score is substantially inferior to the current clinical practice in diagnosis of acute appendicitis[19] as 90 cases of appendicitis were noted to have score < 7 and 9 cases of normal appendix had score ≥ 7.

We conclude that no foolproof scoring system is available till now which can give early and 100% correct diagnosis of acute appendicitis. Yet one should continue to evolve the given diagnostic tools to minimize the chances of negative appendectomy. Keeping in view the large number (74%) of patients with score less than 7 who did have appendicitis, there is no evidence that conservative treatment of acute appendicitis, as an alternate to surgery, should be more widespred3 and a scoring system cannot be substituted for a surgeon's experience.[20]

Those patients who do not suffer from shifting pain in RIF or tenderness in RIF should be investigated further to exclude the possibility of acute appendicitis. Moreover, if modified Alvarado scoring system is to be used, the two most important symptoms and sign (shifting of pain and tenderness in right iliac fossa) should be given maximum points, (i.e. 3.) to make at least 50% of the total score.

REFERENCES

1. Temple CL, Huch,Croft SA, Temple WJ. 'The natural history of appendicitis in adults: a prospective study'. Ann Surg 1995; 221:277.
2. Imber C, Glazer G. 'Management of peritonitis with special reference to appendicitis'. Surgery Inten 199; 47: 253-257
3. Mackin CP, Merei JM et al. 'A prospective evaluation of the modified Alvarado score for acute appendicitis in children'. Ann R Coll Sur Engl 1997; 79: 203-205.
4. Lewis FR, Holcroft JW et al 'Appendicitis: a critical review of diagnosis and treatment in 1,000 cases'. Arch Surg 1975; 110: 677-684.
5. De Bombal FT, Leaper DJ. Et al 'Human and computer-aided diagnosis of abdominal pain: further report with emphasis on the performance of clinicians'. Br Med J 1974; 1:376-380.
6. McFee AS, Rogers W. 'Diagnosing appendicitis in the paediatric patients'. Infections in Surgery 1982; June: 42-49.
7. Detmer DE, Nerers LE, Sikes ED. 'Regional results of acute appendicitis care'. JAMA 1981; 246: 1318-1320.
8. Ackerman NB 'The continuing problems of perforated appendicitis'. Surg Gynae Obstet 1974; 139: 29-32.
9. Hofman J, Rasmussen O. 'Aids in the diagnosis of acute appendicitis'. Br J Surg 1989; 76: 774-90.
10. Robert T, Wilcox, L. William Traverse 'Have the evaluation and treatment of acute appendicitis changed with new technology'. Surg Clin North Am 1997 (Dec); 77 (6): 1 355-70.
11. Ohmann C, Yang Q, Franke C. 'Diagnostic scores for acute appendicitis. Abdominal Pain Study'. Eur J Surg 1995; 161:273-81.
12. Senbanjo RO. 'Management of patients with equivocal signs of appendicitis'. J R Coll Surg Edinb 1997 (Apr); 42(2): 85-8.
13. Shackelford RT. 'Diagnosis of surgical disease'. Vol 2. Philadelphia, WB Saunders, 1968, 1306-1309.
14. Ducholm S, Bagi P, Bud M. (1989). 'Laboratory aid in the diagnosis of acute appendicitis'. 32: 855-9.
15. Gurleyik E, Gurleyik G, Unalmiser S. 'Accuracy of serum C-reactive protein measurement in diagnosis of acute appendicitis compared with surgeon's clinical impression'. Dis Colon Rectum 1995 Dec; 38(12): 1270-74.
16. Fenyo G, Lindeberg G, Blind P, Enoch sson L, Oberg A. 'Diagnostic decision support in suspected acute appendicitis: validiation of a simplified scoring system'. Eur J Surg 1997 Nov; 163(11): 831-8.
17. Owen TD, William H, Stiff G et al. 'Evaluation of Alvarado score in acute appendicitis'. J R Soc Med 1992; 85(Feb): 87-8.
18. Van Way III CW, Murphy JR, Dunn EL et al. 'A feasibility study of computer-aided diagnosis in appendicitis'. Surg Gynecol Obstet 1982; 155: 685-88.
19. Macklin CP, Merei JM, Radcliffe GS, Stringer MD. 'A prospective evaluation of the modified Alvarado scores for acute appendicitis in child ren'. Ann R Coll Surg Engl 1997; 79: 203-205.
20. Barber MD, Mc Laren J, Rainey JB. 'Recurrent appendicitis'. Br J Surg 1997; 84: 110-112.

ILEAL TYPHOID PERFORATION AN EXPERIENCE WITH 42 CASES

HALEEM TAJ, ARSHAD MEHMOOD, ABDUL SALAM, *DAWOOD SHAH.*

ABSTRACT:
This study was carried out from January 1997 to December 1998 in Surgery Department of Sandeman Provincial Teaching Hospital Quetta, on 42 patients hospitalized with ileal typhoid perforation. The majority of patients (80.95%) were between 11-40 year of age with peak incidence in second and third decade and male to female ratio of 3.2:1. Majority of patients came within 48 hours and laparotomy was done in all. Single perforation was found in 39 (92.85%) and multiple perforation in 3 (7.14%) patients. In 35 (83.33%) patients primary closure and peritoneal toilet done while in 7 patients (19.05%) primary repair with proximal loop ileostomy with peritoneal toilet was done. Ileostomy was closed 2-1/2 months after the first surgery. Patients with longer duration of perforation had increased morbidity and mortality. Most of the patients remained admitted for 10 days their average hospital stay being 14 days with minimal stay of 7 days and maximum stay 30 days. The overall mortality was attributed to duration of perforation and condition of patient.

KEY WORDS: Enteric fever, Typhoid perforation, Ileostomy.

INTRODUCTION

Typhoid fever is still a major health problem in developing parts of the world[1,2] with an estimated 540 per 100,000 cases. One of the complications of typhoid fever is ileal perforation.[2,4,5,6] Peyer's patches of terminal ileum are involved with formation of longitudinal ulcerating lesion on the antimesenteric border within 45 cm of ileocaecal junction.[6] Typhoid perforation is solitary in 85% of cases[6,7] and accompanied by peritonitis with little tendency for localization by adjacent abdominal organ.[6] The incidence of perforation is variable being especially high in West Africa (15-33%) and low in Iran and Egypt (1-3%). This high incidence has been attributed to late diagnosis and existence of virulent strains of salmonella typhi.[7] Over 80% of patients are between first and third decades of life.

The usual symptoms are fever and abdominal pain.[8] The diagnosis of typhoid ileal perforation is usually based on history, physical examination, radiology and operative findings. Treatment of typhoid perforation requires surgical intervention after resuscitation, correcting fluids and electrolyte in balance and antibiotic cover. Peritoneal toilet with primary closure of perforation is the standard treatment.[9,10,11] Closure of the perforation with side to side ileo transverse anastomosis can also be done.[11] This disease carries high morbidity rate of 55.4% and mortality rate of 1-39.3%. Late presentation, delay in operation, multiple perforations and drainage of copious quantities of pus and fecal material from peritoneal cavity are the determinant factors of increased morbidity and mortality.[12]

Correspondence:
Dr. Mohammad Haleem Taj, Assistant Professor of Surgery,
Bolan Medical College, Quetta.

