9

Benign Disease of the Diaphragm

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Aims

- To describe diaphramatic eventration and phrenic nerve palsy.
- To describe the diagnosis and management of traumatic diaphragmatic hernias.
- To discuss diagnosis and management of congenital diaphragmatic hernias.

Surgical Anatomy of the Diaphragm

The diaphragm consists of peripherally placed muscular elements that radially insert into the domed, trefoil-shaped central fibrous tendon. The muscular portion consists of three parts, lumbar, costal and sternal, which are separated by muscle-free gaps. These gaps consist of little more than loose connective tissue, pleura and peritoneum. The lumbar muscular part is the strongest and arises from the anterior surface of the upper lumbar vertebrae and intervertebral discs, the crura and arcuate ligaments. The costal part originates from the cartilages of the lower six ribs anterolaterally, interdigitating with muscular slips from the transversus abdominis muscles. The gap between the lumbar and costal elements represents the site of the lumbocostal (Bochdalek's) foramen. The sternal origin arises from the posterior part of the rectus sheath and xiphoid process. The gap between the sternal and costal elements is referred to as the foramen of Morgagni. These foramina are illustrated in Figure 9.1.

The position of the central tendon depends on many factors. These include the respiratory cycle, body habitus and the degree of abdominal distension. During full expiration the dome of the right hemidiaphragm lies approximately at the level of the fourth intercostal space

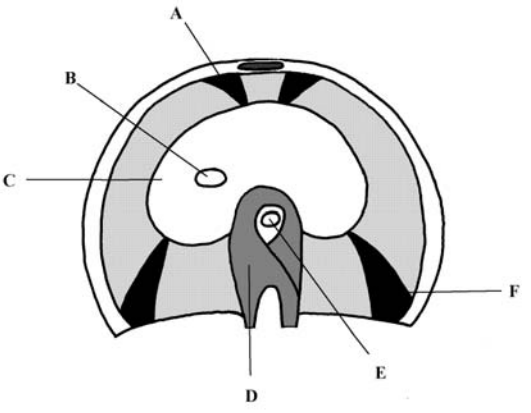

Figure 9.1. Diagram illustrating the position of the common congenital diaphragmatic hernias (viewed from below). A, sternocostal foramen (Morgagni hernia); B, inferior vena cava; C, central tendon of the diaphragm; D, crura and oesophageal hiatus; E, oesophagus; F, lumbocostal foramen (Bochdalek hernia).

and the left hemidiaphragm is usually a space lower. In forced inspiration the domes may move downwards by as much as two intercostal spaces, the central tendon flattens and the costodiaphragmatic recesses enlarge, enabling downwards excursion of the lungs.

The vascular supply to the diaphragm arises from several sources. The peripheral muscular parts are supplied by branches from the lower five intercostal and the subcostal arteries. The pericardiacophrenic arteries, which are terminal branches of the internal mammary artery, supply the fibrous pericardium, phrenic nerve and a small portion of the central tendon. Further blood supply is via the musculophrenic and superior phrenic arteries, all of which supply the cranial aspect of the diaphragm. The posterior aspect is directly vascularised by small branches of the descending thoracic aorta, whilst the caudal aspect is supplied from the inferior phrenic arteries and direct branches from the coeliac trunk.

The nerve supply to the diaphragm reflects its origin as a cervical structure. The primary motor innervation is via the right and left phrenic nerves (C3–5). Both phrenic nerves supply the diaphragm from below, the right passing through the caval foramen, and the left piercing the muscular part anterolateral to the pericardium. The nerves branch into sternal, anterolateral, posterolateral and crural branches that spread radially to the peripheral musculature. The lower intercostal nerves also supply branches to the peripheral muscular parts, but these are primarily proprioceptive rather than motor.

Diaphragmatic Incisions

The radial arrangement of the diaphragmatic neurovascular supply has implications for the placement of incisions. Peripheral circumferential incisions should be placed approximately 3 cm from the costal margin to avoid the radially-placed neurovascular bundle (Figure 9.2). Radial incisions should be limited to the anterolateral portion of diaphragm and not extended back to the hiatus if possible, as this can compromise a significant proportion of phrenic nerve branches. Incision through the central tendon must be located well away from the main phrenic nerve [1,2].

