12. Minimally Invasive Approaches to Congenital Diaphragmatic Hernias

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Introduction

Epidemiology

Congenital diaphragmatic hernia (CDH) occurs in approximately 1 in 2000–3000 live births [1, 2]. 85 % of cases occur on the left, 13 % occur on the right, and 2 % are bilateral [2]. 70 % of the defects are posterolateral or Bochdalek type, 27 % are anteromedial or the Morgagni type, and the remaining cases are considered complete agenesis [1].

Pathophysiology

CDH is the result of failure of the diaphragmatic musculature to fuse during gestation, allowing herniation of abdominal viscera into the thoracic cavity. Anatomically, this results in pulmonary compression with subsequent hypoplasia and arteriole muscular hypertrophy. Postnatally, decreased surface area and hypertrophied and hyperreactive pulmonary arterioles lead to fixed increased vascular resistance and pulmonary hypertension, the primary source of morbidity and mortality in these patients [1, 3].

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Preoperative Evaluation

History

Approximately 66 % of CDH in the developed world are diagnosed by ultrasound in the prenatal period [1]. 40 % of CDH patients have concurrent congenital anomalies, particularly cardiac defects, which are also detected with prenatal ultrasound [1, 2]. Several prenatal measurements have been proposed for determination of CDH severity: lung to head ratio (LHR), observed to expected LHR, total lung volumes (TLV) by MRI, and observed to expected TLV. Each has significant prognostic association but high interobserver variability [4]. When CDH is diagnosed in utero, delivery at or rapid transfer to a tertiary facility equipped for neonatal intensive care, particularly one with ECMO capabilities, is recommended [3].

Exam

Patients with CDH not diagnosed prenatally generally present with respiratory distress. CDH may be suspected by a paucity of breath sounds on the affected side, shifted heart sounds, or the presence of bowel sounds over the lung fields. The abdominal exam demonstrates a normal to scaphoid abdomen [3]. When the diagnosis of CDH is suspected, respiratory status should be carefully assessed. Tachypnea, cyanosis, delayed capillary refill, and progressive hypoxia are indicators of worsening pulmonary hypertension necessitating prompt resuscitation.

After stabilization of the infant, a full physical exam is used to rule out concurrent anomalies, particularly facial dysmorphism, neurologic defects, genitourinary malformations, digital abnormalities, hypertelorism, or organomegaly, which may prompt diagnostic workup for a systemic genetic syndrome [5, 6].

Imaging

Chest X-ray supports the diagnosis of CDH and demonstrates presence of abdominal viscera in the thoracic cavity. If the diagnosis is uncertain, ultrasound may assist in the diagnosis of right-sided defects in particular by identifying the location of the hepatic vasculature. CT or MRI may be used to evaluate cases in which uncertainty about the diagnosis persists.

Echocardiogram, perhaps the most important part of the evaluation, is performed for assessment of pulmonary hypertension and cardiac function and to rule out concurrent congenital cardiac defects.

Postnatal Management

Clinical respiratory status should be carefully assessed and arterial blood gas should be performed [7]. If respiratory function is compromised, prompt intubation is warranted, as this population is known to recover poorly from hypoxia-induced acidosis [3]. Bag-valve mask should be avoided as inflating the intestines may increase pressure on the compromised lungs [8]. A strategy of permissive hypercapnia provides gentle ventilation to support respiratory function while minimizing the risk of barotrauma [3, 5, 7, 8]. High-frequency oscillatory ventilation (HFOV) is used by many centers as primary therapy or as rescue therapy prior to ECMO cannulation [5, 8]. Vasodilators are used with increasing frequency to treat pulmonary hypertension in affected infants; nitric oxide has failed to demonstrate improved outcomes, but small studies show a mortality benefit with use of sildenafil [9–11]. In severe cases, ECMO is the most effective method of providing cardiorespiratory support. ECMO use has varied widely between centers, from 11 to 58 % historically. Its use contributed significantly to the increase in survival in this population, although improved neonatal ventilator therapy in recent years has decreased the necessity for ECMO [7, 12]. While some choose to perform repair on ECMO, we prefer to wait until after stabilization of the patient following decannulation. These patients generally have larger defects and are more likely to require patch repair. In our experience, thoracoscopic repair after ECMO is feasible, though conversion to laparotomy is more common in this patient subset.