PATIENTS AND METHODS

This study was conducted on 42 patients who underwent surgery for ileal typhoid perforation in department of Surgery, Sandeman Provincial Teaching Hospital, Quetta during the period from January 1997 to December 1998. All patients were admitted through casualty department. The diagnosis was based on history, clinical examination and radiological findings. The data so obtained was recorded on proformas and results were evaluated. All the patients were resuscitated before surgical intervention by the administration of intravenous fluids and antibiotics, nasogastric decompression and catheterization. Routine laboratory investigations and Widal test were performed.

After pre-operative management, emergency laparotomy was performed on all patients through midline incision. Peritoneal toilet was done in all cases and then following procedures were carried out where appropriate:
Primary closure of the perforation only and primary closure of perforation with proximal ileostomy or exteriorization of ilium as ileostomy.

RESULTS

A total of 9500 patients were admitted in surgical Department during the study period out of which forty-two patients were with ileal typhoid perforation. Among these forty-two 32 (76.19%) were male. Majority of patients belonged to poor families. Their ages ranged from 1 to 80 years with mean age of 34 years. Table (I).

TABLE-I AGE DISTRIBUTION

Year	No. of Patients	%
11-20	12	28.57
21-30	16	38.10
31-40	06	14.29
41-50	03	07.14
51-60	02	04.76
61-70	02	04.76
71-80	01	02.38

The peak incidence of ileal typhoid perforation was observed during months of June and August.

Majority of patients n = 35 (83.33%) came within 48 hours of perforation while remaining 7 (16.67%) came after 48 hours. The clinical presentation associated with ileal typhoid perforation is depicted in table-II.

TABLE-II CLINICAL SYMPTOMS & SIGNS

Features	No. of Patients	%
H/O Fever	42	100
Abdominal Pain	42	100
Vomiting	30	71.43
Abdominal distension	25	59.52
Constipation	28	66.67
Abdominal Tenderness	41	97.62
Abdominal rigidity	39	92.86
Dehydration	30	71.43
Electrolyte imbalance	31	73.81
Gas Under diaphragm	35	83.33

All base line investigations were performed. TLC ranged from 6200 to 20,000/Cu mm. Widal was positive in 83.33% cases, blood culture positive for salmonella in 23.81 % cases and gas under diaphragm were positive in 83.33% patients.

At laparotomy 39 (92.85%) patients had single perforation while remaining 3 (7.15%) had multiple perforation. All perforations were present at the antimesenteric border. Most perforations (92.85%) were circular and the most proximal was situated 30 cm from the ileocaecal junction. Primary closure was done in35 cases (83.33%) and ileostomy in 7 case (16.67%). In cases managed with primary closure 5 patients died. Three patients died because of fecal fistula formation, two developed multiorgan failure. Two cases, other than those that expired, had fecal fistula, they however got better after conservative management. In patients who under-went ileostomy no death occurred. The mortality rate was 11.9%. The complications in this study were: wound infection 59.52%, wound dehiscence 30.95%, intra abdominal abscess 9.52% and fecal fistula in 11.9% patients.

DISCUSSION

Typhoid fever is still a major health problem in developing parts of the world. The most lethal complication of typhoid fever, ileal perforation[1,2,3,4,5] is still common in the Third world countries with high mortality rate.[7,13] The overall frequency of intestinal perforation in typhoid fever is 3%.[3]

Many therapeutic failures and complications are due to emergence of more virulent and resistant strains of salmonella speies.[14,15,16] In some centres a peak during rainy season has been reported.[7,14,17] This is probably due to contamination of water and food products. In our series maximum typhoid ileal perforation seen in the second and third decade which is consistent with other studies from Africa,[38] Indopak[6] and Europe.[7] Perforation occurs most commonly in 2nd and 3rd weeks of illness.[17,19,20] Majority of patients in this study developed ileal perforation in second week of illness. The presenting symptoms correspond well with the findings of other authors.[18,21,22] Leucocytosis was present in most cases with an average of 12500/ cu mm. Although Leucopenia has been reported to be associated with typhoid fever,[23,24] but in the presence of sepsis and severe peritonitis, tends to modify this picture by causing leucocytosis.[9,25,26] The percentage of positive pneumoperitoneum in our study is quite similar to those reported by many studies which is 62.2%[17] and 78%,[27,28] All the patients were subjected to emergency laparotomy under general anesthesia after vigorous resuscitation and hemodynamic stability as surgical management is preferred by most recent series.[17,25,29] The perforation was always located at the end of illium[1,3,6,7] and was single in 92.86% of cases and multiple in 7.14% cases, so incidence of multiple perforations in our study is slightly less than other studies.[3,7,29] All the perforations were present on the antimesenteric border of terminal ilium mostly, within 10-30 cm from the ileocaecal valve.[30] The average size of perforation was about 7 mm while reported average size is 5 mm. Moderate to severe peritoneal contamination was seen in most of our cases, probably due to delayed referral by general practitioners and quacks.

In this study primary closure (wedge resection and closure of the perforation in two layers)[6] were performed in 35 (83.33%) patients.[9,30,31] In our study5 out of 35 cases operated by primary closure developed fecal fistula with an incidence of 11.90%. The incidence of fecal fistula varies from 40%[24] to 20%.[32] In 7 patients (16.67%) protective loop ileostomy done.[30,31] A protocol of protective

loop ileostomy in all patients presenting with moderate to severe peritoneal contamination and perforation of more than 48 hours duration and perforation situated within 10 cm of ileocecal junction is maintained. So morbidity and mortality has decreased due to above protocol. The main objection against ileostomy has been the need for the second operation with its own morbidity and mortality but in our experiences ileostomy closure has no major complication. Ileostomy closed 2-1/2 month after first surgery.

Mortality rate after primary closure for the patients with typhoid ileal perforation is between 1 and 39.3[1,7] but in this study the mortality after primary closure was 11.90%, but no mortality was observed in cases where protective loop ileostomy was done. In cases of loop ileostomy, fluids started by mouth after 2 days of surgery.

