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A Rare Case of Adult Post Traumatic Diaphragmatic Hernia

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Abstract:

Congenital diaphragmatic hernia (CDH) is a defect of the diaphragm associated with herniation of abdominal viscera into the chest cavity. We are going to report a case of post traumatic adult diaphragmatic hernia in a 45 yr female who presented with complain of severe vomiting and pain in left side of upper abdomen for 7 days with scaphoid abdomen, dull note on percussion and decrease breath sound on left side of chest. X ray and ultrasonography revealed herniation of stomach into left hemi thorax and mediastinal shift to right without significant deviation of trachea. CT scan confirmed the diagnosis and then exploratory laparotomy followed by decompression of stomach was done and the herniated stomach was repositioned in abdominal cavity. Chest tube was given in 6th ICS under supervision before the diaphragmatic defect was closed using prolene 1-0 and a prolene mesh was put over the defect and fixed. Gastrojejunostomy was done as the pyloric part of stomach was found to be fibrosed and stenosed. Post operatively the patient recovered well.

Keywords: ***Traumatic Diaphragmatic Hernia.***

Introduction

Congenital diaphragmatic hernia was first described by the French physician Lazarus Riverius in 1679. Bochdalek hernia was first described in 1848 by the Czechoslovakian anatomist, Vicent Alexander Bochdalek.

Congenital diaphragmatic hernia (CDH) is a defect of the diaphragm associated with herniation of abdominal viscera into the chest cavity. Related conditions of the diaphragm that mimic CDH but are not associated with diaphragmatic defects include diaphragmatic eventration and phrenic nerve palsy. These two conditions result in a mechanically impaired *''high-riding''* diaphragm without visceral herniation. The locations of the defect in CDH include posterolateral defect (foramen of Bochdalek), anterior/retrosternal defect (foramen of Morgagni), crural defect (paraesophageal hernia), and diaphragmatic agenesis (Fig. 1). Of these, Bochdalek CDH and agenesis are the most common and are associated with the greatest complexity and mortality risk.[1] The diaphragm starts to form during the fourth week of embryological development from four components of mesodermal structures. Lateral extension of the septum transversum from the midline joining to the lateral margins of the pleuroperitoneal fold results in the formation of the membranous (central tendon) and muscular diaphragm. The esophageal mesentery extends into the pleuroperitoneal fold to form the crural and dorsal portions of the diaphragm. Abdominal wall-derived mesenchyme migrates inward to fuse with the other components. Finally, lung expansion is thought to promote the fusion of all layers, resulting in a single fibromuscular tissue that physically separates the thoracic and abdominal cavities by the end of the 12th week. Diaphragmatic defects are thought to result from the failure of one or more of these layers to fuse with each other. In the case of Bochdalek hernias, abnormal development of the pleuroperitoneal fold (muscular diaphragm) is thought to result in the typical posterolateral defect as demonstrated through animal models of CDH. Because the right pleuroperitoneal fold is thought to close first, left-sided Bochdalek hernias occur more frequently than right-sided CDH. The most severe CDH cases include complete diaphragmatic agenesis (ie, the complete absence of a muscular diaphragm) and bilateral CDH.[2] The incidence of CDH is approximately 1 in 4000 live births, although the population incidence is slightly higher.16 Left-sided CDH is more common (80% of cases) compared to right-sided CDH (20%), but right-sided CDH is reported to have a greater mortality risk.[3] The major cause of mortality in infants with CDH are pulmonary hypoplasia, pulmonary hypertension, and associated congenital anomalies, especially cardiac defects.

Trauma to the diaphragm may be direct or indirect, and herniation may be obscured by concomitantinjuries and may remain occult for many years. The early physical signs and symptoms are meagre before the abdominal organs have penetrateddeeply into the thorax. The progress of injury can be divided into three phases: (1) initial, (2) latent, and (3) obstructive. Most traumatic hernias occur on the left side

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because of the diminished buffering force on the undersurface of the left hemidiaphragm. Roentgenograms are most often misinterpreted as indicating eventration of the diaphragm, gastric dilatation, or lesions in the lower lung fields or pleura.A dilated stomach in the left pleural cavity may simulate a pneumothorax. Diaphragmatic injury should always be considered in conjunction with trauma to the liver, kidneys, and spleen. We are reporting a case of traumatic diaphragmatic hernia with delayed presentation.

