

Case Report

Postoperative gastric outlet obstruction following hiatal hernia repair in an infant: A case report

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ABSTRACT

Background: Infantile hypertrophic pyloric stenosis (IHPS) is an exceedingly rare cause of postoperative emesis in a case of hiatal hernia. Occasionally it may simulate other etiology of gastric outlet obstruction.

Case Presentation: A 32-day-old male baby presented with respiratory distress and vomiting since birth. Diagnosis of eventration of left hemi diaphragm was made on CT Chest. At surgery, hiatal hernia with an intrathoracic stomach was found, which was repaired. On 5th postoperative day, the baby developed vomiting after feeding which gradually turned to be projectile in nature over a week. Contrast meal performed showed malpositioned stomach with delayed emptying. At re-operation, a well-formed olive of pylorus was encountered; Ramstedt pyloromyotomy was done. Postoperative course remained uneventful.

Conclusion: IHPS is a rarely described association with hiatal hernia. Pyloric stenosis should be considered in differential diagnoses of postoperative emesis in infants with hiatal hernia.

Keywords: Hiatal hernia, Infantile hypertrophic pyloric stenosis, Postoperative emesis, Gastric outlet obstruction.

INTRODUCTION

Hiatal hernia (HH) is characterized by gastric herniation to the chest through a widened esophageal hiatus. Vomiting, gastroesophageal reflux (GER), and recurrent chest infections secondary to aspiration etc., are various indications of HH repair.[1] HH is repaired with and without addition of fundoplication.[2] Postoperative emesis in a case of HH especially treated without fundoplication is attributed to GER, though projectile nonbilious emesis is a characteristic of gastric outlet obstruction.[2] Infantile hypertrophic pyloric stenosis is a well-known etiology of projectile nonbilious vomiting in early infancy. Herein, we report a case with HH complicated by gastric outlet obstruction secondary to development of IHPS in the postoperative period.

CASE REPORT

A 32-day-old male was born at 37-week gestation via C-section, with birth weight of 1.5kg, and product of con-

sanguineous marriage. There was no history of delayed cry and APGR was 9 after 5 minutes of birth. Antenatal scans did not show any significant findings except for small-for-gestational-age fetus. The baby was admitted in a private clinic, next day of his birth, for respiratory distress and intolerance to feed. The patient was referred to our department for further management with a diagnosis of eventration of left hemi-diaphragm. On examination, the infant was vitally stable with decreased air-entry on the left lower zone of the chest; and right sided reducible indirect inguinal hernia was also noted. On X-ray chest there was suspicion of eventration of left hemi-diaphragm (Fig. 1). CT scan chest showed stomach and spleen herniating to the left thoracic cavity and the diagnosis of congenital left diaphragmatic hernia was made. (Fig. 2). Echocardiography showed mild pulmonary hypertension for that cardiac consultation was taken.

Patient was optimized and operated on electively at the age of 25 days. Operative findings were a HH with herni-

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ation of complete stomach along with gastroesophageal junction (GEJ) into the chest cavity. The stomach was pulled back and HH was repaired. Right sided deep inguinal ring was also closed by a purse-string suture. Initial postoperative period was uneventful, and the patient was allowed oral sips on the 2nd postoperative day which the patient tolerated well. Next day the feed was started. On 5th postoperative day, the baby had 2 episodes of non-bilious vomiting which were attributed to GER. Feeding was continued with the management of GER with 45° prop-up position and small frequent feeds. The baby initially tolerated feeds for 2 days but later the frequency of vomiting increased and also it turned to projectile at occasions. Feeding was withheld and a contrast meal study was done which showed massively distended and malpositioned stomach with delayed emptying (Fig. 3).

baby was kept nil by mouth for 6 hours and allowed orally according to the feeding protocol of IHPS. Baby was fully fed within 24 hours and discharged in good condition. The baby is on our telephonic follow-up for 3 months (due to COVID-19 outbreak) and is doing fine without any vomiting.