REFERENCES

1. Noorani MA;Sial I;Mal-V.typhoid perforation of small bowel a study of 72 cases. J-R Coll-Surg-Edinb. 1997 Aug 42 (4) 274-6.
2. A Meh EA, Dogo-PM, attah-MM Nmadu PT. Complications of Three operation for typhoid perforation Br - J - surg 1997 Apr : 84(4): 558-9.
3. Van - Basten - JP;Stockenbrugger - R. Typhoid perforation. A review of literature since 1960, Trop - Geogr - Med. 1994; 46 (6): 336-9.
4. Jemni L et al. Complication of typhoid fever. Med. - Tropic. Mar-Apr 1989; 49(2) 189-91.
5. Kizilcan F et al. Complication of typhoid fever requiring laparatomy during childhood, J-Pead-Surg Nov 1993 28(11) 1490-3.
6. Cuscheri A, Gile GR, Moosa AR. Essential surgical Practice, 3rd edition Butterworths-Heinemann, London 1995: 1321.
7. Gibney EJ. Typhoid Perforation. Br J Surg. 1989; 76: 887-9.
8. Meier DE, Tarplly J.L. typhoid intestinal perforations in Nigerian children. Worel-J- Surg. 1998 Mar; 22(3): 319-23.
9. Akhter AT. Primary Closure compard with ileostomy in case of typhoid perforation. JAMA-J. Pak Sep 93.
10. Akoh AJ. Prognostic factor in typhoid perforation. East Afr Med. J 93 70 (1) 18-21.
11. Pak - DK Evaluation of best surgical procedure in typhoid perforation an experience of 60 cases. Trop - Doct. 1998 Jan; 28 (1): 16-8.
12. Adesunkanmi AR, Ajeco OG. Prognostic factors in typhoid ileal perforation a prospective study of 50 patients. J.R. Coll Surg.Edinburgh, 1997 Dec 46 (6): 395-9.
13. Santillana M. Surgical Complication of typhoid fever: enteric perforation world J surg 1991; 15: 170-5.
14. Adesun Kanmie AR; Ajao OG. Typhoid ileal perforation: value of dehayed primary closure of abdominal wound1; Afr-J- Med. -Sci 1996 Dec;25 (4): 311-5.
15. Archampong EQ. Typhoid ileal perforation: why such modalities Br. J. Surg: 1976: 63:315-7.
16. Yousaf M, Sadik a. Ofloxacin in treatment of typhoid fever unresponsive to chloramphanicol clin, therap. 1990 12: 44-7.
17. Rathore AH; Khan, IA, Sagheer W, Prognostic indeces of typhoid perforation, Ann - Trop - Med- parousitogy June 1978 81(3) 283-9.
18. Ojao OG. Abdominal emergencies in a tropical African population Br. J. Surg. 1981; 68: 341-2.
19. Ndububa DA Erhabor EG, Akinola DO. Typhoid and paratyphoid fever, a retrospective study, Trop-Gastroenterol 1992 Apr-Jun; 13(2): 56-63.
20. Vogal-H et al. Intestinal perforation in Juvinile abdominal typhoid: Rontgenvlatter Mar 1993, 36 (3) 75-8.
21. Akgum Y; Bacr B; Boylu-S;Abam N; Tacyldiz I, typhoid enteric perforation Br. J surg 19959 Nov;28(11): 1512-5.
22. Maqbool S, Hashmi A, Fasih S, Iqbal J, Typhoid fever in children, proceedings of Sheikh Zaid Post Graduate Medical Institute symposium 1993; 7: 525.
23. Mock CN, Amaral J, Visser LE. Improvement in survival from typhoid perforation. Ann. Surg. 1992; 215: 244-9.
24. Nguyen VS, Typhoid perforation in tropics. Apropos of 83 cases J-Chir - Paris, Feb 1994; 131 (2): 90-95.
25. Gupta. V et al. Perforated typhoid enteritis in Children. Postgraduate Me. J-Jan 1994, 70 (819) 19-22.
26. Paredes C, Cruz J, Diaz Plasercia J Prevost M. Prognostic factor in typhoid perforation; Rev-Gastroenterol peru; 1993; 13(1): 13-19.
27. Badejo QA, Arigbabu AD, Operative treatment of typhoid perforation with peritoneal irrigation a comparative study, Gut; 1980; 21:141-45.
28. Nadkarnni Km; s hetty SD, Kagzi RS, Pinto Ac, Bhaderao-RA. Small bowel perforation, a study of 32 cases. Arch-Surg 1981; 116:53-7.
29. Abantanga FA. Complications of typhoid perforation of the ilium in Children after surgery. East-Afr - Med- J. 1997 Dec 74(12) : 800-2.
30. Iqbal SA, Sial KH; Memon MM. Typhoid perforation of bowel Pak-J surg Apr-Jun 1994 Vol (10) :2.
31. Naseer T. Complications and management of Typhoid fever specialist Aug 1994.
32. Singh CP, Singh N, Bar GK, Lal G, Kumar H Efficacy of Cipiofloxacen and Norfloxacin in multi drug resistant enteric fever in adults J-2nd -Med - Assoc. Jun 1993; 91(6) 156-7.

ANXIETY AS A PSYCHOLOGICAL RISK FACTOR IN THE DISORDERS OF CARDIOVASCULAR SYSTEM

SEEMA MUNAF, GHAUS-UR-REHMAN.

ABSTRACT:
The present study is aimed at emphasizing the role of anxiety as a psychological risk factor in the development of cardiovascular disorders. It was hypothesized that "mean trait anxiety" scores of cardiac patients will be more as compared to non-cardiac patients. Fifty male cardiac patients were selected from the in-cardiac patients and fifty non-cardiac patients were selected to match the variables of age, sex, residential area and education. Their levels of anxiety were measured through trait scale of STAI and TAT cards. ANOVA was applied to get the statistical significance of the results. The findings suggest that the mean trait anxiety scores of cardiac patients were significantly higher as compared to the non-cardiac patients.

KEY WORDS: Cardiovascular systems, Disorders, psychological risk factors

INTRODUCTION

The term psychosomatic is derived from the Greek words psyche (mind) and soma (body). Literally, psychosomatic refers to mind body relationships. As traditionally defined, psychosomatic disorders are physical illnesses whose development and expression have been significantly influenced by psychological and cultural factors as well.[1]

Studies in psychosomatic medicine have broadened in scope and now range from global evaluation of emotion, stress and sickness to unraveling particular relations between emotions and body responses or conditions. It has been recognized that emotional states, especially when they persist over long periods, can profoundly affect various organs of the body, sometimes directly damaging them and in others predispose them to infections or failure from precipitating causes.[2] Emotions and some particular traits are also believed to be contributory in the general sense to emotions that can have a direct effect on the body's immune system and increase susceptibility to diseases.

Many psychological and sociological variables have been examined in studies of behavior patterns related to heart diseases. Probably the most significant personality type encountered in the stress theory is that of the Type 'A' personality. The Type 'A' personality has the following traits:

- Sustained drive towards poorly defined goals.
- Hostility and competitiveness
- High level of ambition.
- Preoccupation with work and deadlines.
- Irritability and low tolerance of frustration.

A number of studies have supported the hypothesis that Type 'A' people are more prone to heart diseases.[3]

Type 'B' people by contrast don't have these characteristics and are able to unwind and relax. Type 'B' individuals are able to relax, without feeling guilty and work without becoming agitated. They lack sense of urgency, with its accompanying impatience. Feelings of hostility are not easily aroused in these individuals and they show little need to display or discuss achievements. High level of epinephrine and norepinephrine in the blood during stress has also been noted in the Type 'A' people.[4]

The somatic manifestations of anxiety differ greatly in intensity and duration, depending on the type of anxiety, disorder and the presence or absence of stress. Moreover, the subjective experience of somatic manifestations correlates poorly with actual physiologic states and reactions.[5] Thus, patients who complain of palpitations may show only small changes in heart rate on exposure to psychological stress, while others with

Correspondence:
Dr. Seema Munaf, Associate Professor, Institute of Clinical Psychology, University of Karachi,

minimal complaints may exhibit considerable changes. Psychic anxiety, specifically the tendency to worry and to anticipate harmful events, appears to be independent of the somatic manifestations of anxiety.[6] Thus, patients who experience comparable levels of psychic anxiety may differ greatly in the type and severity of accompanying physical symptoms. This is particularly true for milder forms of anxiety.