Case report

A 55yr female came to SOPD with c/o of vomiting and pain in left side of abdomen for last 7 days. h/o of trauma to left lateral side of abdomen 5 years back.

Fig1, 2: Preoperative Pic of Patient

On examination the tongue was dry and the skin was lax and pinch sign was positive suggesting the patient to be severly dehydrated. The abdomen was scaphoid. On palpation the abdomen was soft tender in left upper quadrant. The left lung was dull on percussion and the there was diminished breath sound on left lung. The apex beat was shifted to 5th intercoastal space near the left sternal border. There was no tracheal deviation. Initially it was very difficult to put the nasogastric tube but finally it was given successfully. Then on chest xray we found the herniation of stomach in left thorax with mediastinal shift to right.

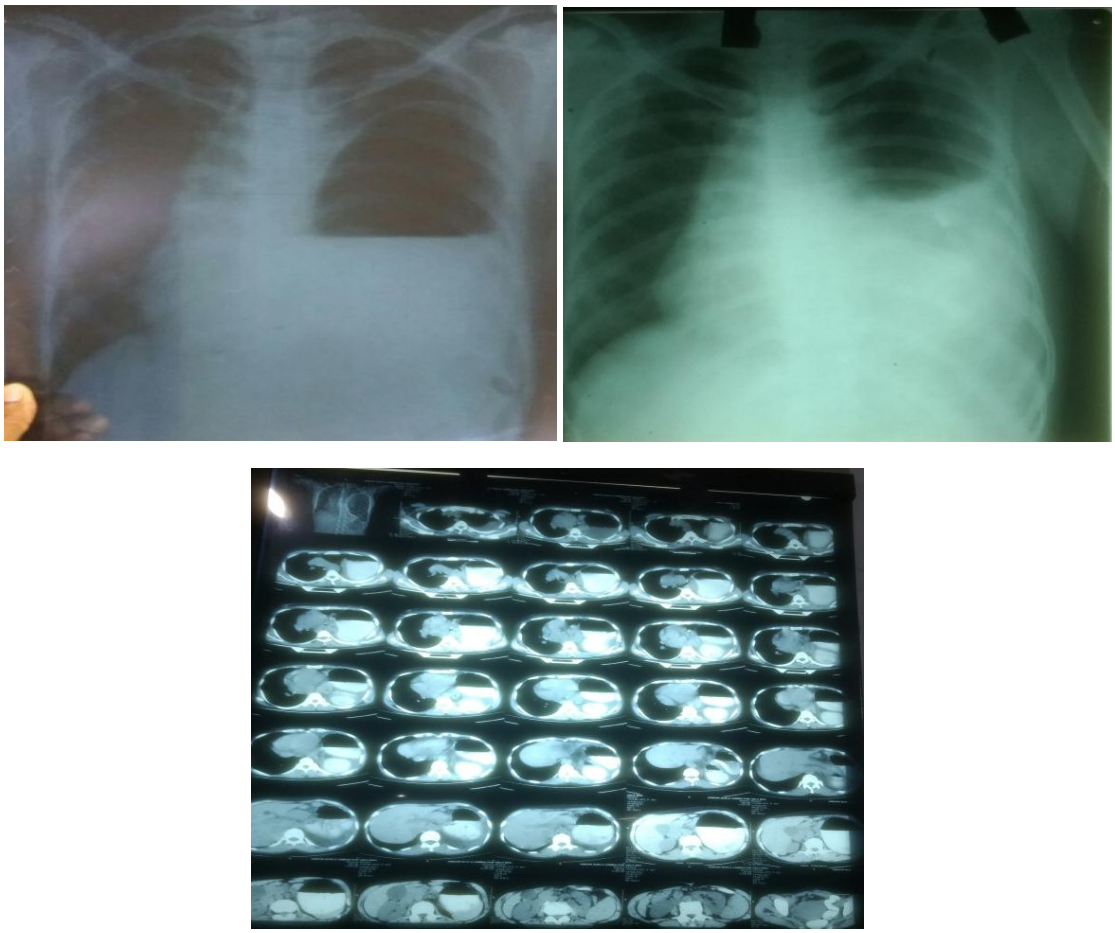

Fig 3, 4, 5: Showing Chest Xray before Ryle's Tube, After Ryle's Tube and Cect Thoraxrespectively.