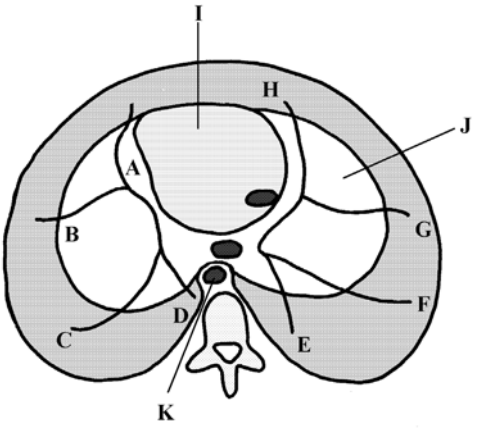

Figure 9.2. Diagram illustrating the branches of the phrenic nerve (viewed from above). A, left sternal; B, left anterolateral; C, left posterolateral; D, left crural; E, right crural; F, right posterolateral; G, right anterolateral; H, right sternal; I, fibrous pericardium and inferior vena cava; J, central tendon of the diaphragm; K, aorta.

Openings in the Diaphragm

There are three main openings through, or in the case of the aortic foramen, behind the diaphragm, with a variable number of other structures that pass from abdomen to thorax. The caval foramen is located on the right at the level of T8 within the central tendon. On the left is the oesophageal foramen, at the level of T10. This is formed by the right crus with contribution from the left crus anteriorly. The aortic opening lies behind the diaphragm at its lowest point, opposite the T12 vertebra. The aortic opening is bounded by the interdigitating crura and median arcuate ligament anteriorly and the vertebral column posteriorly. The contents of each foramen and a summary of other structures that traverse the diaphragm are listed in Table 9.1 [1,3].

Congenital Diaphragmatic Hernias

The first description of a congenital diaphragmatic hernia (CDH) has been attributed to Riverius in 1679 [4,5]. Morgani and Bochdalek subsequently described their eponymous hernias in 1769 and 1840 respectively [4]. The

Table 9.1. Summary of structures passing from the abdomen to thorax via the diaphragm

Diaphragmatic foramen	Position	Contents
Caval	Central tendon opposite T8	Inferior vena cava Right phrenic nerve[a] Lymphatic vessels
Oesophageal	Between right and left crus at level of T10	Oesophagus L and R vagal trunks Oesophageal branches of L gastric artery + veins/lymphatics. Phrenicoabdominal nerve
Aortic	Behind diaphragm at the level of T12	Descending thoracic aorta Aortic plexus Azygos vein Thoracic duct
Structures crossing the diaphragm via other openings		Greater, lesser and least splanchnic nerves Sympathetic trunk Subcostal neurovascular bundle Lower five intercostal nerves Inferior hemiazygos vein. Left and right* phrenic nerves Superior epigastric vessels Lymphatics

* Some texts state that the right phrenic nerve pierces the diaphragm next to the caval foramen rather than passing through it. [1,4].

first successful repair of a neonatal CDH is attributed to Gross in 1946 [6].

Congenital diaphragmatic hernias are uncommon with a prevalence of between 1 in 2000 and 1 in 5000 overall. Most occur sporadically although a few cases may be seen as part of a familial condition, Fryns syndrome [7]. The male to female ratio is equal for the more common forms of CDH. Small hernias may escape detection in the neonatal period, presenting later in life. Congenital diaphragmatic abnormalities can be classified in a variety of ways depending on whether embryological, anatomical, or clinical criteria are used. This can cause confusion: for example some texts consider pleuroperitoneal canal defects synonymous with Bochdalek's hernia. Table 9.2 summarises the more common defects and their main features.