Perhaps the best known example of anxiety is the feeling of fright. This is the acute anxiety experience in responses to a sudden situation perceived as dangerous. Fright is accompanied by an increase in heart rate, systolic and diastolic blood pressures, skin conductance and muscle tension. Such a transitory anxiety reaction may be psychologically upsetting, but is of little physical consequence to a healthy person. In those who have cardiovascular illness, however, sudden fright may have serious consequences.[7,8]

Quantitative overload is the condition of having too much work to do in the available time. It is an obvious source of stress and clearly linked to stress related diseases. For example, a study of young heart attack patients found that 70 percent of them were working more than 60 hours per week. This is supported by correlation between work overload and heart disease as well as relationship to excessive drinking and low self esteem.[9]

The personality variable of internal or external controls also influence an individual's reaction to stress. Persons rated high on internal control believe that they can control the forces and events that shape their lives. Those scoring high on external control believe that life is determined by events and forces beyond their control.[10] Apparently, the belief that one is in control of one's life significantly reduces the effects of stress.[11]

Corporation executives have high stress position. It is generally believed that executives have a great deal of stress at work and consequently, have a much higher rate of heart attacks than the general population. This too is not true. High level executives have 40 percent fewer heart attacks than middle level managers, who are assumed to work under less stressful conditions.[12,13,14]

PURPOSE OF STUDY

The purpose of the present study is to investigate whether Trait Anxiety, contributes in the development of cardiac disorders or not. Trait anxiety ('A' Trait) refers to relatively stable individual differences in anxiety proneness, that is, to differentiate between people in the tendency to respond to situations perceived as threatening with elevations A-State intensity.

Through the survey of literature we have found that emotions and personality traits play an important role in the causation of various physical illnesses including cardiac. Admittedly, the available researches cannot be truly representative because of limited similar populations. Our research will be the first one of its kind in Pakistan, emphasizing the above-mentioned psychological risk factors.

This study is unique in the sense that the data has been directly collected from the modern cardiac units of hospitals at Karachi. This representative sample was easily available as the patients from other provinces also seek treatment in this city.

This research will be of sufficient help to cardiologists in their recommendations. It will also be helpful to psychiatrists, psychologists, and social workers to re-educate the general masses regarding the psychological risk factors contributing to cardiac diseases.

PATIENTS AND METHODS

Fifty male cardiac patients between 30 and 60 years of age were selected from the indoor patients of cardiac units of hospitals at Karachi. They were selected in five groups, 10 patients each with Angina pectoris (AP), Myocardial infarction (MI), Angioplasty (minimum of two coronary arteries blocked - (pre-bypass group), coronary arteries bypass grafting, CABG - (post-bypass group) and those who were not recommended for any invasive procedure (treatment group).

These five patient groups i.e. angina pectoris, myocardial infarction pre- bypass, post-bypass, and treatment group were collectively called Cardiac disease categories. Each group's disease status is different and hence their problems are different and we had a variety of patients in our sample, selected patients were those who were admitted in the hospital for emergency treatment but were not in the intensive care units and were clinically diagnosed as cardiac patients after prolonged invasive (medical) procedures (Experimental Group).

In another group fifty males participants from Karachi who matched with the experimental group on the variables like age, sex, residential area and education were selected as Control Group or Non cardiac patients. We termed them as patients because they may be suffering from some physical disease, other than cardiovascular disorder. The participants in this sample did not have any established or clinically diagnosed cardiovascular disorder.

The "Slosson Oral Reading Test" (SORT), was first administered to all the selected participants and only those were included in the sample who successfully completed the sixth grade reading level to make sure that

all the participants understood the English language of the State Trait Anxiety inventory.[16] Before and after the administration of "SORT", the participants willing consent was sought as to whether they were willing to participate in the study or not.

The entire testing procedure was divided into two phases. The first phase took place in the morning session. After necessary establishment of rapport with the patients, a structured clinical interview was conducted to elicit information required for this research. This procedure was adopted keeping in view firstly; the patients poor health condition and secondly, to abide by the hospital administration's instruction "not to over stress the patients." A wide variety of information was collected through this interview. The interview lasted for 30-40 minutes for each patient. Care was exercised that the patients remain comfortable. They were asked not to sit but remain in resting position on their beds.

In the second phase, after four hours long break, (evening session) , a Trait Anxiety scale of STAI was administered. For patients' convenience, the instruction of the STAI was written at the top of the questionnaires. It was followed by administration of TAT card No.11 and card No.15. For TAT administration, the standard instructions were orally delivered in the following manner: this is a test of imagination. You will develop a story for each picture shown to you at a time, keeping in view, "What led up to the present situation? What are the characters thinking and feeling? What would be the final outcome of the story? You have five minutes to complete a story, do you understand what I mean, if not, I shall repeat" After the instructions were delivered, it was made sure that the patients understood what the examiner aimed at.

After collection of data for both cardiac and non-cardiac patients from all Cardiac Units and Cardiovascular Institutes of Karachi, the test sheets were scored. The Trait Anxiety Scale of STAI was scored according to the manual's directions.

TAT responses were measured through five point rating scale as under:
1= Very low, 2= Low, 3= Moderate, 4= High, 5= Very High

In order to assess the inter-rater reliability for TAT scoring, a sample of ten patients was selected. Every tenth case was selected and rated by another trained and qualified rater. The person-product moment correlation coefficient was computed for all the variables.

The mean scores of Trait Anxiety, of cardiac and non-cardiac groups were calculated. The differences between the mean score for both the cardiac and non cardiac patients were tested by the t-test in order to inquire into statistical significance of the differences. To test further whether the cardiac disease categories differ significantly among each other, one way analysis of variance (ANOVA) was applied.

RESULTS

The difference of Mean Trait Anxiety between the cardiac patients and non-cardiac patients when A-Trait scale of STAI was administered is shown in Table I.

TABLE-I T-TEST SHOWING THE DIFFERENCE

Groups	Mean	df	T	P	Level of Significance
Cardiac Patients (N=50)	47.24	98	*2.79	**P<0.1	Significant
Non cardiac Patients (N=50)	44.74				

Note:
** = P<.05, one tailed*
***=P<.01, one tailed*

T-Test: showing the difference of Mean Trait Anxiety between the Cardiac Patients and Non-cardiac Patients when TAT was administered is shown in Table II.

TABLE-II T-TEST SHOWING THE DIFFERENCE WHEN TAT WAS ADMINISTERED

Groups	Mean	df	T	P	Level of Significance
Cardiac Patients (N=50)	2.64	98	*2.76	**P<.01	Significant
Non cardiac Patients (N=50)	2.08				

Note:
** = P<.05, one tailed*
***=P<.01, one tailed*

The analysis of variance showing the difference of Mean Trait Anxiety among five groups of Cardiac Patients when A-Trait Scale of STAI was administered is shown in Table III.