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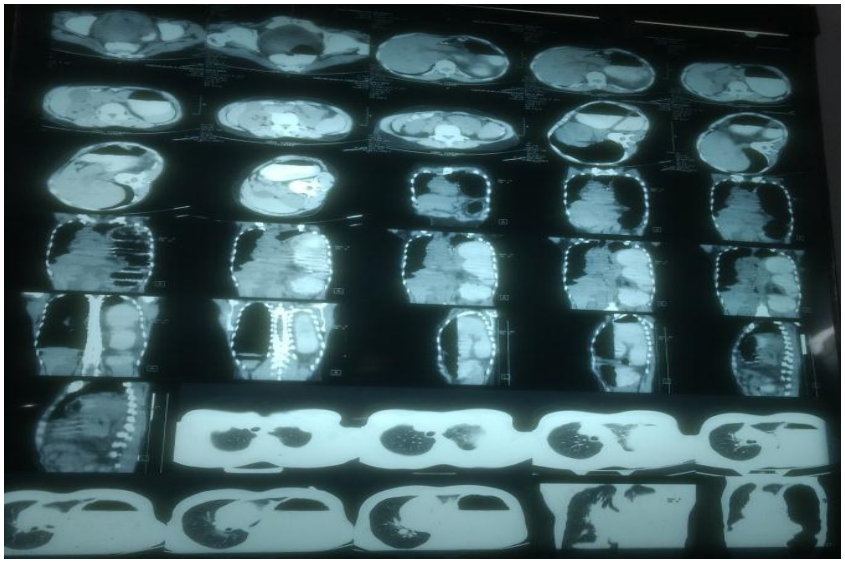

Fig 6: Showing Cect Thorax

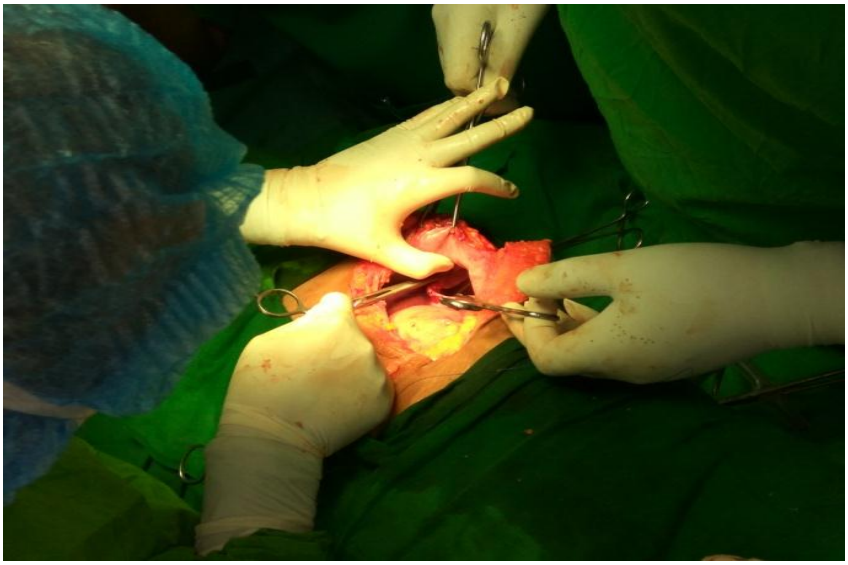

Fig 7: Showing the Defect in Diaphragm Intraoperatively

On ultrasonography and ct scan it was confirmed. Then we planned for exploratory laparotomy. On exploration we found that a part of the stomach was in the left hemithorax through a defect in lateral part of left diaphragm. The stomach was decompressed and put back to abdominal cavity. Then the chest tube was given in 6th intercoastal space in left side of chest under supervision through the defect.