Surgical Indications

CDH was previously treated as a surgical emergency with all patients undergoing repair within the first 24 h of life. Many studies have since demonstrated high rates of respiratory failure and subsequent morbidity and mortality in patients undergoing early repair, prompting most surgeons to delay surgical intervention until respiratory status has been stabilized. Nevertheless, recent data has questioned the actual clinical benefit of the generally accepted delayed repair [13].

Most centers use physiologic criteria to determine readiness of the CDH patient for surgery, whether or not the patient previously required ECMO. Stabilization of respiratory status is determined by minimal clinical evidence of pulmonary hypertension, preductal saturation between 85 and 95 % on <50 % inspired O_2, no preductal or postductal SaO_2 gradient, and/or mean arterial pressure (MAP) that is normal for gestational age [1, 2, 8, 14].

Indications

With rare exception, all CDH patients who are clinically stable to undergo surgical repair are candidates for attempted thoracoscopy. Exceptions include those not able to be weaned off of ECMO and those with unrepaired severe complex cardiac disease or non-resolving pulmonary hypertension [15, 36, 37].

Technique

Special Considerations

Thoracoscopic repairs are more challenging technically and more dangerous for labile infants. CO_2 insufflation increases pressure on hypoplastic lungs. Hypercarbia necessitates increased respiration, increasing the risk of barotrauma, hypoxemia, and acidosis [15].

Patient Positioning and Prep

We will describe the technique for repair of left-sided CDH, as it is the more common type.

1. Place the patient on the operating room table in one of two positions (Fig. 12.1):
   (a) Horizontally at the foot of the bed with the surgeon at the patient's head facing the screen and the assistant at the foot of the bed on the patient's left side
   (b) Longitudinally at the head of the bed with all extraneous pieces of the bed broken down with the surgeon at the patient's head and the assistant on the left

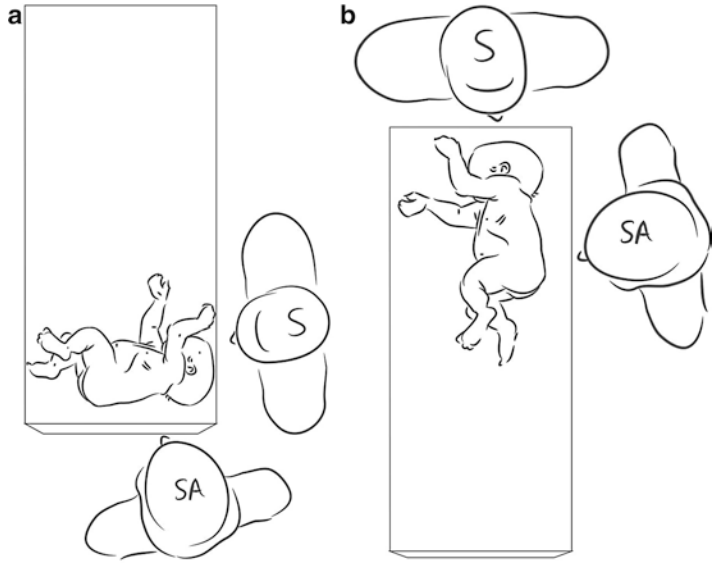

Fig. 12.1. Two variations on standard positioning for thoracoscopic repair of L CDH: option 1 (**a**) and option 2 (**b**).

2. Position the patient in the lateral decubitus position with the affected side up and slightly angled toward supine.
   (a) Coordinate carefully with anesthesia to ensure that the patient's head and the joints of the ET tube connection remain at or slightly below the level of the patient's body so that they will not interfere with or become dislodged during thoracoscopic instrumentation.
   (b) A bronchial blocker is not necessary as the remaining lung on the affected side is hypoplastic and will be further compressed with thoracoscopic insufflation.
3. Place a "jelly roll" to support the posterior aspect of the patient and a cushion between the patient's arms. Use other appropriate cushioning as needed.
   (a) Place the shoulder roll in a readily accessible fashion so that it can be removed and allow sterile repositioning of the patient to a supine position if conversion to subcostal laparotomy is necessary.