TABLE-III ANALYSIS OF VARIANCE WHEN A-TRAIT SCALE WAS ADMINISTERED

Source of variance	SS	df	MS	F	P	Level of significance
Between Disease Categories	233.32	4	58.33	*3.74	**P<.05	Significant
Within Disease Categories	701.8	45	15.59			
Total	935.12	49				

Note:
** = P<.05*
***=P<.01*

An analysis of variance showing the difference of Mean Trait Anxiety among five groups of Cardiac patients when TAT was administered is shown in Table IV.

TABLE-IV ANALYSIS OF VARIANCE WHEN TAT WAS ADMINISTERED

Source of variance	SS	df	MS	F	P	Level of significant
Between Disease Categories	4.12	4	1.03	*6.25	**P<.05	Significance
Within Disease Categories	7.4	45	.16			
Total	11.52	49				

Note:
** = P<.05*
***=P<.01*

T-TEST: showing difference of Mean Trait Anxiety between five groups of Cardiac patients when A-Trait Scale of STAI was administered is shown in Table V.

TABLE-V T-TEST SHOWING DIFFERENCE WHEN A-TRAIT SCALE OF STAI WAS ADMINISTERED

Types of Patients Mean Trait Score (%)	Myocardial Infarction $\bar{Y}$ = 45.4	Pre by Pass $\bar{Y}$ = 46.0	Post by Pass $\bar{Y}$ = 45.9	Treatment Group $\bar{Y}$ = 47.6
Angina 51.3	**3.197	*2.319	**3.068	1.881
Myocardial Infarction 45.4	-	-.324	-.4382	-1.527
Prebypass	-	-	.0566	-.810
Post bypass 45.9	--	-	-1.278	
Treatment Group 47.6	-	-	-	-

Note:
** = P<.05*
***= P<.01*

T-TEST showing difference of Mean Trait Anxiety between five groups of Cardiac patients when TAT was administered is shown in Table VI.

DISCUSSION

The hypothesis that "The Mean Trait Anxiety score of cardiac patients will be more as compared to Non-cardiac patients" is supported by the data and is significant at P<.01 level.

According to Table No. I when Trait Anxiety was measured through A-Trait scale of STAI and Table No. II when TAT cards were used it is quite clear that the mean Trait Anxiety of cardiac patients was significantly high, as compared to non-cardiac patients.

Results of the analysis of variance are shown in Table

TABLE-VI DIFFERENCE OF MEAN TRAIT ANXIETY WHERE TAT WAS ADMINISTERED

Types of Patients Mean Trait Score (%)	Myocardial Infarction $\bar{Y}$ = 2.8	Pre by Pass $\bar{Y}$ = 2.9	Post by Pass $\bar{Y}$ = 2.8	Treatment Group $\bar{Y}$ = 2.1
Angina 2.6	-.7748	-1.0533	-.2581	2.035
Myocardial Infarction 2.8	-	-.4472	0	0
Prebypass 2.9	-	-	.4472	**3.8948
Post bypass 2.8	--	-	-1.278	
Treatment Group 2.1	-	-	-	1.328

Note:
***= P<.01*

No. III when Trait anxiety (A-trait Scale) was administered and in table No. IV when TAT cards were used, this explains that there is a significant difference in the level of trait anxiety among the cardiac disease categories of angina, myocardial infarction (MI) pre-bypass, post-bypass and treatment groups.

Table No. V shows that angina has shown insignificant difference with treatment group and trait anxiety has equally contributed in the development of cardiovascular problems i.e. both in angina and in treatment group.

Similarly, Table No. VI indicates that when Trait Anxiety was measured through projective technique, the trait anxiety of pre-bypass patients was more as compared to the treatment group. There was no significant difference between angina and MI patients, angina and pre bypass patients, angina and post bypass patients, angina and treatment pre bypass and treatment group. The reason may be that projective techniques give true picture of the traits of individual by virtue of lesser cultural influence.

Table No. IV shows that there is a clear difference among all the disease categories (F=6.25, df=4,45,p<.01) as regard to Trait Anxiety. Table No. VI indicates that this significant difference is only due to treatment groups. This may be because the treatment group patients appear to be more satisfied with their health status as compared to the pre-bypass patients.

In concluding the discussion of this hypothesis, one can say that anxiety is an important psychological Risk Factor in the development of cardiovascular disorders. The emotional and bodily constitution of man is regulated a great extent by the hormones of the endocrine systems.

REFERENCES

1. Lipowisky, Z.J. What does the word Psychosomatic really means? A historical and semantic inquiry. Psychosomatic Medicine, 1984, 46, 153-171.
2. Dunbar, H.F. Emotions and bodily changes. 3rd edition, New York: Columbia University Press, 1946.
3. Rosenman, R.H., Friedman, M., Jenkins, C.D., Straus, R., Wurm, M., Kositcheck, R. The prediction of immunity to coronary heart disease. Journal of the American Medical Association, 1966, 198, 1159.
4. Glass, D.C., Contrada, R., 7 Snow, B. Stress, type A Behavior, and Coronary Disease. Weekly Psychology Update, 1980, 1, 2-6.
5. Mc Leod, D.R., Hoehn-Saric, R., Stefan, R.L. somatic symptoms of anxiety: Comparison of self-report and Physiological measures. Biological Psychiatry, 1985, 21, 301-310.
6. Tamaren, A.J., Carney, R.M., Alien. Predictive validity of the cognitive vs somatic anxiety distinction: Pavlov Biological Science, 1985. 20, 177-180.
7. Lown, B., DeSilva, R.A., & Lenson, R. Roles of Psychological stress and autonomic nervous system changes in provocation of ventricular premature complexes. American Journal of Cardiology, 1978, 43, 979-985.
8. Natelson, B.H. Neurocardiology: an interdisciplinary area for the 80s. Archives of Neural Cardiology, 1985, 42.
9. Sales, S.M. Organizational role as a risk factor in coronary disease. Administrative Science Quarterly, 1969, 14, 325-336.
10. Rotter, J.B. Some probvlems and Misconceptions related to the construct of internal versus external control of reinforcement. Journal of consulting and Clinical Psychology, 1975, 43, 56-67.
11. Gemmill G.R., & Heister, W.J. Fatalism as a factor in Managerial Job Satisfaction, JobStrain, and Mobility. Personnel Psychology, 1972, 25, 241-250.
12. Cooper, C.L., & Marshal, J. OccupationalSources of Stress: a review of literature relating to coronary heart Disease and Mental ill health. Journal of Occupational Psychology, 1976, 11-28.
13. Glass, D.C. Stress, behavior patterns, and coronary disease. American Scientist. 1977, 65, 177-187.
14. Pelletier, K.R. Healthy people in unhealthy places: Stress and fitness at work. New York: delacete, 1984.
15. National Institute of Cardiovascular Disorders. Statistics of Cardiac Mortality in Pakistan, brief No.18, Karachi, Pakistan, 1999.
16. Spielberger, C.D., Gorsuch, R.L., and Lushene, R.E. Manual for State-Trait Anxiety Inventory. Consulting Psychologist Press. Inc., California, 1970.
17. Jenkins, C.D. Behavioral risks factors in coronary artery disease. Annual Review of Medicine. 1978, 29, 543-562.
18. Ossified, A.M., Lebovites, B.Z., Shekelle, R.B., & Paul, O. A prospective study of the relationship between personality and coronary heart disease. Journal of chronic Diseases, 1967, 17, 265-276.