The most common form of CDH is the posterolateral Bochdalek hernia, which involves the left side in 80% of cases. Most of what follows regarding prognosis and treatment refers to this type of CDH. Pleuroperitoneal canal and septum transversum defects are far less common. The advent of routine prenatal ultrasonography has resulted in the majority (>90%) of cases of CDH being picked up before the 25th week of gestation. The prevalence of associated congenital malformations is variable, and their presence greatly influences outcome and survival. Most common are defects affecting the heart, brain, genitourinary system and limbs [8] The incidence of potentially lethal associated chromosomal anomalies (including trisomy 13, 18 and 21) ranges between 30% and 50% in most studies, and [8]. A recent study has proposed an association between CDH and anomalies affecting the long arm of chromosome 15 (15q24–26) [9]. The chest x-ray in Figure 9.3 illustrates the appearances of a large right diaphragmatic hernia with associated pulmonary hypoplasia in a neonate.

Much of the morbidity and mortality of CDH results from induced changes in the cardiopulmonary circulation. A widely accepted explanation for these changes is that pulmonary development, particularly bronchial branching, is impeded by the mass effect of herniated abdominal viscera, resulting in alveolar hypoplasia and pulmonary hypertension. This theory is supported by the finding that the

Table 9.2. Summary of developmental anomalies affecting the diaphragm

Condition	Anatomical Features	Epidemiology
Diaphragmatic agenesis	Complete absence of diaphragm	Very rare defect
Diaphragmatocele	Failure of muscle development Diaphragm consists of fibrous sheet only	Very rare defect
Eventration	Muscular part of diaphragm deficient with normal but sparsely distributed muscle cells. Phrenic nerves normal. Diaphragm sits in elevated position and attenuated muscle allows abdominal viscera to bulge into thorax	More commonly seen in males. Associated with malrotation of gut in proportion of cases
Pleuroperitonal canal defect	Also known as "hernia diaphragmatica spuria". The canals fail to close in week 8 leaving a defect in the lateral muscular part of the diaphragm	Rare defect
Bochdalek hernia	True hernia through the lumbocostal triangle. More than 85% on left. Associated with pulmonary hypoplasia, malrotation of the gut, tracheo-oesophageal fistula and cardiac defects	Approximately 1:4000 all births. Equal sex incidence
Morgagni hernia	Retrosternal hernia through the right sternocostal gap. Sac initially present but may regress and be difficult to identify. Commonly presents late and may be exacerbated by trauma. Left sternocostal hernia often known as Larrey's hernia	Uncommon
Septum transversum defects	Failure of development that affects both diaphragm and pericardium	Very rare

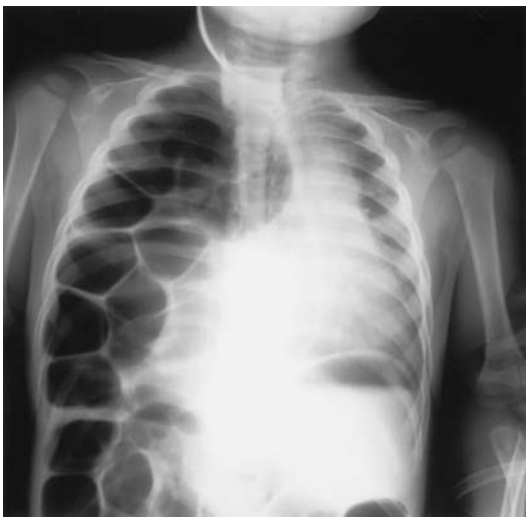

Figure 9.3. Chest X-ray of a large right diaphagmatic hernia in a neonate.

degree of hypoplasia and overall mortality relate to the size of the diaphragmatic defect, and the time that it develops in relation to gestational age. However, pulmonary hypoplasia is also seen in the lung contralateral to the CDH. Some animal experiments have suggested that lung hypoplasia occurs simultaneously with the diaphragmatic malformation rather than as a result of it [10]. The pulmonary circulation is also affected by the presence of a hernia. Pulmonary artery branching is intimately related to bronchial development: reduced branching results in arterial hypoplasia and excess arteriolar wall muscle development. This in turn contributes to the development of pulmonary hypertension [11].

