EMBRIOLOGICAL AND ANATOMICAL BASES IN SURGERY OF CONGENITAL STRUCTURAL DEFECTS OF THE DIAPHRAGM

(Review of literature)

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INTRODUCTION

Congenital diaphragmatic defects are one of the leading causes of morbidity and mortality in neonates. Often the defect dimensions, precise anatomical details or defect type directly influence the prognosis of these malformations [1]. For a surgeon, knowing the aspect of embryology and surgical anatomy of the diaphragm is critical in understanding the mechanisms of development of normal variations and congenital malformations that are rarely encountered in surgical practice [31].

EMBRYOLOGY

The diaphragm is a critical organ in maintaining normal breathing, and is also a barrier between thoracic and abdominal cavities [9, 10, 28]. Being closely linked to the formation of body cavities, the structural elements of the diaphragm begin to develop in early gestation [9], in the 4th to 12th week of intrauterine development [27,37]. The diaphragm is derived from several embryonic sources, some of them being unidentified [35]. At present, the diaphragm is thought to be derived from four mesodermal structures [8], including: *transverse septum*, two lateral pleuroperitoneal envelopes, dorsal mesentery of the esophagus, and the body wall muscles [4, 11].

The anterior region of the diaphragm originates in the transverse septum, and later this tissue develops into the mature central tendon and the non-muscular mesenchyme [32]. The transverse septum is the first structure in development of the diaphragm and initially serves as a barrier between the thoracic and abdominal cavities [35], and as a coherent mesodermal structure is formed at the end of the third week of gestation. In the fourth week of gestation, the incomplete separation of pericardial and peritoneal cavities takes place, with the formation of two adjacent esophageal apertures (pericardioperitoneal canal). Originally located at the occipital level, superior to cervical somites (C3), this embryonic structure represents a frontal caudal dorsal inclination, initially connected to the body wall, and the ends at the cranial edges of the middle intestine. During the descending migration, the transverse septum passes past the cervical segments III, IV and V, a process accompanied by the migration of myogenic stem cells from these somites and the local differentiation of myoblasts into the tissue of the pleuropericardial folds, referred to as the posthepatic mesenchymal plaque. The transverse septum does not extend all the way to the dorsal wall, the pleural and peritoneal cavities remain open, being called pleuroperitoneal ducts (canals) (20, 41).

In the 5th week of gestation, neural structures in the cervical IV and V segments of the spinal cord develop. Nerve fibers penetrate into the transverse septum through the pleuropericardial folds, where they form the phrenic nerve. The detachment of these folds from the somatopleure to form the pericardium, determines the localization of the phrenic nerve between the pericardium and the mediastinal pleura [11, 41].

Pleuroperitoneal folds are two transient structures formed by the fusion of pleuropericardial folds [9], located on either side of the esophagus, attached to the dorsolateral wall of the body, with the free edge protruding at the caudal end of the pericardioperitoneal canals [35], with the transverse septum. In the 6th - 8th week of gestation, growing medially and ventrally, these folds merge with the dorsal mesentery of the esophagus and a portion of the transverse septum, causing separation of the pleural and peritoneal cavities. Closure of the communications is also determined by the extensive increase of the liver tissue and muscle structures in these membranes, from which only a small dorsolateral portion of the diaphragm will develop [4, 27, 41].

The dorsal mesentery of the esophagus contributes to the attachment of the middle intestine to the trunk wall, eventually forming the median portion of the diaphragm. The crura of the diaphragm develop from muscle fibers growing in the esophageal mesentery [4].

The contribution of the muscle wall of the trunk to the formation of the lateral diaphragm portion is determined by the enlargement of pleural cavities during weeks 9-12 of gestation. As a result, the body wall is divided into two layers: external, from which the thoracic wall will later develop, and internal, embedded in the diaphragm [41].

Several hypotheses attempt to explain the origin of congenital diaphragmatic defects. According to some, congenital diaphragmatic hernias are a consequence of the vicious development of the lung or the developmental disturbances of the phrenic nerve

[21]. Other scientists believe that the origin of the congenital diaphragmatic hernias is in the non-muscular portions of the fetal diaphragm and results from the failure of fusion of one or several of these embryonic structures [8, 11, 13, 45]. In cases where the membranes merge and the muscle fibers fail to migrate from the cervical myotomes, a hernia with sac appears [11]. The development of these defects is based on some disorders of the retinoid signaling embryogenic mechanisms and of some transcription factors [2, 16, 34, 43]. For example, Bochdalek hernia (the abnormal development of the pleuroperitoneal fold) results in the typical posterolateral defect [9], and the fusion defect of the transverse septum with the lateral wall of the trunk leads to the anterior diaphragmatic hernia (Morgagni hernia) [46].