A VALIDATION STUDY OF BESSON SCORE FOR DIFFERENTIAL DIAGNOSIS OF STROKE SUBTYPES

SAADAT H. ZAIDI, JAMAL ARA, S. WAJID AHMED, *RUKHSANA SATTAR, SHABBIR HUSSAIN.*

ABSTRACT:

This study was carried out to validate the predictive accuracy of stroke scoring system developed by Besson and colleagues for clinical diagnosis of nonhemorrhagic infarction. The Besson scoring system calculated as

(2 x alcohol consumption) + (1.5 x plantar response) + (3 x headache) + (3 x history of hypertension) - (5 x history of transient neurological deficit) - (2 x peripheral arterial disease) - (1.5 x history of hyperrlipidemia) - (2.5 x atrial fibrillation on admission) was applied prospectively to 100 consecutive acute stroke patients admitted to medical Unit I, Ward-5, JPMC, Karachi. A score less than 1 predicted presence of infarction. A score greater than 1 was considered uncertain. Patients previously receiving anticoagulant therapy were excluded from the study. CT scan was done in every case for definitive diagnosis.

The score was uncertain in 80 patients. In 20 patients it predicted presence of infarction. Out of these, 19 patients were found to have infarction on CT and one patient was found to have cerebral hemorrhage. Thus the scoring system had a sensitivity of 32.8%, specificity of 97.6% and a positive predictive value of 95% (C.I. = 73 -99%) for diagnosis of ischemic infarction. A score of ≥ 9 was found to be a cutoff point at which the Besson score diagnosed cerebral hemorrhage.

KEY WORDS: Diagnosis; stroke subtypes, hemorrhagic stroke; ischemic infarction.

INTRODUCTION

"Stroke" is the second most common cause of death after ischemic heart disease[1] throughout the world! Of the two third individuals who survive a stroke, at least half are disabled.[2] Compared to western world, stroke in the developing world is less well documented. However one of the consensus statements from Asia-Pacific conference predicts that "in the next 30 years, the burden of stroke will grow most in the developing world rather than the developed world".[3] Pakistan is also burdened with this problem. Both primary prevention and effective treatment of stroke are the needs of the hour.

The management of a patient with acute stroke is based on the knowledge of stroke type-hemorrhagic or ischemic.[4] The most accurate method of differentiating the two stroke types is computed tomography.[5] Unfortunately, CT scan facilities are both expensive and available in only large hospitals in Asia.[3] On the other hand, the traditional unstructured clinical diagnosis of acute stroke has been shown to be unreliable in distinguishing between pathological subtypes of strok.[6-8] Therefore to provide a substitute for CT scan and to improve the clinician's ability to accurately predict the stroke subtype at beside, stroke scoring systems have been developed. These scoring systems are based on clinical data. The three most important ones are the Allen score developed in england9, the Siriraj score developed in Thailand[10] and the Besson score developed in France.[11] The Besson score is claimed by its developers to be the only score that predicts infarction with a positive predictive value of 100%.[11] These scoring systems have been found to be superior to traditional clinical diagnosis in predicting stroke subtypes.[12-14]

PURPOSE OF STUDY

The purpose of our study was to validate the predictive accuracy of Besson score and to determine whether it can replace CT as a clinical means of stroke subtype diagnosis.

Correspondence:
Dr. Saadat H. Zaidi, Medical Unit I, Ward 5,
Jinnah Postgraduate Medical Centre, Karachi.

PATIENTS AND METHODS

One hundred adult patients meeting the WHO's criteria of 'acute stroke syndrome' admitted to Medical Unit I, ward-5, Jinnah Postgraduate Medical Centre, Karachi were included in this prospective study. Clinical data as laid down in the score (Table I) was recorded prospectively and CT scan head was done in every patient. Other relevant investigations were also done in every patient. Patients with TIA, subarachnoid hemorrhage, on anticoagulant therapy and with non diagnostic CT (tumor or hemorrhagic infarction) were excluded from the study.

TABLE-I THE CALCULATION OF THE BESSON SCORE

I. Stroke Score = (2 x alcohol consumption) + (1.5 x plantar response) + (3 x headache) + (3 x history of hypertension) - (5 x history of TIA) - (2 x peripheral arterial disease) - (1.5 x history of hyperlipidaemia) - (2.5 x atrial fibrillation on admission)

II. Alcohol consumption is scored as '0' if absent or unknown and '1' if patient drank alcohol every day, whatever the amount.

History of hyperlipidaemia, history of hyperlipdaemia, and history of transient neurological deficit (including amaurosis fugax) is scored as '0' if absentor unknown, and '1' if present.

Plantar responses are scored '0' if absent, '1' if extensor ipsilaterally to deficit, '2' if extensor contralaterally to deficit, and '3' if they were both extensor.

Peripheral arterial disease is scored as '0' if absent, '1' when the patient has a documented history of lower limb claudication or if physical examination showed the loss of at least one arterial ankle pulse.

Headache within 2 hours before onset, and/or headache after onset is scored '0' if absent and '1' if present.

The Besson score:

A score below '1' indicates non-haemorrhagic infarct. A score '>1' represents an equivocal result, needing a CT scan to verify the diagnosis.

RESULTS

The number of patients with CT proved ischemic infarction was 58 with 34 (58%) being male and 24 (42%) being female. The number of patients with CT proven hemorrhagic stroke was 42, with 17 (40%) being males and 25 (60%) being females.

The number of patients correctly predicted by the Besson score to have ischemic infarction was 19 (32%) with ischemic infarction and 19% of all patients). The number of patients falsely predicted by the Besson score to have infarction was '1'. The number of patients predicted by Besson score to have uncertain results was 80. The sensitivity, specificity, and positive predictive values of the Besson score for ischemic infarction were 32.8%, 97.6%, and 95% (95%CI = 73-99%).

Table-1 is about the prediction of ICH by the Besson score is given in Table II. It gives a comparison between the two groups, i.e. those with scores ≥ 9 (71 patients) and those with scores ≥ 9 (9 patients) for absence of infarction and presence of only ICH. On a "chi-square test" for hamogeneity, the difference between two groups was highly significant (P = 0.005). Thus score of ≥9 on the Besson scale can be used to predict ICH.

TABLE-II PREDICTION OF INTERACEREBRAL HAEMORRHAGE

The Besson Score	Patients with Infarction	Patients with Intracerebral Haemorrhage
≥ 9	0	9
≥ 1, <9	39	32

X2 = 7.81, P < 0.005 (STATISTICALLY SIGNIFICANT) FOR THE COMPARISON BETWEEN GROUPS THE P-VALUE INDICATES THAT BESSON SCORE OF ≥9 CAN BE USED AS A CUT OFF VALUE TO PREDICT ICH.

DISCUSSION

The Besson score is designed to specifically identify patients with non hemorrhagic stroke. A clinical tool like Besson score can be very helpful in reducing both the morbidity and mortality of ischemic stroke if it is found to be as reliable as CT scan in stroke subtype diagnosis in non affording stroke patients in a developing country like ours.