During pregnancy fetal oxygenation is maintained by the placenta. A right to left shunt across the foramen ovale ensures that most of the lungs are bypassed. The first few gasps of air at the time of birth reduce pulmonary resistance and raise the oxygen tension in the pulmonary

veins, inducing closure of the foramen ovale and ductus arteriosum. In neonates with CDH the conversion from a fetal circulation is opposed by hypoxaemic pulmonary vasoconstriction secondary to alveolar hypoplasia. Persistence of the fetal circulation induces a vicious cycle of increasing hypoxia and pulmonary hypertension that invariably results in critical respiratory failure. This may be further compounded by the presence of associated cardiac abnormalities.

Perinatal Management and Timing of Surgery

With improvements in diagnosis, paediatric anaesthesia and intensive care the number of neonates surviving surgical repair of CDH has steadily increased [5]. There is a greater understanding of the pathophysiological cardiopulmonary changes that accompany CDH, and more effective treatments for conditions often seen in association with CDH, such as heart disease. However, the morbidity and mortality of infants with CDH remains substantial.

It was initially believed that the poor outcome associated with CDH was predominantly a result of continued compression of the lung after birth. Surgery was therefore undertaken immediately after delivery to minimise this effect. Experimental work in the 1980s suggested that some of the consequences of CDH could be further reduced if the diaphragmatic repair was undertaken prenatally. Although encouraging results were reported in animal models, the results of surgery in humans have been disappointing. The biggest obstacle was how to avoid of inducing preterm labour in the mother, which was seen in almost all pregnancies.

An improved understanding of the relationship between pulmonary hypoplasia, hypertension and outcome has led to a shift in management. It is now accepted that the optimal approach is to undertake surgery once cardiac and respiratory function has been optimised and stabilised in the first few hours of life. The primary goal is correction of hypoxaemia through ventilation without barotrauma, thereby interrupting the vicious cycle of hypoxaemia, pulmonary vasoconstriction and reduced pulmonary compliance that is otherwise seen in the immediate postnatal period. Thoughts have also changed regarding the methods of ventilation, with a shift away from aggressive hyperventilation. Instead, the goal is to maintain oxygenation with the minimum of ventilatory pressure, and a degree of permissive hypercapnia. Introduction of this approach appears to have reduced the morbidity and mortality associated with pulmonary barotrauma [5]. Other novel ventilatory methods that have been investigated in CDH include high frequency oscillatory ventilation (HFOV) and extracorporeal membrane oxygenation (ECMO).

Extracorporeal membrane oxygenation is a method of cardiopulmonary bypass that enables arterial blood oxygenation and removal of CO_2 via an extracorporeal venoarterial or venovenous circuit. By reducing pulmonary hypoxaemia without the need for mechanical ventilation, the problems of pulmonary hypertension and barotrauma are avoided. Although an established treatment in other forms of neonatal respiratory distress, the use of ECMO is associated with significant morbidity and mortality in its own right, predominantly due to bleeding complications and neurological injury. There is also some controversy regarding patient selection, optimum timing and duration of ECMO treatment. Overall the impact of perioperative ECMO on survival in CDH has been investigated in several units and remains unproven [5,11].

As the vast majority of CDH are diagnosed prenatally this enables some degree of planning, as far as delivery is concerned. Clinical signs at birth include respiratory distress associated with a scaphoid abdomen, evidence of intrathoracic stomach or bowel, and signs of mediastinal shift [11]. Immediately following delivery a nasogastric tube should be passed to decompress the stomach and intestine, thereby reducing the risk of aspiration and intrathoracic strangulation of abdominal viscera. The neonate should then be intubated and ventilated to maintain arterial oxygenation, and an appropriate attention paid to fluid balance and temperature control. Prolonged ventilation via a facemask produces gastric distension and predisposes to aspiration, and should therefore be avoided. The presence of associated defects should be ascertained and investigated as appropriate. Transfer to a specialist centre is essential once the neonate is stabilised.

