Case Report:

Congenital Mesenteric Defect: A Rare Cause of Internal Herniation:

S H DAR S AHMED M A SHEIKH
Department of Paediatric Surgery, Mayo Hospital, lahore
Correspondence to Dr. Sajid Hameed Dar, Assistant Professor

Internal herniation into a mesenteric defect is a rare entity. The case of one and a half-year-old girl is reported who presented with signs and symptoms of mid gut obstruction. Lapartomy revealed a large sized defect in the mesentery of small intestine with evidence of malrotation. Embryology and clinical management of the disease is discussed along with review of the literature.
Key words: Internal herniation, mesenteric defect.

The primitive gut has two mesenteries: ventral and dorsal. During the first twelve weeks of gestation the gut undergoes rapid transformations: increase in length, physiological herniation, return of the herniated gut, counter clock wise rotation through an arc of 270 degrees and fixation of the gut to posterior parieties which continues into postnatal life. During the process of fixation of gut the primitive mesenteries are partially resorbed with the development of new mesenteries. The mesentery of small intestine (with right and left leaves) merges with the posterior parietal peritoneum. Any deviation from this normal process of fixation results in congenital bands, kinks in the gut and development of various abnormal fossae (Duodenal fossae, caecal fossae and fossa intersigmoidea). These may serve as potential sites of internal hernia. Vary rarely anomalies of intestinal fixation result in defects in the mesentery of small intestine transverse colon and omentum. Usually these are small defects which can snare up a small portion of intestine, leading to incarceration and infarction of intestine. Very rarely these defects are large in size with free to and fro transit of intestinal coils. Usually this leads to recurrent bouts of pain abdomen, vomiting, failure to thrive and constipation. These recurrent bouts of incomplete obstruction may continue into adult life or complete gut obstruction may supervene any time.

Case report

One and a half year old girl presented, in the emergency of Department of Paediatric Surgery, Mayo Hospital, Lahore with the complaints of excessive crying, vomiting, abdominal distension & absolute constipation for the last 3 days. For the last one day the child was listless and febrile. The child had been crying episodically with drawing up of legs. The vomitus was greenish yellow in color. There was also previous history of bouts of crying which would subside of its own or with medication.

At the time of admission the child weighed 7 kg, was severely dehydrated and pale . Her pulse was 140/min with temperature 102 F. On examination abdomen was distended more so in the central part and right side. The abdomen was tender on the right side and coils of intestine could be felt . No other mass or viscus was palpable. Bowel sounds were absent. Rectal examination revealed only mucous staining of the finger.

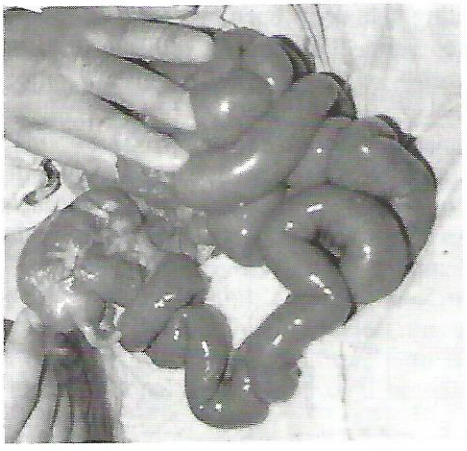
Her haemoglobin was 7.5 G% with TLC of 7500/mm3 and normal differential count. Serum sodium was 128 mol/ L and serum potassium. 3.5 mol/L . Blood urea was 60mg/dl. X-ray of abdomen in erect posture revealed multiple air fluid levels in whole abdomen , distended caecum and clustering of coils in the central part and right side of abdomen.

Pre-operative resuscitation with correction of fluid and electrolyte imbalance and haemoglobin deficit was carried out in the next few hours.

Celiotomy was performed through supra umbilical transverse incision. Whole of the small intestine, caecum and ascending colon was grossly distended. Beyond hepatic flexure the colon was collapsed. There was a large triangular shaped, defect in the mesentery of small intestine through which coils of distal ilium, caecum and ascending colon had herniated and undergone one and a half circle clock wise twist. Almost whole of ilium was devoid of mesentry. Caecum and ascending colon were non-fixed Dudeno jejunal flexure was at the mid line instead of being on left side. Mesenteries of the transverse and sigmoid colon were normal. The left half of root of mesentery was normal with normal arcades of superior mesenteric artery branches. Posterior parietal peritoneum was intact. The blood supply of the ilium was through a single medium sized vessel from superior mesenteric artery. It was running in a frill of remnant of mesentery and sending out perpendicular branches going directly into the substance of ilium and caecum. (as shown in photograph). The vessel supplying the ilium and caecum was anastomosing with branches of right colic artery which was coursing in the upper margin of defect. The wall of ilium (distal part) and right sided colon were dusky, oedemotous and thickened with few areas of ecchymoses. The herniated gut was reduced (untwisting in anti clockwise direction). Hepatic flexure showed evidence of chronic pressure with narrowing of lumen by about 50% and serosa at this point showed , 0.5 cm wide, area of greyish white discoloration encircling 3/4 of the diameter of colon.