Unfortunately our study found that the Besson score did not predict ischemic infarction with a positive predictive value of 100% and it had a sensitivity of only 32.8% for infarction. In the validation study of this score done on 200 patients by Besson and colleagues (1995), the positive predictive value for infarction was 100% (95% C.I. 93-100%). However in this study, the score correctly predicted infarction in only 43% of patients with infarction11. Two recent studies, one conducted by Weir and Lees (1996) in UK on 991 stroke patients15 and the other conducted by Madder and Mandel (1998) in USA on 61 patients16 showed that this score cannot reliably exclude hemorrhage while diagnosing infarction and that it has limited applicability in infarction population. However all the studies including ours have found that Besson score has better positive predictive value than Siriraj and Allen scores in diagnosing infarction. It means that Besson score is not affected due to its design by the substantial overlap between clinical presentation of hemorrhage and infarction8,9,17. The cost of this increased sensitivity to infarction is of low proportion in stroke population to whom it is applicable15.

In their original study, Besson and colleagues (1995) found no cut off value to define a positive predictive value of close to 100% in the diagnosis of cerebral hemorrhage11. In our study, at the score of '9' or above, nine patients were found to have CT proven hemorrhage and no patient was found to have an infarction. Thus a score of '9' can be considered as a cut off value, at or above which we can classify a patient as having intracerebral hemorrhage. However, this finding needs to be validated in further studies.

Therefore since Besson score has a limited applicability in infarction population and as it cannot diagnose infarction with a positive predictive value of 100% it cannot be used for therapeutic decision making in infarction patients. CT remains essential for this purpose.

REFERENCES

1. Murray CJL, Lopez AD. Mortality by cause for eight regions of world: Global Burden of Disease Study. Lancet. 1997;349:1269-76.
2. Bonita R, Solomon N, Broad JB. Prevalence of stroke and stroke-related disability: estimates from Auckland Stroke Studies. Stroke. 1997;28:1898-1902.
3. Niphon Poungvarin. Stroke in the developing world. Lancet. 1998; 352: (suppl III) 19-22.
4. Phillippe Vuadens, Julien Bogousslavsky. Diagnosis as guide to stroke therapy. Lancet. 1998; 352: (suppl III) 5-9.
5. Sandercok PAG, Molyneux A, Warlow CP. Value of computed tomography in patients with stroke: Oxfordshire Community Stroke Project. Br Med J. 1985; 290: 193-7.
6. Von Arbin M, De Faire U, Helmers C, Miah K, Murray V. Accuracy of bedside diagnosis in stroke. Stroke. 1981;12: 288-93.
7. Harrison MJG. Clinical distinction of cerebral hemorrhage and infarction. Post Grad Med J. 1980; 56: 629-32.
8. Schaafsma S. On differential diagnosis between cerebral hemorrhage and infarction. J Neurol Sci. 1968; 7: 83-95.
9. Allen CMC. Clinical Diagnosis of acute stroke syndrome. Q J Med. 1983; 42: 515-23.
10. Poungvarin N, Viriyavejakul A, Komontri siriraj stroke score and validation study to distinguish supratentorial intracebral hemorrhage from infarction. Br Med J. 1991; 302: 1565-67.
11. Besson G,Robert C, Hommel M, Perret J. Is it clinically possible to distinguish nonhaemorrhagic infarction from hemorrhagic stroke? Stroke. 1995; 26" 1205-09.
12. Celani MG, Ceravolo MG, Duca E, Minciotti P, Caputo N, Orlandini M, Ricci S, Provinciali L. Was it infarction or hemorrhage? A clinical diagnosis bymeans of Allan score. Jneurol. 1992; 239 (7): 411-3.
13. Celani MG, Righetti E, Miglacci R, Zampolini M, Antoniutti L, Grandi FC, Ricci S. Comparability and validity of two clinical scores in earlydifferential diagnosis of acute stroke. Br Med J. 1994; 308: 1674-1676.
14. Daga MK, Sarin K, Negi VS. Comparison of Siriraj and Guy's Hospital score to differentiate supratentorial ischemic and hemorrhagic strokes in India population. J Assoc Physicians India. 1994 Apr;42 (4): 302-3.
15. Weir CJ, MurrayGD. Scoring Systems for the Differential Diagnosis of Ischemic and Hemorrhagic Stroke. Stroke 1996; 27(2): 337-338.
16. Madder TJ, Mandel A.A new clinical scoring system fails to differentiate hemorrhagic from ischemic stroke when used in the acute care setting. J Emerg Med 1998 Jan-Feb:16(1): 9-13.
17. Panzer RJ, Feibel JH, Barker WH, Griner PF. Predicting the likelihood of hemorrhage in patients with stroke. Arch Intern Med. 1985; 145: 1800-1803.

HAEMOPERITONEUM BY TAENIA INFESTATION

A CASE REPORT

NIZAM UD DIN DANWER

ABSTRACT:
Taenia infestation which usually produces mild symptoms like indigestion, diarrhoea, constipation, vomiting, loss of appetite and anaemia may occasionally lead to intestinal perforation and present as haemoperitoneum.

KEY WORDS: Intestinal Perforation, Taenia Infestation, Haemoperitoneum.

CASE REPORT

A 27 year old patient was operated as a case of appendicitis at a peripheral hospital. He was re-admitted there with fever, pain abdomen, gross anaemia and jaundice and then transferred to CMH, Sialkot and admitted as a case of infective hepatitis. On the next day of admission, surgical opinion was sought and with diagnosis of peritonitis urgent exploratory laparotomy was planned. His serum bilirubin was 96.0 mmol and serum urea 34.4 mmol.

Ultrasound examination revealed enlarged liver and large fluid collection in the subhepatic region (16 x 12 cm) extending from RIF and another collection (15 x 5.7 cm) in the pelvis with thick walls.

At laparotomy peritoneal cavity was full of dark blood. After peritoneal toilet all the viscerae were examined. On separating fibrinous patch from the small intestine a one cm wide circular perforation in the ileum at antemesentric border, with bleeding edges and a head of tape worm protruding through appeared. The wall of intestine all along and around perforation was normal. There was no inflammation or change in colour of ileum. Mesenteric lymph nodes were not palpable. After extracting the tapeworm the intestine was repaired. His general condition improved gradually. He was allowed fluids after one week and passed stools on 9th post-operative day. His serum bilirubin level decreased gradually. Post operatively abdominal incisional wound required resuturing due to infection. He was later discharged.

DISCUSSION

Taenia infestation usually produces mild GIT symptoms like diarrhoea, constipation, vomiting, loss of appetite and anaemia may occasionally lead to intestinal wall perforation and present as haemoperitoneum. Intestinal perforation and peritonitis has been reported in literature as a rare cause. But the sole presentation of haemoperitoneum with jaundice due to gradual bleeding into peritoneal cavity and absorption of blood leading to presentation of jaundice is a rarity.[1]

REFERENCES

1. Ahmed S. Intestinal perforation by Taenia Infestation, AFMJ 1990 17-18.

Correspondence:
Brig. (Dr.) Nizam Ud din Danwer, Head Department of Surgery,
CMH, Sialkot.

OBTURATOR HERNIA

A CASE REPORT

KULSOOM JUNEJO, MOHBAT ALI, IRSHAD WAHEED.