CLASSIFICATION

Classification of diaphragmatic hernias proposed by Harrington S.W. in the 1930s. proved to be quite widespread. The author distinguished two main types of diaphragmatic hernias: traumatic and non-traumatic [14, 44], including:

I. Non-traumatic hernias:

A. Congenital hernias (usually without sac):
1. through the pleuroperitoneal hiatus (Bochdalek foramen);
2. through a space left by the partial absence of the diaphragm (posterior part);
3. through the esophageal hiatus (as a result of muscle deficiency);
4. through the esophageal hiatus in the case of a short congenital esophagus (the stomach remains partially or entirely above the diaphragm);
5. through the anterior subcostosternal spaces (foramen of Morgagni) with the presence of the hernial sac.

B. Hernias acquired after birth:
1. through the esophageal hiatus (it usually has a hernial sac);
2. through the fusion region of the diaphragmatic primordium;
3. other locations.

II. Traumatic hernias:

A. Direct injuries - in any part where an object can penetrate into the diaphragm;
B. Indirect injuries - at any point, but usually at the embryological fusion points;

C. Diaphragm rupture following inflammatory necrosis (subphrenic abscess, necrosis due to drainage tube pressure).

Jackson R.L., and Welcome H.C. (1941) [23] classified the diaphragmatic hernias into 3 groups:
1. Congenital (true and false);
2. Acquired (true and false);
3. Traumatic (true and false).

In 1952, Gudbjerg C.E. [18] argued that the lack of a clear classification of diaphragmatic abnormalities was determined by the uncertainty regarding their pathogenesis and proposed a new classification, reuniting diaphragmatic abnormalities into three groups:
1. *Diaphragmatic hernias:*
   a) congenital (true with sac and false without sac);
   b) traumatic
   2. *Relaxation of the diaphragm:*
   a) congenital (defective development of the musculature);
   b) paralytic (resection of the phrenic nerve, tumors).
   3. *Absence of the diaphragm.*

The classification of diaphragmatic hernias proposed by Petrovskii B.V. (1966), quite useful in practical activity, included the following forms: 1) congenital - diaphragmopleural, hiatal, parasternal, phrenopericardial hernias and 2) acquired, divided into traumatic and non-traumatic[5]. In 1988, Isakov Iu.F. proposed the classification of congenital diaphragmatic hernias, namely : 1) embryonic hernias (false); 2) fetal hernias (true hernias) and 3) relaxation of the diaphragm [5].

In 2007, Trojan V.V. [5] proposed an anatomical classification of the congenital diaphragmatic hernias:
1. Hernias of the posterior region of the diaphragm.
2. Hernias of the diaphragmatic vault.
3. Hernias of the anterior region of the diaphragm:
   a) Retrosternal hernias (true);
   b) Phrenicopericardial hernias.
4. Hiatal hernias:
   a) Oesophageal hernias;
   b) Para-oesophageal hernias.

Based on some intraoperative observations, some authors have tried to classify the congenital diaphragmatic defects, according to their dimensions, into: A) defects surrounded by muscles; B) small defects (less than 50%); C) major defects (over 50%) and D) absence of the hemidiaphragm [36].

It is believed that most congenital diaphragmatic defects develop in "classical" locations such as posterolateral (Bochdalek), parasternal (Morgagni-Larrey hernia) and central and anterior hernia (Cantrell transverse septum hernia) [1], several concomitant diaphragmatic defects being observed in some cases [6, 25].

The congenital diaphragmatic defects originating from the lumbocostal triangle are the most prevalent and were first described in 1848 by Vincent Alexander Bochdalek, who reported an incidence ranging from 5% to 25% of the total diaphragmatic hernias [1]. Although Bochdalek hernia manifests in neonatal period by severe progressive respiratory distress [54], cases of late-onset manifestations were

reported in adults [19, 33]. More often (up to 90% of cases) the malformation is found on the left, rarely the bilateral form being detected [26].