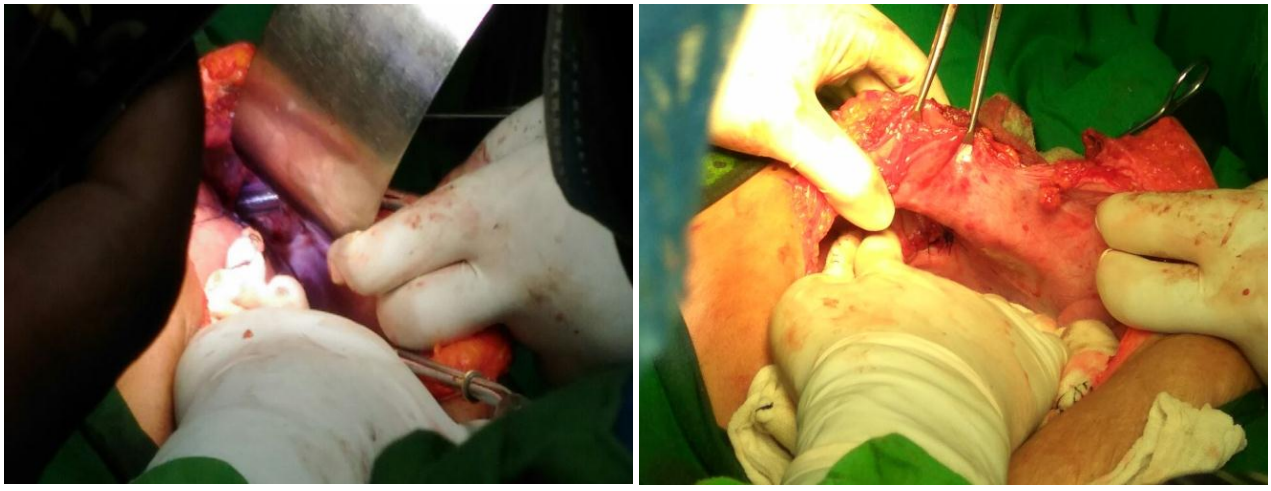

Fig 8, 9: Showing ICT Placement and Repair of Defect Respectively

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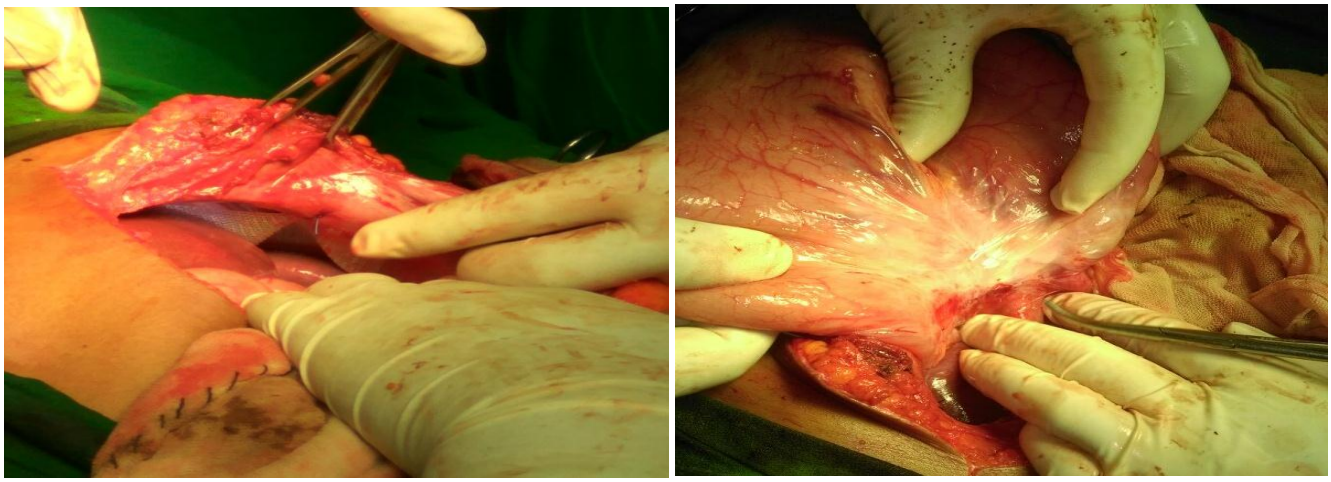

Fig 10, 11: Showing Mesh Repair of Defect and Stenosed Pyloric Part of Stomach

Then the diaphragmatic defect was closed with prolene 1-0. Then prolene mesh was fixed all over the defect. Then the stomach was found to be fibrosed and stenosed at pyloric region. So posterior gastrojejunostomy was done.

Abdominal drain kit was given in left side of abdomen near the anastomotic site. Hemostasis maintained. Abdomen closed in layers. Post operatively the patient recovered well except the patient had to stay for a long period because of daily pleural fluid collection in chest tube.