Figure 1: Chest X-ray showing intrathoracic gastric shadow

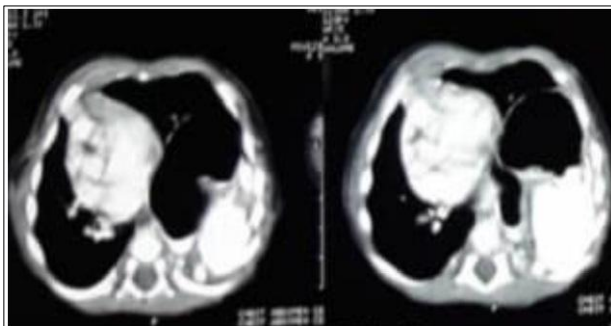


Figure 2: CT scan chest showing intrathoracic stomach, and suspicion of eventration of diaphragm

Patient was re-explored with suspicion of gastric volvulus; but at operation, the stomach was found adherent with liver causing its malposition. After adhesiolysis, significant hypertrophy of pylorus was found (Fig. 4). Ramstedt's pyloromyotomy was done and the stomach was placed in its normal position. Postoperatively, the

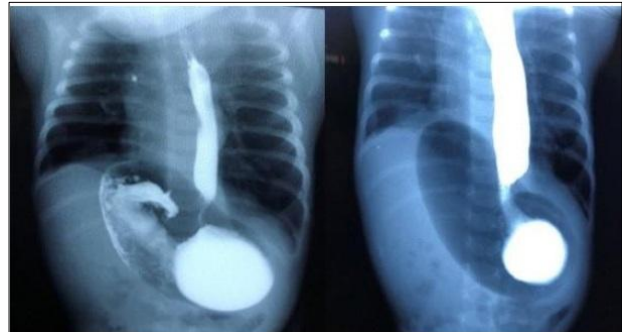


Figure 3: Showing Malpositioned stomach with gastric outlet obstruction

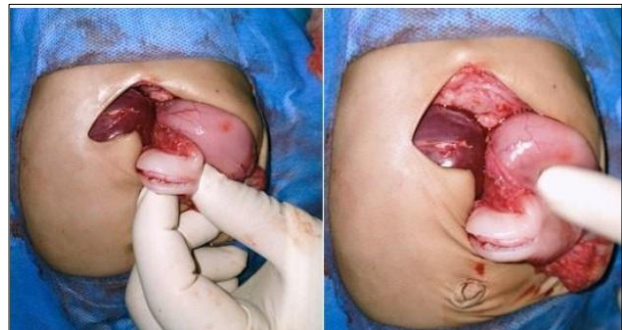


Figure 4: Operative figure showing IHPS as a cause of gastric outlet obstruction

DISCUSSION

HH is classified in four types; type-I is sliding hernia and characterized by intra-thoracic GEJ; type-II is paraesophageal hernia as characterized by intra-abdominal GEJ and a part of fundus herniates alongside of the esophagus; type-III is mix of type I and II; and lastly type-IV HH which are characterized by herniation of viscera other than stomach such as small or large bowel.[1] In the index case, as the GEJ was inside the thorax and also the entire stomach was herniated, therefore it was a type III hiatal hernia.

A handful no. of cases of HH have been associated with IHPS and few authors considered presence of IHPS as predisposing factor in development of infantile HH. [3] Roviralta referred the association of HH and IHPS in infants as the phrenopyloric syndrome. He suggested that the increased intra-abdominal and intra gastric pressure caused by vomiting leads to herniation of the stomach through a congenitally lax hiatus. [4]

IHPS leading to development of HH or concomitantly occurring with HH is debatable. Few cases of HH are reported wherein IHPS was developed after repair of HH. Iijima et al. reported 3 cases of HH associated with IHPS.

In their one case, on 14th day of life (DOL), there was no evidence of pyloric stenosis; however, during conservative management of GER, the baby developed symptomatic IHPS at the age of 1 month. [3] Similarly, in the index case, the pylorus was not noticeable at first surgery. During the postoperative period, the baby might develop pyloric thickening gradually which was also coincided by the gradual development of projectile nonbilious vomiting in our case.

IHPS is not only associated with HH but also other types of congenital diaphragmatic hernia (CDH). Redman et al. reported three cases with Bochdalek hernia who developed IHPS after successful repair of CDH in the postoperative period ranging from 1 week to 10-weeks. [5] Winckworth et al. reported a case of congenital intrathoracic stomach, secondary to congenital short esophagus, not associated with CDH or HH, developed pyloric stenosis necessitating thoracotomy and pyloromyotomy. [6] Contrast upper GI series, and ultrasonography can pick the associated IHPS. In the index case, we did not perform ultrasound because on contrast series a malpositioned stomach with gastric outlet obstruction was giving us suspicion of gastric volvulus. Treatment is straightforward Ramstedt pyloromyotomy.

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In conclusion, IHPS should be suspected in every case of HH [3]. Postoperative projectile non-bilious vomiting in a case of HH should warrant inspection of pylorus with ultrasonography to diagnose associated IHPS. If it is not present, one should move to other investigations to find out etiology of emesis.

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