(b) Beanbags are often too large for patients of this size and are generally more cumbersome than helpful.

4. Position the arms in a neutral position with respect to abduction/adduction and reaching superiorly at approximately 120° to avoid interference with the ipsilateral port.
5. Tape the hips and ipsilateral shoulder to the bed to further support the patient's position.
6. Prep the left thorax and abdomen from the patient's spine to the anterior midline, superiorly beyond the tip of the scapula and inferiorly to the pelvis. This will allow sterile repositioning without re-prepping if conversion to laparotomy becomes necessary.
7. Drape the patient to expose the left hemithorax in a manner that allows visualization of important landmarks: the ipsilateral nipple, the spine, and the tip of the scapula.

(a) Place a removable sterile towel over the abdomen during thoracoscopy.

Trocar/Port Placement

1. Access the thorax using the Veress technique. Hold respirations temporarily and place the Veress needle just posteriorly to the tip of the scapula at approximately the fourth intercostal space.
2. Insufflate to a pressure of 3 mmHg at a flow of 1 L/min.

(a) Warn the anesthesia team to expect a temporary increase in the patient's end-tidal CO_2 that will usually resolve without intervention. Communication with the anesthesia team is essential at this point to ensure that the patient is tolerating the insufflation pressure.

3. If the patient tolerates initial insufflation, raise the pressure to 5 mmHg.
4. Replace the Veress needle with a 4-mm trocar and laparoscope.
5. Place two additional 3.5-mm ports approximately 3 cm on either side of the first port and one to two rib spaces below it (Fig. 12.2).

(a) Reduce instrument torque with the overlying rib by angling the trocars at a 45° angle or even tunneling a rib space caudad.

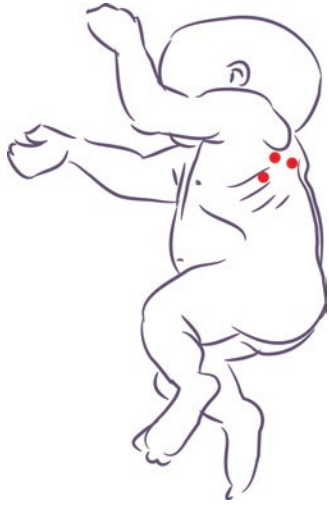

Fig. 12.2. Standard trocar placement for thoracoscopic repair of L CDH.

(b) Place the ports as cephalad as possible to facilitate this angling, enable easier reduction of the abdominal viscera, and allow room for thoracoscopic dissection and repair in an already restricted workspace.
(c) Take care not to place the ports too far medially and laterally as this causes collisions with the patient's arms and increases torque on the instruments when trying to operate in the superior aspect of the surgical field.

Reduction of Abdominal Viscera

1. Use an adjustable grasper and a bowel grasper to gently reduce the abdominal viscera from the chest in the following order: the small bowel, colon, stomach, and spleen (Fig. 12.3a).

   (a) The smaller dimensions of the adjustable grasper facilitate reduction and dissection in the restricted surgical field.
   (b) If necessary, divide diaphragmatic attachments to the colon and other viscera with the hook electrocautery to enable complete reduction of the viscera and unfurling of the diaphragmatic edge.

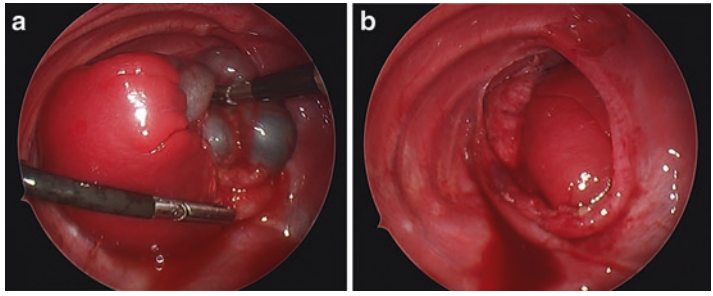

Fig. 12.3. (**a**) Abdominal contents are gently reduced into the abdominal cavity. (**b**) A splenic cap prevents migration of the abdominal contents back into the thoracic cavity during repair.

