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HERNIATION THROUGH THE DIAPHRAGM INTO THE PERICARDIUM*

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Herniation through the diaphragm has been well documented in the literature and it has been established that various congenital anomalies of the diaphragm represent the basis for the vast majority of non-traumatic herniation. The usual textbooks of surgery, as well as articles on this subject, list the types of congenital diaphragmatic hernia with which we are all familiar, namely (a) through the hiatus pleuropertionalis foramen of Bochdalek, (b) through the dome of the diaphragm due to partial absence of the posterior portion of the muscle, (c) through the esophageal hiatus, and (d) through the foramen of Morgagni.

That herniation can occur through the middle leaflet of the central tendon with direct entrance into the pericardial sac has been rarely described. Indeed its occurrence is so unusual that it has not been included in the usual discussions on diaphragmatic hernia.

In 1948 Harrington† reported on his extensive personal experience with 400 cases of diaphragmatic hernia, not one of which included a defect in the pericardial sac. A collection of 821 cases reviewed by Hedbloom‡ in 1934 made no reference of herniation into the pericardial sac.

The Library of the American Medical Association has reports of eleven cases in the world literature. Of these, eight were autopsy findings of stillbirths or neonatal deaths in whom defects between the pericardial sac and diaphragm were associated with other anomalies of such magnitude as to be incompatible with life. The remaining three cases§,|| were successfully corrected surgically. Two of these were congenital defects in infants and one was a 48 year old African native with a history of trauma.

The case which we wish to present is that of a 53 year old woman who was admitted to the Stamford Hospital emergency room following an automobile accident. Besides the initial shock, which was promptly corrected, the injuries sustained consisted of compound comminuted fractures of the right tibia and fibula, comminuted fracture of the left radius and ulna, fractured 4th, 5th, and 6th ribs on the right, and multiple abrasions and contusions. An abdominal examination revealed no signs of internal injury and the lungs were clear and fully expanded. She was taken to the operating room where an open reduction of the compound fracture of the tibia was performed and the fractured radius and ulna were reduced. The patient remained in the hospital for a period of eight months during which time her entire care was orthopedic. Approximately one month after admission a chest film indicated a small pleural effusion on the left but was otherwise negative.

Thirteen months after the accident the patient was readmitted because of persistent post-prandial epigastric pain which radiated upward into the left chest. She reported that something seemed to "flop over" when she turned on her left side in bed.

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A chest film showed a large bizarre shadow filling the base of the left chest and containing fluid levels. The barium studies of the stomach, small and large bowel indicated that almost the entire stomach and a considerable portion of the transverse colon were in the left chest.

With a preoperative diagnosis of a traumatic diaphragmatic hernia the patient was taken to the operating room and the left chest was opened through the bed of the 8th rib. To our astonishment the left pleural cavity was normal, and the diaphragm

Figure 7
Preoperative x-ray of chest showing radiolucent shadows above the left diaphragm.

Figure 8
Preoperative barium enema.

Figure 1
Defect in the diaphragm containing the herniated stomach and colon.
Herniation

on this side was free of any defect; however, the pericardial sac was distended and upon palpation the presence of bowel could be detected within it. We therefore extended our incision across the costal margin into the abdomen. The true nature of our problem was then apparent. A large round hole was present in the central leaflet of the diaphragm measuring about 8 cm. in diameter with the left lobe of the liver entering it on its right side and with the stomach and colon filling the remainder of the defect (Fig. 1). There were no adhesions and no peritoneal sac. When the contents were reduced the heart was freely pulsating against the surface of the liver. The pericardium was smoothly continuous with the peritoneum (Fig. 2). A remarkable finding was the absence of a triangular ligament leaving the left lobe of the liver unsupported. Thus, this portion of the liver could be deflected into the opening. We elected to bridge this defect with a fascia lata graft which was readily done and when completed the liver assumed a position entirely covering the graft and conveniently served to buttress it. The patient’s postoperative course was uneventful and at the present time — one year later — there is no evidence of recurrence.

Figure 2

Defect as seen after removing herniated viscera showing peritoneopericardial communication and exposed heart.

To explain this hernia entirely on trauma leaves much to be desired. The smooth continuity between pericardium and peritoneum, the absence of adhesions, and the undeveloped triangular ligament argue strongly for a congenital anomaly. Yet it is difficult to understand how such a defect could persist for 53 years without producing
symptoms. However, O'Brien reports that during dissection of a 63 year old cadaver a similar defect was found in the central leaflet of the diaphragm. The abdominal viscera were prevented from entering the pericardium by the blockade produced by the left lobe of the liver.

A review of the embryological development of the diaphragm will explain the probable origin of this defect, illustrated in Fig. 3 to 6. The middle leaflet of the

![Diaphragm Diagram](FROM DODDS)

**Figure 3**
Upper peritoneal cavity (Coelomic Cavity) with pleural recess (shaded area) before development of pleuroperitoneal membrane.

![Diaphragm Diagram](FROM DODDS)

**Figure 4**
Pleural cavities separated from peritoneal cavities by pleuroperitoneal membrane.
Herniation

Figure 5 and Figure 6
Pericardial cavity decreases in size with enlarging pleural cavities but retains its relation to middle leaflet of diaphragm (former septum transversum).

central tendon of the diaphragm is formed from the transverse septum which early in development extends across the coelomic cavity. It is this structure which descends from the level of the 4th cervical myotome that carries with it the cervical nerve innervation to the diaphragm. The coelomic cavity above this septum remains as the pericardial sac after the pleural cavities expand on either side with their growing lungs. Fusing with the transverse septum is the pleuroperitoneal membrane which proceeds from the lateral wall. It is a double layered serous membrane, being pleura above and peritoneum below and between which the striated muscle extends to the central tendon. Therefore a defect in this transverse septum or future central tendon will be distinctly different from failure of some portion of the pleuroperitoneal membrane. The former will necessitate a defect into the cephalic coelomic cavity or future pericardium. The latter defect would be through the muscular diaphragm into the pleural cavity.

It is suggested that this case represents a very unusual defect in the diaphragm due to failure of the developing transverse septum to separate the two portions of the coelomic cavity. The congenital basis for the defect is supported by the failure to find evidence of trauma at surgery plus the absence of a triangular ligament. Nevertheless, the relationship of trauma cannot be denied and must be assumed to be responsible for forcing the bowel into a preexisting defect.

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