ABSTRACT:
A 65 years old lady was admitted with distal bowel obstruction and pain in right lower abdomen radiating to ipsilateral thigh and knee-Howship Romberg sign. A preoperative suspicion of obturator hernia was made, confirmed per operatively. Although the diagnosis of obturator hernia is difficult to establish but it should be suspected in frail old women with small bowel obstruction and obturator neuralgia.

KEY WORDS: Obturator Hernia, Diagnosis

INTRODUCTION

Obturator hernia is an uncommon entity. It occurs most often in elderly debilitated women hence the nick name " Little old lady's hernia".[1] Hernia passes through the obturator foramen, following the path of obturator nerve and vessels. The diameter of obturator canal is larger in females therefore it is five times more common in women than men.[2] The most frequent clinical sign of obturator hernia is intestinal obstruction followed by Howship Romberg sign. Its diagnosis is usually made at laparotomy for small bowel obstruction.[3,4]

CASE REPORT

A 65 year old lady was admitted with a five days history of pain in right lower abdomen radiating to ipsilateral thigh and knee (Howship Romberg sign).vomiting and absolute constipation for one day. Physical examination revealed tenderness in right lower abdomen and exaggerated gut sounds. rectal and per vaginal examinations were unremarkable. Plain abdominal radiograph revealed picture of small bowel obstruction with multiple air fluid levels. A preoperative diagnosis of small bowel obstruction; secondary to obturator hernia was suspected. Patient underwent a midline laparotomy after resuscitation. Per operative findings were right obturator hernia of Richter's type (confirmed pre-operative suspicion). Hernia was reduced by gentle manipulation and traction on small bowel. Herniated knuckle of ileum was non viable and resection anastomosis performed. Hernia repair was done with simple interrupted prolene sutures at the neck of the sac. Patients recovery was smooth and uneventful and she was discharged after 6 days.

DISCUSSION

Obturator hernia is a rare cause of mechanical intestinal obstruction that occurs predominantly in elderly emaciated women. The obturator foramen is entirely covered by a fibro-osseous membrane except at the anterio-superior aspect - the site of obturator canal. It is about 2-3 cm long and houses the obturator vessels and nerve. The internal orifice is closed by parietal peritoneum and pre peritoneal fat. Herniation begins with entrance of preperitoneal fat and connective tissue into the pelvic orifice of obturator canal, followed by a dimple in the peritoneum over the internal opening. Finally the symptoms are produced by entrance of an organ usually ileum into the sac.[5] Initially the hernia may reduce and produce partial or complete obstruction. The herniated sac may follow anterior or posterior divisions of obturator nerve. More commonly the hernia lies between the pectineus and obturator externus muscle's path of the anterior division (as in our case). But in some cases it lies between the superior and middle fasciculi of obturator externus muscle's path of posterior division[6].

Patients usually present with partial or complete mechanical small bowel obstruction. The presence of pain in ipsilateral thigh and knee aggravated by abduction and extension-"Howship Romberg sign", caused by

Correspondence:
Dr. Kulsoom Junejo, Surgical Ward 3,
Jinnah Postgraduate Medical Centre, Karachi

compression of branches of the obsturator nerve by the hernial sac, is present only in one third of the cases5. Hernia is never externally visible and a palpable mass is detected only occasionally. Thus the diagnosis of obturator hernia is difficult to establish and can only be made in 25-33% of cases[7,8] often the correct diagnosis is stumbled upon as a result of surprising intra operative finding. More recently however with the use of ultrasonography, computed tomography and laparoscopy the diagnostic yield has improved, paving way for decline in morbidity and mortality related to delayed diagnosis9. Computed tomography is the optimal technique for detecting mass in obturator foramen10. Therefore early use of CT in appropriate clinical setting should improve the prognosis.[11,12]

The often debilitated condition of these patients and frequent delay in diagnosis, combines to produce significant mortality and morbidity in 30% cases.[13] Therefore only operation is all that is needed; not a preoperative diagnosis. These hernias typically can be reduced by traction as adhesions are uncommon. Bowel resection is necessitated in upto 80% cases.[2]

There are several ways of repairing the hernial defect but because of rarity of the problem no one has adequate experience to advocate resoundingly any single approach.

We used simple interrupted prolene sutures at the neck of the sac. Although there are several techniques for repairing the defect,[14,15] some prefer leaving it open and the recurrence rate of unclosed defects is not known (Table I).

It is concluded that the clinician should have a high index of suspicion for obturator hernia in old debilitated women presenting with small bowel obstruction and obturator neuralgia especially with no past history of abdominal surgery. Imaging techniques should be used only if appropriate to help establish a diagnosis. Surgical intervention is the only effective treatment and delays should be avoided to decrease the morbidity and mortality of these cases, which is thought to be very high as compared to other abdominal hernias.

REFERENCES

1. Yip AW, Ahchong AK, Lam LH. Obturatoe hernia - a continuing diagnostic challenge. Surgery 1993 , 113: 266-269.
2. Bailey and Love's. Short practice of surgery. Hernias-oburator hernia. 22nd Edition Pg-898.
3. Keogan MT , Paulson EK- Gastro-intestinal case of the day. Obturator hernia causing small bowel obstruction. AJR 1995, 165: 192-193.
4. Locy, Lorentz TG, Low Pwk. Obturator hernia presenting as ' small bowel obstruction. Am J Surg 1994 , 167 : 396-8.
5. Bergstein JM, Condon RE. Obturator hernia. Current diagnosis and treatment. Surgery 1996: 199: 133-136.
6. Gurlich.R , Lindner.J. Obturator hernia Rozhl Chir. 1996 June 75(6) 309-9.
7. Anne D.Walling, Pit falls in diagnosing obturator hernia. (Tips from other Journals) Issue Jan 15 1998.
8. H Pippert and P.Messmer. Obturator hernia. Current diagnosis and treatment. Surgery 1996: 199 : 133-136.
9. Ijiri R, Kana maru H, Yokoyama H, Shirakawa M, Hashimoto H, Yoshino G.Obturator hernia - the usefulness of computed tomography in diagnosis. Surgery 1996: 119: 137-140.
10. Yokohama Y, Yamaguchi A, Isogai M, Hor A, Kaneoka Y Thirty six cases of obturator hernia- does computed tomography contribute to post-operative outcome? World J. Surg 1999, 23: 214-216.
11. Walker AP. Biomaterials in hernia repair. In :Nyhus LM, Condon RE, eds. Hernia. Philadelphia: Lippincott, 1995: 534-40.
12. S Kandalatis CJ, Androulatis J, Colborn GL , Skandalakis JE. Obturator hernia. Embryology, Anatomy and Surgical applications. Surg.Clin.North Am, 2000 Feb: 80(1): 71-84
13. DN.Lobe, D.J.Clark and AP Barlow. Obturatoe hernia : a new technique for repair. JR coll surg Edinb, 43 Feb 1998, 33-34
14. Chan MY, Swaminathan I, Ng BK. Obturator hernia case report. Ann Acad Med Singapore. 1994 Nov. 23(6): 911-3.
15. G.R.Giles and N.A.Halasz - The Abdomial wall and Hernias-obturator hernia. Essential Surgical Practice by A.Cuschieri. Third edition Pg-1443.