Discussion

Diaphragmatic injury accounts for about 0.8-1.6% of blunt trauma abdomen. Nearly about 4-6% of patients who undergo surgery for trauma have a diaphragmatic injury. 2 The most common injuries occur on left side, of about 68.5% of the patients and right side injuries accounts for 24.2%, and 1.5% had bilateral rupture, 0.9% had pericardial rupture, and 4.9% were unclassifi ed in the present collective review.[6] Many autopsy studies have revealed that incidence of rupture is almost equal on bilateral sides, but the greater force needed for the right rupture is associated with more grave injuries. A positive pressure gradient of 7-20 cm of H2O between the intraperitoneal and the intrapleural cavities forces the contents into the thorax. With severe blunt trauma, the pressures may rise to as high as 100 cm of water.7-8

Traumatic injuries to the diaphragm are of two types: 1) direct and 2) indirect. Direct injuries are usually penetrating injuries and are associated with less other visceral injuries whereas indirect injuries are usually blunt trauma to abdomen and thorax and they have poor prognosis as they are usually associated with cranial, skeletal, spinal and visceral injuries. Our case is a rare case of traumatic diaphragmatic hernia in which the mode of injury is indirect i.e blunt trauma to abdomen and thorax 5 years back. The patient was managed conservatively and the diaphragmatic injury remained undiagnosed without any acute symptoms.

The immediate and remote events that follow traumatic rupture of the diaphragm may be classified into three phases: (1) initial or acute, (2) latent and (3) obstructive. The initial phase begins with the original trauma and ends with the apparent recovery from the primary injuries. It is during this phase, when the management of the immediately recognizable injuries is paramount, that the diaphragmatic rent is likely to remain undetected, especially when rupture occurs after indirect blunt trauma. Diaphragmatic injuries are frequently discovered during abdominal or thoracic explorations for penetrating trauma. The latent period varies considerably; during this phase the patient often has vague upper abdominal distress due to intermittent incarceration of the herniated viscera. The obstructive phase may occur at any time to terminate the latent phase. Incarcerated viscera become obstructed, leading to congestion, strangulation, and necrosis if the condition is not promptly recognized and relieved. Symptoms are due to the space-occupying effects of the abdominal viscera and are modified only by the degree of protrusion into the thorax and the complications that may develop. During the initial phase, herniation of abdominal viscera may not be established sufficiently to cause distress. Gastrointestinal and cardiorespiratory symptoms become prominent during the latent phase. Vascular congestion may produce bleeding into the displaced stomach, small bowel, or colon. Dyspnea, cough, and palpitations due to pressure on the lung and heart are frequently noted. In the obstructive phase, obstipation, nausea, vomiting,and abdominal distention are often profound, indicating that intestinal obstruction has developed.

Physical Signs: Early in the initial phase, before abdominal viscera protrude into the thorax, physical signs are minimal. After herniation occurs, diminished respiratory excursion is often evident. Dullness or tympany to percussion, diminished to absent breath sounds borborygmi, and displacement of the cardiac dullness to the right are often recognized. Severe respiratory embarrassment may develop,

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often without a visible thoracic injury. The obstructive phase is characterized by signs of high or low gastrointestinal obstruction, depending on whether the stomach, small intestine, or colon is incarcerated. If the stomach is obstructed in the thorax, the abdomen may appear grossly normal. Some degree of abdominal distention develops when small bowel, colon, or both are involved, although if a significant portion of the intestinal tract is in the pleural cavity, the abdomen may be deceptively flat. In our case the patient c/o of vomiting and pain in left side of abdomen for last 7 days, h/o of trauma to left lateral side of abdomen 5 years back. On examination the tongue was dry and the skin was lax and pinch sign was positive suggesting the patient to be severly dehydrated. The abdomen was scaphoid. On palpation the abdomen was soft tender in left upper quadrant. The left lung was dull on percussion and the there was diminished breath sound on left lung. The apex beat was shifted to 5th intercoastal space near the left sternal border. There was no tracheal deviation. Initially it was very difficult to put the nasogastric tube but finally it was given successfully.