(c) Use two blunt/atraumatic graspers to reduce the viscera toward the anteromedial or right upper aspect of the field.
(d) Cover the viscera with the splenic cap to prevent the return of the viscera to the chest (Fig. 12.3b).

Diaphragm Repair

1. Place a Surgisis SIS underlay (Cook Medical, Bloomington, IN). It is our practice to do so whether or not a prosthetic patch is required, although at this time evidence is limited regarding its success in reducing recurrence rates. We do not cauterize the edge of the diaphragm peritoneum prior to repair, as there is no well-known evidence that this impacts outcomes.

   (a) Cut the SIS to size, leaving at least 1 cm excess in all directions, and roll it gently.
   (b) Remove one of the two lateral trocars and pass the contralateral grasper transthoracically through the vacant port site.
   (c) Use the grasper to drag the rolled mesh into the thorax (Fig. 12.4).
   (d) Unfurl the SIS on the abdominal side of the diaphragm.
   (e) Fasten the SIS to the diaphragm by including it in every second or third stitch of the diaphragmatic closure.

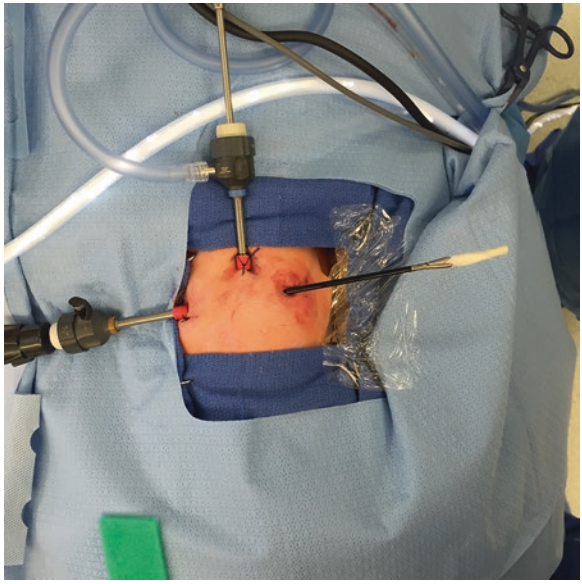

Fig. 12.4. SIS mesh is pulled into the thorax through a vacant port site.

2. Close the diaphragm defect by passing stitches every 8–10 mm. Pass stitches via trocar or transthoracically.
   (a) If placing via trocar, it is our practice to use a 2-0 silk on a ski needle, which can be passed through a 3.5-mm port. The lack of memory in the silk facilitates easier intracorporeal knot tying.
   (b) If placing stitches transthoracically, a standard RB needle may be used rather than a ski needle.
3. Begin primary repair of the diaphragm from the medial to lateral aspect to evaluate whether a patch is required.
   (a) We prefer to begin medially out of concern that although the lateral stitch may successfully approximate the edges of the diaphragm, it may cause undue tension on the repair that only increases upon approximation of the remainder of the diaphragm. By working toward the lateral side, we evaluate whether the lateral edges can be brought together without tension after the rest of the defect is approximated.

(b) Alternatively, the benefit of beginning laterally is that areas of redundancy in the medial diaphragm, if present, can be incorporated into the lateral stitch to re-approximate the lateral defect. This potentially alleviates the need for a prosthetic patch in a subset of patients.
(c) If necessary, ask the assistant to perform external compression of the chest wall when placing lateral stitches, as this is the most challenging part of the repair.
(d) If no patch is necessary, proceed to step 7.

4. If a patch is necessary, begin by placing a pericostal or "rib stitch" to approximate the lateral portion of the defect (Fig. 12.5a). It is easiest to place these first, as they will hang the patch in position while leaving adequate mobility to place the remainder of the stitches. The "rib stitch" may be placed using a variety of techniques:

(a) *Extracorporeal-assisted rib stitches*. Begin with a 2-mm nick in the skin overlying the anticipated rib (Fig. 12.5b). Place the stitch using one of two devices:

i. (*Preferred)* Place a Prolene or silk stitch transthoracically using a standard needle driver.