Post reduction gut was found normal . Greyish white patch of discoloration at hepatic flexure was buried with Lambert sutures. The upper left part of mesenteric defect was approximated with interrupted non- absorable sutures. As the triangular shaped defect was broader towards lower right side, approximation of the edges would have resulted in clustering and kinking of coils of intestines. So in this area the edges of the defect were tacked to post parietal peritoneum.

Post operative recovery was uneventful. The patient is asymptomatic up to one year of follow up.



Photograph showing mesenteric defect and internal herniation

Discussion

The dramatic events in the development of GIT are completed by 12th week of gestation[1]. During the return of the physiological hernia both the rotation and fixation of the gut go hand in hand with fixation lagging behind. In fact the fixation of the coils of intestine continues into many months of postnatal life[2]. The sub- hepatic location of the neonatal caecum is no longer considered a pathological entity. The broad term of malrotation really refers to all abnormalities of intestinal rotation and attachment[3]. So it is rare to encounter anomalies of rotation without any abnormal attachment of intestine. It is the combination of both which produces a spectrum of clinical presentations including spaces for internal hernia.

Internal hernia is the name given to herniation of intestine through an aperture (natural or acquired) within the peritoneal cavity[4]. Initially it was thought to be an acquired phenomenon: a portion of intestine pushing a fold of peritoneum, enlarging it and finally getting entrapped in it. It was the work of Andrew who explained it on the basis of anomalies of intestinal rotation[5]. It is also essential to differentiate between true internal hernia (having a sac) and free peritoneal herniation (without a sac) through a defect in any mesentery or loops formed by congenital bands and cords[6]. Explanation of this hernia is difficult. In some cases a previous history of fall or other injury is forthcoming. A line of force of sufficient intensity can produce a rent in the mesenteric root which many months later is the aetiologic agent for an internal hernia[7]. In vast majority of such cases so such history of trauma can be documented. Instead most of these hernia have a congenital origin. The four major internal harniae are:

- Paraduodenal preferably termed mesocolic
- Into foramen of Winslow
- Mesenteric hernia
- Omental hernia

All extremely rare, these may be an incidental finding at laparotomy. Mesenteric hernia can be present in any one of the mesenteries of small intestine, transverse and sigmoid colon. Failure of parts of the original membranes to disappear and minor alterations in the development of secondary mesenteries can result in variable sized mesenteric defects. Local blood vessels are also arranged abnormally as seen in our case. Usually small, mesenteric defects can be large in size allowing free intestinal herniation[8]. There are no clinical findings or investigation which can give us a pre-operative diagnosis of mesenteric hernia. These defects are managed as the situation presents itself. During closure of these defects, care should be taken not to strangulate the marginal vessel which runs very close to the edge. A search should also made for any evidence of malrotation and correct it, if required.

References

1. Longman J (ed) in Medical Embryology Williams and Wilkins, Baltimore P-227, 1989.
2. Dott NM: Anomalies of intestinal rotation: Their embryology and surgical aspects, with report of 5 cases . Brit J surg 11:251, 1923.
3. Filston HC, Kirks DR : Malrotation - the ubiquitous anomaly, J Pediatr Surg 16:614, 1981.
4. Soper R.T., Anderson A.E: Abdominal hernia, in Liechty R.D., Soper R.T, Synopsis of of Surgery, The C.V. Mosby Company London, P. 245-256, 1980.
5. Andrew E : Duodenal hernia. A misnomer, Surg., Gynec & Obstet: 37: 740, 1923.
6. Benson C.D. Mustard W.T., Ravitch M.M., Cynder Jr W.H. , Welch K.J., Paediatric Surgery, year Book Medical Publishers, Chicago P 749-70.
7. Ficcara B.J., in emergency surgery, F.A. Davis Company Philadelphia, P-244, 1953.
8. Janin Yetal: Mesenteric Hernia Surg Gynaecal Obstet 1980: 150: 747