Surgical Procedures and Outcome

Surgical repair involves reduction of herniated viscera into the abdomen, with resection of the hernial sac and diaphragmatic repair. Morgani hernias are usually small and can be closed without a patch. It is often more difficult to close larger defects primarily, in which case surgical mesh or autologous muscle flaps are options. Surgery is usually performed through an abdominal rather than thoracic approach, and may need to be combined with other procedures, e.g. correction of intestinal malrotation.

The results of surgery for CDH remain disappointing despite advances in perioperative management. Much of this mortality relates to the presence of other associated congenital defects. Some bias has probably been introduced by better survival of the sickest infants, who previously would have died shortly after birth. Earlier identification and better perinatal management enables these infants to survive long enough to become potential surgical candidates. Overall survival rates approach 50%, with results from centres that routinely use ECMO reported as higher, in the region of 65% [11]. Long-term complications of surgery for CDH include patch disruption, recurrent herniation, and chest wall deformity. It is also common for children with CDH to suffer with chronic gastro-oesophageal reflux disease, probably as a result of impaired diaphragmatic motility [12]. The effect of correction on pulmonary function is unpredictable and a proportion of surviving infants remain respiratory cripples [11]. Such children inevitably require long-term follow-up by a multidisciplinary team to enable effective management of their many medical problems.

Eventration of the Diaphragm and Phrenic Nerve Palsy

Diaphragmatic eventration is an uncommon condition that can mimic both CDH and traumatic herniation. Eventration is caused by a paucity or absence of the muscular parts of one or both hemidiaphragms, which is otherwise normally innervated. The peripheral muscle is unable to contract adequately against the upward force of the abdominal viscera, gradually becoming stretched and attenuated until the dome of the diaphragm lies at an elevated position. In distinction from hernias, the muscular elements are intact and in continuity with the chest wall. Eventration is more common in males, and is associated with malrotation of the gut and possibly other congenital myopathies. The majority of cases affect the left side. The underlying cause of eventration is unclear. There appears to be an association with CDH and it has been suggested that premature return of abdominal contents during fetal development may compromise muscular growth. Complete eventration, in common with CDH, is associated with ipsilateral pulmonary hypoplasia. Histological examination of eventrated diaphragm shows muscle cells to be present but sparsely distributed with associated scarring, inflammation and fibrosis [13].

Phrenic nerve palsy can result in a clinical and radiological appearance that may be difficult to distinguish from eventration on clinical or radiological grounds. The nerve palsy may be congenital or acquired, but with time leads to atrophy of the muscular elements resulting in elevation of the central tendon. True congenital phrenic palsy is uncommon, with a reported incidence of 0.03–0.5% of neonates [4]. However, acquired palsy can result from numerous pathological processes, which include neuromuscular disorders such as poliomyelitis, neoplastic invasion, trauma and iatrogenic injury. In children phrenic nerve palsy is a recognised complication of perinatal trauma and congenital heart surgery [1].

Symptoms and Diagnosis

Minor degrees of eventration may be asymptomatic. More severe forms usually present with breathlessness secondary to pulmonary compression, particularly in the supine position. Both eventration and phrenic palsy can have serious consequences in the newborn. The accessory muscles of respiration are poorly developed in infants, who are consequently far more reliant on diaphragmatic contraction than are adults. In addition, the thoracic cage is softer and therefore more compliant. Respiratory distress may develop rapidly and the effects of

diaphragmatic paralysis are compounded by the presence of paradoxical respiration in the supine position. Abnormal outwards excursion of the lateral chest wall during inspiration due to unopposed intercostal muscle contraction may be clinically apparent. The other common presentation of eventration relates to the digestive system, with symptoms of reflux, belching and vomiting, and poor feeding in children. More serious consequences include the development of gastric volvulus or strangulation [13].