1. Grasp the needle inside the chest with the intrathoracic needle driver; pass it through the mesh patch and then to the outside of the chest on the other side of the rib (Fig. 12.5c).
2. When the needle has passed halfway through the chest wall, grasp the tip with the standard needle driver, paying careful attention not to remove it completely from the skin (Fig. 12.5d).
3. Use a backhand technique to pass the needle driver into the initial 2-mm nick, and tie the stitch (Fig. 12.5e).

ii. Pass a suture with the needle through the chest wall and the patch using the needle driver. Remove the needle. Place an 18-gauge angiocatheter through the nick on the other side of the rib. At this point, one of two options are possible:

1. Thread the free end retrograde out of the chest through the angiocatheter (Fig. 12.5f).
2. Pass a loop of a Prolene or nylon free tie through the angiocatheter and use this to lasso the free end of the stitch (Fig. 12.5g).

iii. Use the Carter-Thomason suture passer (CooperSurgical, Trumbull, CT) to transthoracically place and retrieve a free tie around the rib.

(b) *Completely intracorporeal rib stitches*. Ask the assistant to externally compress the chest wall, and use a sharply angled 18-gauge needle with silk or Prolene suture. This method can be extremely technically challenging with the greatest likelihood of intercostal bleeding.

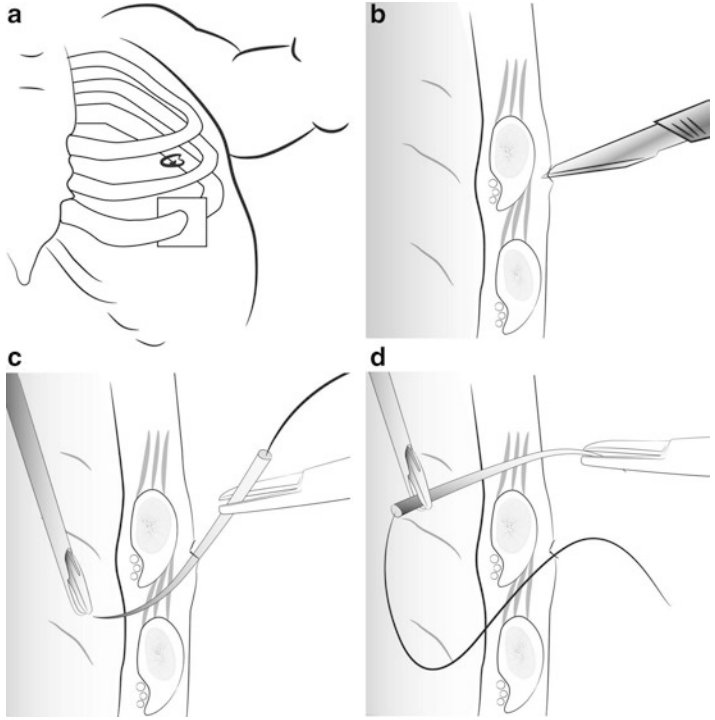

Fig. 12.5. Extracorporeal-assisted rib stitches. (**a**) Location of the rib stitch. (**b**) 2-mm nick overlying the anticipated rib. (**c**) A transthoracic rib stitch is performed by passing the needle from the outside of the body to the thoracic cavity, through the diaphragmatic edge, and (**d**) back outside the thoracic cavity, followed by (**e**) a backhand technique to pass the needle driver into the initial 2-mm nick. (**f**) Alternatively, the free end of the rib stitch is passed back out of the thoracic cavity through an angiocatheter. (**g**) A loop of Prolene or nylon is used to lasso the free end.