In 1959, Bingham described an anatomical entity characterized by the absence of the hemidiaphragm (diaphragmatic agenesia), distinct from the postero-lateral defects [49]. Cases of bilateral absence of the diaphragm are also described [48].

The congenital Morgagni hernia is relatively rare, constituting approximately 3-5% of all types of congenital diaphragmatic hernias. Approximately 90% of this type of hernias appear on the right, 2% on the left, and in 8% of the cases bilateral forms are found. The hernial sac is present in over 95% of cases [15]. Although congenital Morgagni hernia is specific to children, asymptomatic forms were primarily diagnosed in adults and even in elderly patients [30, 50].

The congenital intrapericardial diaphragmatic hernia is characterized by herniation of abdominal viscera in the pericardial cavity, being caused by developmental anomalies of the retrosternal transverse septum with persistence of the pericardioperitoneal canal. This entity is also referred to as peritoneoperi-cardial diaphragmatic hernia, which may be associated with other congenital defects at the midline and pentalogy of Cantrell [24, 38]. The first case of peritoneopericardial diaphragmatic hernia was described in 1903 by De Cardinal and Bourderou [42].

The central tendon defects are casuistically diagnosed, characterized by congenital hernia of the abdominal content through the central tendon of the diaphragm. The embryogenic aspects of these defects remain unclear [9, 29].

Diaphragmatic eventrations, a nosological entity described in the literature as diaphragm atrophy, non-paralytic eventration, neurogenic aplasia of the diaphragm, etc., are congenital defects of rare development of the muscle portion of the diaphragm. From a histological point of view, it is characterized by fibro-elastic changes, which replace the muscular tissue layer and extend between the pleura and the peritoneum, keeping the diaphragm anatomical structure intact, thereby differentiating from the congenital diaphragmatic hernias and diaphragmatic paralysis. The incidence of this malformation is below 0.05% [53]. The left hemidiaphragm is more frequently affected. Congenital diaphragmatic eventrations are divided into: complete, partial and bilateral. Partial eventrations, based on the embryological segments that contribute to the formation of the normal diaphragm, can be classified into the following types: anterior, posterolateral and medial [17, 52]. Multiple eventrations of a single hemidiaphragm are also reported [51].

In 1774, Jean Louis Petit was the first to observe this entity during the necropsy, the term *eventration* was later proposed by Beclard (1829) [47]. In 1923, Morrison published the first case of diaphragmatic eventration subjected to surgical treatment [22, 53].

ANATOMY

The diaphragm is a dome-shaped musculofibrous septum that separates the thoracic cavity from the abdominal cavity, maintaining the pressure gradient between the two cavities and participating in the regulation of esophageal contractions, being the most important respiratory muscle. The name of the organ comes from the words of Greek origin *dia* (between) and *phragma* (fence) [12, 31, 39, 40]. The main element of the diaphragm is the phrenic muscle, covered by a homonymous fascia (upper and lower), parietal pleura (upper), peritoneum (on the abdominal side) and traversed by vessels, nerves and esophagus. The diaphragm vault consists of two domes: right one, a slightly higher one, the top of which corresponds to the intercostal IV space on the right medioclavicular line and the left one, with the highest point on the V rib level along the left medioclavicular line [7].

The diaphragm consists of a central non-contracting tendon, considered central aponeurosis, two major muscular portions (costal and crural diaphragm) and a minor sternal muscle portion [4, 41].

The lumbar (crural) side of the diaphragm (*pars lumbalis*) is represented by the pillars of the diaphragm (right and left) - the strongest part of the diaphragm [7]. The right diaphragm pillar is located on the anterior surface of the lumbar vertebrae (L_{1-4} on the right and L_{1-2} on the left), on the intervertebral discs and the anterior longitudinal ligament. The right diaphragm pillar, larger than the left, is directed towards the middle of the central tendon, on both sides of the upper median plane, forming by branching, in more than 60% of individuals, the esophageal hiatus, in the rest of the cases, both pillars of the diaphragm participate in its formation. Split muscle fibers meet again to form the anterior edge of the aorta hiatus. Although the esophageal pillar is made up of muscular and tendinous tissue, only the tendinous side is strong enough to deal with sutures during surgery. The left diaphragm pillar is facing up, to the left of the esophageal hiatus. A portion of this pillar reaches the central tendon [4].