The diagnosis of eventration and/or phrenic palsy is usually suggested by the presence of an elevated hemidiaphragm on standard posteroanterior and lateral chest radiography. The diaphragmatic contour is unbroken, in distinction from CDH or traumatic hernia, and the gastric fundus is in a subdiaphragmatic position. These findings can be confirmed by computed tomography. Diaphragmatic movement is best confirmed by fluoroscopy, with normal but reduced movement seen in eventration. In contrast phrenic nerve palsy is associated with true paradoxical movement, i.e. elevation during inspiration. The diagnosis may only be confirmed beyond doubt at surgery via thoracoscopy or thoracotomy, at which point the integrity of the diaphragm can be confirmed. The phrenic nerve can also be assessed by direct stimulation.

Surgical Management

The need for surgical intervention for either eventration or phrenic palsies depends on many factors. In infants the need for surgery is high in all but the most minor of cases, for the reasons listed earlier in this section. Acquired phrenic nerve palsies in infants are twice as likely as congenital palsies to require surgical intervention. As with CDH the priority should be stabilisation and ventilatory support in the first instance, with surgical repair undertaken once this has been achieved, usually within 2 weeks of the commencement of mechanical ventilation. In adults surgery is reserved for those with symptoms of dyspnoea or gastrointestinal disturbance after exclusion of other underlying pathologies.

Surgical treatment of eventration is primarily that of diaphragmatic plication via an open or thoracoscopic approach. The slack muscle and redundant central tendon are gathered in a series of radial pleats located to avoid the branches of the phrenic nerve [14]. The pleats are formed by the use of deep mattress sutures using heavy non-absorbable sutures. These may need to be buttressed with Teflon as the diaphragmatic tissue is often thin [15]. An alternative method that is suitable for localised eventration is to resect the affected part of the diaphragm and oppose normal edges in a two-layer repair [13]. Diaphragmatic plication has also been described via a thoracoscopic approach [16]. With both methods protection of underlying viscera and avoidance of excess tension are paramount.

Results of Surgery

In both infants and adults it is essential to exclude other causes of dyspnoea, e.g. congenital cardiac disease or pulmonary conditions, and to correct exacerbating factors such as obesity, wherever possible. The results of surgery for eventration in infants are good with low perioperative morbidity and mortality and good functional results in the longer term [13]. In adults the results in selected patients also appear good, with demonstrable and prolonged improvement in respiratory function and symptoms [15,17,18].

Traumatic Diaphragmatic Rupture

Incidence and Aetiology

Diaphragmatic hernia (rupture) is a relatively uncommon and frequently undiagnosed sequel to both blunt and penetrating trauma involving the upper abdomen and thorax. First descriptions of this condition are attributed to Paré and Sennertus in the sixteenth century [19]. It was not until the nineteenth century that surgical treatments were attempted [20]. The true incidence of diaphragmatic rupture can be difficult to define because of the association with multiple injuries and tendency for late presentation. The incidence appears to be rising. However, it is unclear whether this is a true increase, or a reflection of increased awareness, improved diagnosis or better survival in polytrauma patients. Mansour cites an incidence of 0.8–1.6% in blunt thoracoabdominal trauma, rising to between 4 and 6% in those undergoing

laparotomy or thoracotomy for trauma [19]. Rosati cites an incidence of up to 7% in blunt trauma, rising to 10–15% in penetrating thoracoabdominal trauma [20]. An injury scoring system specific to the diaphragm has been devised by the Organ Injury Scaling Committee of the American Association for the Surgery on Trauma. This grades the injury on a scale I–V depending on the nature of the injury (contusion versus laceration), the size of the defect and the total amount of tissue loss [21].

There are two potential mechanisms of injury in blunt trauma. One is the forceful herniation of contents through one of the weaker areas of the diaphragm, e.g. lumbocostal foramen. The other is a radial tear at the musculotendinous boundary of the diaphragm secondary to a sudden increase in intra-abdominal pressure against a closed glottis. Under normal circumstances a pressure differential of up to 20 mmHg exists across the diaphragm. However, during coughing or straining, the transdiaphragmatic pressure difference can rise to more than 100 mmHg. The forces acting on the chest and abdomen during road traffic accidents or falls may momentarily reach ten times this force [4]. Once the initial tear has been caused the influence of the transdiaphrgamatic pressure gradient, combined with the effects of coughing etc., will further widen the defect, pushing abdominal viscera into the chest.