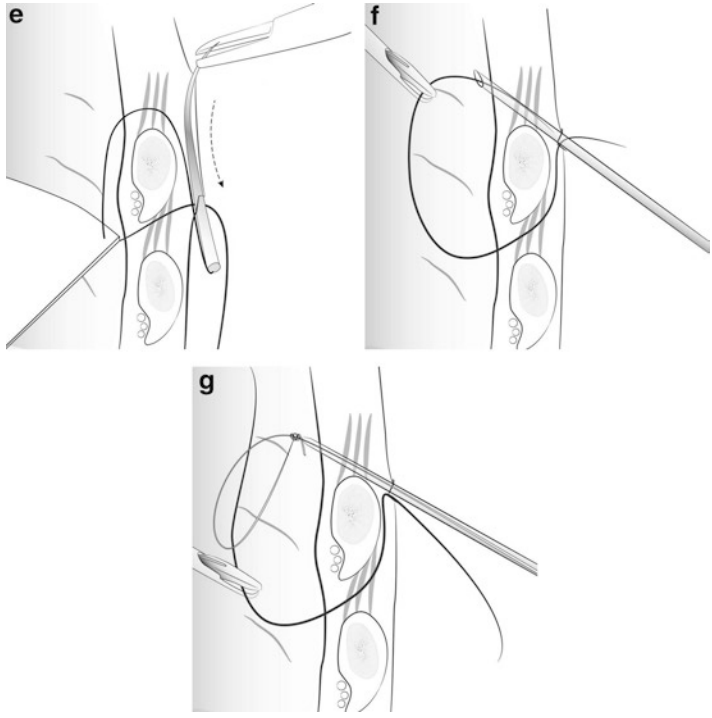

Fig. 12.5. (continued).

5. Prepare a Gore-Tex soft tissue patch (Gore, Newark, DE) and cut it to size.
   (a) Each dimension of the patch should be 5–10 mm longer than the defect in order to recreate the smooth curved contour of a natural diaphragm without undue tension.
   (b) If necessary, enlarge a port site by placing a small nick adjacent to the existing opening in order to accommodate the patch. Pass the patch into the thoracic cavity as described for the SIS mesh above:
      i. Remove one of the two lateral trocars and pass the contralateral grasper transthoracically through the vacant port site.
      ii. Use the grasper to drag the rolled patch into the thorax.

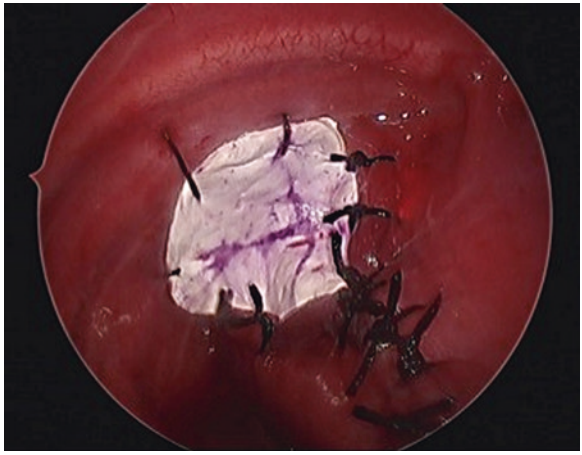

Fig. 12.6. A patch is used to cover the lateral aspect of the diaphragmatic defect.

6. Sew the patch into the remaining diaphragmatic defect (Fig. 12.6).
   (a) It is our practice to sew the patch in an interrupted fashion with approximately 8–10-mm distance in between stitches.
   (b) If a continuous repair is chosen, use multiple continuous segments so that a single break will not unravel the entire repair. Leave a long tail on the individual knots, and sew the tail of one segment to the running stitch on the connecting segment, in order to avoid introducing a loop into the knot.
7. Perform appropriate closure. The necessity of a chest tube for these operations is still a point of debate. If a chest tube is not deemed necessary, remove all trocars, evacuate the CO_2 in the chest, and close the skin.
   (a) If a port site was enlarged to introduce a patch, re-approximate the fascial layer with a single absorbable stitch.
   (b) Seal the trocar sites with Dermabond (Ethicon, Somerville, NJ).

Pearls

- Placement of ports high in the chest and angling inferiorly or tunneling provides the largest operative field while maintaining optimal ergonomics.
- Thoracoscopic insufflation pressures should be in the range of 4–5 mmHg and may often be reduced to 0 after the viscera has been reduced into the abdomen.
- It is often necessary to unfurl the rim of diaphragm and separate it from the abdominal viscera in order to appreciate the actual size of the defect as well as the usage diaphragmatic tissue.
- The patch should be sized to the defect with an additional 5–10 mm in each dimension to create a smooth, curved contour.