There are also described accessory pillars (intermediate), placed laterally by the main pillars, represented by muscular bundles originated on the vertebral body L2, which, with the lateral pillars, delimit a narrow space for the splanchnic nerves [7].

The lateral pillars (or the arches bundles) start from the medial arcuate ligament, (*lig. arcuatum mediale*), passing as a bridge over the psoas muscle, coming onto the body of the first lumbar vertebra and the intervertebral disc L1-L2, and laterally at the top of the transversal apophysis L1, sometimes L2, and the lateral arcuate ligament (*lig. arcuatum laterale*) that passes over the quadratus lumborum muscle, joining the tip of the transverse apophysis L1 and/or L2 with the lower edge of the free end of the 12th rib. Through the space delimited by the

lateral and medial pillars, the sympathetic chain and the ascending lumbar veins pass, which continue with the azygos veins (on the right) and hemiazygos veins (on the left). The median arcuate ligament (*lig. arcuatum medianum*) contributes to preaortic solidarising of the two diaphragmatic pillars, consisting of aponeurotico-tendinous fibers [7].

The costal part of the diaphragm (*pars costalis*) originates from the inner surface and the upper edges of the six lower (caudal) ribs and radiates in the central tendon [11]. At the limit of the costal and lumbar portions, the muscle fibers are missing, giving rise to a triangular space with its base at the 12th rib (lumbocostal triangle, Bochdalek). This area, covered only by the diaphragm fasciae, parietal pleura (upper) and the retrorenal fascia of the parietal (lower) peritoneum, makes it possible to spread infections from the thoracic cavity (with subpleural adipose connective tissue) in the retroperitoneal space (with pararenal adipose tissue, Gerota) in double directions [7, 11].

The sternal part (*pars sternalis*) of the diaphragm is the smallest and extends from the posterior surface of the right muscle and behind the xiphoid process to the central tendon of the diaphragm. Laterally to the sternal portion of the diaphragm, the coastal parts are. Between these portions, the diaphragm is covered only by connective tissue, the sternocostal triangle or foramina of Morgagni is on the right, and sternocostal triangle or Larrey`s triangle is on the left [11].

The central tendon is an aponeurotic form, which has the shape of a clover (trefoil) leaf and consists of three leaflets (one anterior and two lateral) separated by slight crevices. Between the anterior and the lateral right leaflet, moved posteriorly, there is the inferior vena cava which is inextensible and ovoid, being crossed by the homonymous vein and bordered by tendinous fibers, some of them being circularly arranged, others radially oriented and ascending towards the vein adventitia, solidarising with the orifice border. Thus, the circulatory onset of the lower vena cava is not influenced by the diaphragm movements during breathing [4, 7].

The vascularization of the diaphragm occurs in the branches of the pericardophrenic, musculophrenic, upper and lower diaphragmatic arteries, which form a rich anastomotic network with the VI-IX intercostal arteries, the subcostal arteries and the branches of the internal thoracic artery. Two veins, accompany the homonymous arteries and flow into the azygos veins, internal thoracic veins and inferior vena cava, at this level being made cavocaval and portacaval anastomoses [7].

The diaphragm innervation is ensured by the phrenic nerves that originate in the cervical plexus (C3-C4), to which they reach around the inferior vena cava orifice (right) and lateral and anterior (left), respectively, dividing, consecutively, in an anterior and a posterior branch. Some of the posterior branches continue with the phrenico-abdominal ones that penetrate the diaphragm, being distributed in the subdiaphragmatic parietal and visceral peritoneum with the ligaments of the supramesocolic organs, especially the liver. At the diaphragm motor innervation, intercostal IX-XI nerves participate, oriented especially to the periphery of the coastal portion. Pain, which radiates to the right shoulder and the supraclavicular region in hepatobiliary disorders, and those radiating to the left shoulder, in pancreatic and spleen diseases (Eiselsberg sign), are transmitted via the phrenico-abdominal branches and the trunk of the phrenic nerve [7].

Therefore, the various congenital diaphragmatic defects require a thorough evaluation, with rigorous documentation of anatomical forms in order to elucidate the biology of the development of these malformations and the individual adaptation of the surgical reconstruction.

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