Spontaneous healing of diaphragmatic injury does not occur. However, small defects may be temporarily plugged with omentum, preventing early visceral herniation. Less commonly direct trauma produces dehiscence of the muscular parts of the diaphragm from the chest wall. Diaphragmatic ruptures appear to be far more common on the left (80–90% of reported cases) with a small percentage bilateral (1–5%) [22]. However, the incidence of right-sided ruptures is significantly higher in some series, particularly those that include post-mortem findings [23].

Most large studies have shown that up to 40% of subsequently confirmed diaphragmatic ruptures are diagnosed preoperatively, with a similar proportion found unexpectedly at the time of thoracotomy or laparotomy [22,24]. The remaining cases have a delayed presentation: a small defect enlarges with time until the signs and symptoms of pulmonary compression, visceral strangulation, perforation or haemorrhage become apparent. Herniation may also occur after penetrating injury to the central tendon. Because of the domed shape of the diaphragm, the path of a penetrating object may cause an injury in more than one place, and small tears may be easily missed at laparotomy or thoracoscopy/thoracotomy. Diaphragmatic rupture has also been described spontaneously and in pregnancy, particularly during labour.

The overall mortality for patients with diaphragmatic rupture is fairly constant in several series, at 10–20% [4,20,22,23]. The majority of early fatal cases are secondary to associated injuries, particularly those involving the thorax and abdomen, as this group of patients have been shown to have high overall injury severity scores.

Clinical and Radiological Diagnosis

The diagnosis of traumatic diaphragmatic rupture requires careful assessment and a high index of suspicion in patients with an appropriate mechanism of injury. It can be obscured by the presence of associated injuries which can be life-threatening in their own right. It has been estimated that between 7 and 66% of patients with polytrauma have a diaphragmatic rupture which is initially missed or misdiagnosed [24]. Correct diagnosis relies heavily on radiological investigations. Chest X-ray (CXR) is the most commonly available, and is very useful as an initial screening tool. The passage of a nasogastric tube helps to confirm the position of the stomach. Radiological features range from obvious loss of diaphragmatic contour associated with displacement of the stomach or bowel into the chest, through to more subtle signs. These include irregularity or elevation of the diaphragm and lower lobe atelectasis. These features can be misinterpreted as, or concealed by, those of a loculated hydrothorax. The CXR appearances of a left traumatic diaphragmatic hernia are shown in Figure 9.4. Overall CXR is diagnostic or suggestive of diaphragmatic rupture in 28–64% of cases [25]. Ultrasonography is valuable in confirming the diagnosis of diaphragmatic rupture, and has the advantages of being safe, portable, repeatable and readily available in most hospitals. The diagnostic sensitivity of ultrasound has been estimated at up to 82% [23]. However, it is less useful in the

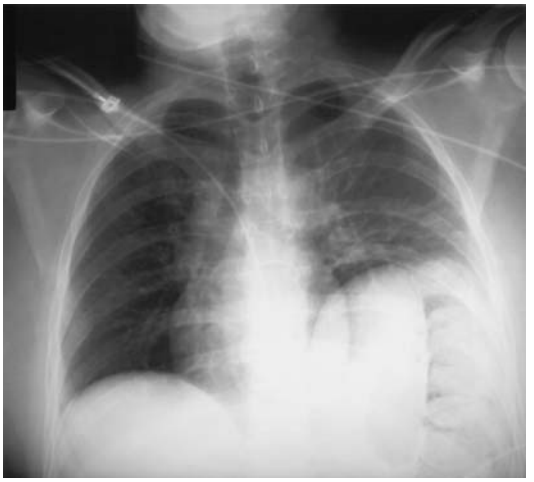

Figure 9.4. Chest X-ray of a left traumatic diaphragmatic hernia.

presence of significant chest wall trauma, surgical emphysema or pneumothorax.