Pitfalls

- An initial rise in end-tidal CO_2 is expected after insufflation and may prompt conversion to an open procedure. Give the infant time to acclimate before aborting the thoracoscopic approach.
- Even a small amount of splenic bleeding can greatly obscure the operative field. The spleen should be handled extremely gently.
- The esophagus and aorta are in close vicinity to the repair site and at risk of injury. Pay attention to the insertion of the esophagus and the course of the aorta when suturing the posterior aspect of the defect.

Postoperative Care

Outcomes

Postnatal survival has improved significantly in recent years with the advent of ECMO and advanced neonatal care. Overall survival rates at tertiary centers with these capabilities are reported between 70 and 92 % with better outcomes in infants with isolated CDH [3, 16].

Several factors have been shown to significantly impact CDH survival. The most validated prenatal prognostic measurement is the estimation of the degree of pulmonary hypertension using observed to expected LHR, with estimated 0 % survival if the value is <15 % and >75 % survival for a ratio >45 % [2, 17–20]. Postnatally, average survival is lower in premature infants (53.5 %), and rates decrease with decreasing gestational age [16]. Associated anomalies are a poor

prognostic factor; infants with major cardiac defects in particular have a reported 36 % survival rate [3]. In patients with liver herniation, survival was 45 % vs. 93 % in a matching cohort without liver herniation [19]. Survival rates in the ECMO subset range between 50 and 86 % [14, 16, 21, 22].

Complications

Surgical complications include recurrence, tension pneumothorax, intestinal adhesion obstruction, and musculoskeletal deformity.

The primary concern after CDH repair is the risk of hernia recurrence. Recurrence rates have been reported between 10.8 and 41 % overall with a bimodal incidence between 1 and 3 months and between 10 and 36 months after initial repair [23–27]. Large defects requiring synthetic patches and presence of the stomach or liver in the thoracic cavity increase risk of recurrence [23, 24, 28]. Recurrence rates were historically lower after open repair than after thoracoscopy, although improvement in technique has resulted in more similar outcomes in recent years [28, 29]. Recurrences may be repaired laparoscopically or thoracoscopically. It is our practice to repair most of them through the abdomen to achieve better visualization for dissection of the abdominal viscera and safe repair in the setting of thoracic adhesions. When necessary, conversion to an open procedure is performed via a subcostal laparotomy.

Obligate postoperative pneumothorax occurs in nearly all infants after CDH repair. Clinically significant pneumothorax, however, is estimated to occur in up to 30 % of patients, and the percentage of patients requiring intervention is as high as 16.4 % [30–32]. Chylothorax has been reported in 4.6 % of patients postoperatively with higher risk after ECMO use or patch repair [33].

Musculoskeletal deformity is theorized to result from tension of the patch on the growing chest wall, with higher risk in patients with large defects or postoperative empyema [28, 34, 35]. Chest wall deformities, the majority of them pectus excavatum and most of them mild, occur in up to 48 % of patients [28, 35]. Scoliosis is reported in up to 27 % of surviving CDH patients [26, 28, 34].

Intestinal adhesion obstruction after CDH repair is as high as 20 %, a significantly higher rate than in the general pediatric population undergoing laparotomy (2.2 %) [23, 26, 27, 35]. In our experience, the rate of postoperative obstruction is significantly reduced following thoracoscopic repair as compared to open laparotomy.

Summary

- The majority of CDH are the Bochdalek subtype, and left-sided defects are significantly more common than right sided.
- The most common cause of morbidity and mortality in CDH patients is pulmonary hypoplasia and pulmonary hypertension.
- Many cases of CDH are diagnosed prenatally. Postnatally, CDH is suspected by characteristic chest X-ray in the setting of respiratory distress.
- Surgical correction is indicated in all patients with CDH. Surgery is generally delayed, pending stabilization of respiratory status as observed by physiologic criteria.
- The advent of ECMO and improved neonatal care has significantly improved the prognosis of infants with CDH.
- The most common and concerning complication of CDH is recurrence, occurring in the first 3 months of life or between 1 and 3 years in up to 41 % of patients.

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