Early in the initial phase, films of the chest may not suggest diaphragmatic. rupture. However, serial roentgenograms often reveal an unidentified shadow above the diaphragm, indicating an abdominal viscus in the pleural cavity. Nasogastric intubation may clarify otherwise obscure findings if the tube passes into the stomach above the level of the diaphragm. The herniated stomach may give the appearance of an uncomplicated or even a tension pneumothorax and this possibility must be kept in mind if a tube thoracostomy is placed. If herniation has occurred, insertion of a chest tube will not relieve cardiorespiratory distress clinically or radiologically. The lack of relief should suggest the presence of a traumatic diaphragmatic hernia. Gastric dilation with elevation of the diaphragm may be confused with herniation, especially in the acute phase, shortly after injury. Chest films, especially in the latent phase, are often confusing. A homogenous density and displacement of the cardiac shadow to the right should suggest a diaphragmatic hernia. Multiple areas of air fluid loculation may give the impression of a primary lesion of the lung or pleural space. In our case we got confused with encysted hydrothorax and after diagnostic pleural tapping we got fluid which appeared purulent but the colour of fluid matched with the colour of ryle's tube aspirate which again suggested the diagnosis of diaphragmatic hernia. So we took decision for a CECT thorax and abdomen before further intervention. CECT thorax confirmed our diagnosis. Then we planned exploratory laparotomy for the repair of the diaphragmatic hernia. There is lot of debate about which approach should be preferred wheter to do thoracotomy for repair or laparotomy. In our case we preferred laparotomy because the patient had history of vomiting so we were concerened about any stricture in stomach or part of intestine and we aspirated the ryles tube by changing the posture of patient from supine to sitting position and after decompression of stomach we did repeat x ray and the position of ryles tube along with stomach changed which gave us an idea about the free mobility of stomach and we could probably asses that the tere is no adhesion of stomach to thoracic structures and also the literature supports that adhesion to thorax is unsual presentation. Thus for us it was better to have an abdominal approach and thus we gave a t incision in abdomen and explored it we decompressed the stomach and repositioned it in its usual position. Then chest tube was given in 6th intercoastal space under supervision through the defect and the defect was closed and a prolene mesh was given. We got a defect of size.... cmx ...cm in left posterior aspect of diaphragm because Wounds of the diaphragm do not heal spontaneously; often the omentum or other viscera plug the laceration, thereby preventing acute herniation. However, this same mechanism separates the muscle edges, preventing their union. Traumatic ruptures of the diaphragm are twelve times more common on the left side due to the protection afforded by the liver. Diaphragmatic tears are most common in the dome and the posterior half which are the areas of embryonic weakness. Then the stomach was found to be fibrosed and stenosed at pyloric region. So posterior gastrojejunostomy was done. Abdominal drain kit was given in left side of abdomen near the anastomotic site. The patient was shifted to ICU post operatively and was kept in positive pressure ventilation. On post-operative day 3 the patient was weaned from the ventilator. Post operatively the patient was resuscitated well with i.v fluids, blood transfusion, broad spectrum antibiotic coverage and chest physiotherapy and nutritional supplement. We faced only one problem which was prolonged drainage of pleural fluid and peritoneal fluid through the drains. The cause might be due to irritation from the prolene mesh or pancreatitis or decresed serum albumin.

However finally the patient was discharged with full recovery.

Conclusion

Knowledge of diaphragmatic hernia is essential for both the physician and the surgeon in atypical abdominal and respiratory discomfort, especially when there is history of trauma. This hernia is amenable to correction by minimal access surgery and requires a prompt diagnosis aided by a high index of suspicion.

References

[1] Lally KP, Lally PA, Van Meurs KP, et al. Treatment evolution in high-risk congenital diaphragmatic hernia. Ten years' experience with

DOI: 10.23958/ijirms/vol02-i4/04

diaphragmatic agenesis. Ann Surg 2006; 244(4):505–13.

[2] Skandalakis JE, Gray SW, Ricketts RR. The diaphragm. In: Skandalakis JE, Gray SW, editors, Embryology for surgeons, vol.1.

[3] Baltimore (MD) Williams & Wilkins; 1994.p.491-539 Hedrick HL, Crombleholme TM, Flake AW, et al. Right congenital diaphragmatic hernia: prenatal assessment and outcome. J PediatrSurg 2004; 39(3):319-23

[4] Cameron JL. Diaphragmatic injury. In: Current Surgical Therapy. 7th ed. St Louis, MO: Mosby-Year Book; 2001. p. 1095-100.

[5] Graivier L, Freeark RJ. Traumatic diaphragmatic hernia. Arch Surg. 1963; 86:363-73.

[6] Grimes of Traumatic injuries of the diaphragm. Diaphragmatic hernia. Am J Surg 1974; 128:175-81.

DOI: 10.23958/ijirms/vol02-i4/04