Computed tomography (CT) is another diagnostic modality that is readily available in most hospitals and is commonly used to assess patients with thoracic and abdominal trauma. The main disadvantage of CT is that the diaphragm itself is difficult to directly image because of its axial position, and cannot be accurately distinguished from the liver on the right. Sagittal reconstructions and spiral CT, which are now becoming more widely available, are of greater value. Overall CT has been found to have a diagnostic sensitivity of 33–83% and specificity of 76–100%, and is considered the gold standard for diagnosing chronic herniation [4]. Magnetic resonance imaging (MRI) has the advantage of being able to produce sagittal and coronal images that facilitate the diagnosis of diaphragmatic injury. Unfortunately MRI is less available than CT in most centres, and excludes the examination of unstable patients requiring monitoring or ventilation because of the effects that the associated magnetic field has on metallic objects.

Laparoscopy and thoracoscopy have both been investigated in the diagnosis of diaphragmatic rupture. Smith and colleagues have reported their results using laparoscopy in 133 patients with thoracocabdominal injury [26]. They were able to identify and repair a diaphragmatic injury in only four cases (3%). The laparoscopy was diagnostic only, with no injury of any type identified, in over half of the cases ($n = 72$, 54%). This study excluded patients with cardiorespiratory instability and complex trauma, who are the group most likely to have sustained a significant diaphragmatic injury. The disadvantages of laparoscopy are that it requires a general anaesthetic and the induction of a pneumoperitoneum. Both of these can exacerbate cardiorespiratory instability, and the latter can precipitate tension pneumothorax in the presence of a diaphragmatic defect. The technique is expensive, operator-dependent and poor at visualising the right hemidiaphragm. Thoracoscopy has the advantage of better visualisation of either hemidiaphragm, but requires that the patient can tolerate single-lung anaesthesia, and can be hindered by the presence of pulmonary injury of intrathoracic adhesions. Overall it would seem that neither technique is of great value as a routine screening tool in the evaluation of diaphragmatic trauma per se.

Surgical Management

In contrast to congenital hernias, diaphragmatic ruptures are better approached from the thorax [4]. The absence of a hernial sac and the presence of associated pulmonary and chest wall injuries predisposes to adhesions that may require careful dissection before the herniated organs can be returned to the abdomen. Thoracotomy or thoracoabdominal approaches are therefore recommended, and the latter has the advantage of enabling abdominal exploration at the same time. Most diaphragmatic lacerations or tears can be repaired directly with a one- or two-layer technique using non-absorbable suture. Peripheral injuries may require reattachment of the diaphragm to the chest wall. Chronic large defects occasionally have to be repaired using a prosthetic patch.

The Chronic Sequelae of Missed Traumatic Diaphragmatic Rupture

Despite the fact that a proportion of diaphragmatic injuries may initially be missed, most will eventually become clinically apparent in patients who survive. Symptoms may reflect pulmonary complications such as basal atelectasis, hydrothorax or mediastinal and pulmonary compression. Alternatively gastrointestinal symptoms secondary to visceral displacement,

incarceration, strangulation, perforation or haemorrhage may predominate. This may produce a diagnostic conundrum as the symptoms may not localise to the abdomen. Only a small percentage of hernias will remain asymptomatic long term. Of those that do result in strangulation, the majority (85%) occur within 3 to 5 years of the initial injury [27,28]. For this reason all traumatic diaphragmatic hernias should be electively repaired once the patient's condition has been stabilised.

Summary

Conditions affecting the diaphragm are generally uncommon in surgical practice but can affect all ages. The consequences of diaphragmatic herniation of any cause can be life-threatening, yet the diagnosis may be obscured by associated conditions and the effects of trauma. A thorough understanding of the developmental anatomy of the diaphragm aids diagnosis and facilitates surgical repair.

Questions

1. Name the foramina of the diaphragm and structures passing through.
2. On which side do Bochdalek hernias most frequently occur?
3. Describe the cardiopulmonary changes that may be induced by a congenital diaphragmatic hernia.
4. How is the diagnosis of eventration made?
5. Describe the surgical treatment of traumatic hernias of the diaphragm.

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