Clinical-evolutive, diagnostic and treatmental particularities in traumatic diaphragmatic hernia in children. Clinical case presentation and literature review

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Diaphragmatic-traumatic hernia represents an entity in the child. Frequency reduced, delayed clinical manifestations, nonspecific symptomatology determines multiple diagnostic difficulties, which increases the risk of developing severe complications.

The authors present the case of a 4-year-old child with late manifestations of traumatic diaphragmatic hernia. The certainty diagnosis was established at thoracic CT with dynamic contrast in angiographic regime. The child underwent surgical treatment.

The authors concluding that the primary repair with the application of nonabsorbable sutures and the concomitant use of the acellular pericardial mold for consolidation is an effective option in the surgical reconstruction of traumatic diaphragmatic defects in children.

The authors declare no conflicts of interests.

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Introduction

Traumatic diaphragmatic hernia (HDT) is a rare disease in children. The incidence of HDT ranging from 0.08% to 8% is reported in the literature, with mortality rates ranging from 16.6% to 33.3% [3]. The reduced frequency, the non-specificity of the symptoms, the clinical evolution with associated lesions causes multiple diagnostic difficulties in 50–70% of cases, a high index of suspicion being necessary [8]. Late manifestation of traumatic lesions of the diaphragm is studied more in adults than in children [4]. In this context we present the following clinical case.

Case report

Patient S., 4 years old, was transferred on 24.01.2020 to our institution from a district hospital where she was hospitalized on 19.01.2020, being suspected clinically-radiologically a paraesophageal hiatal hernia, but was not excluded and a destructive pulmonary process with pulmonary abscess formation (fig. 1).

At the time of hospitalization, the patient had unexplained violent abdominal pain in the epigastric region and in the left hypochondria, fever (38.5 °C), constipation, the symptomatology being present for 4 days.

From anamnesis: a month ago the child was treated in the hospital for a respiratory infection. Upon discharge, the therapeutic effect was confirmed radiologically, intrathoracic formations were not detected. Two weeks before the onset of the new symptoms, the baby fell, hitting the chair, the accident being overlooked by the mother. On 19.01.2020, during the game, the child became suddenly agitated, the listed symptoms appeared, which over the years worsened significantly.

The clinical examination revealed the serious general condition, conditioned by the presence of signs of exsicosis, stable hemodynamics. Pale skin, cyanosis of the nasal lip triangle, tachypnea and tachycardia. Auscultative, on the left side, breathing did not occur, except for the apical area. Palpation – painful abdomen all over the surface, predominantly in the epigastric region and in the left hypochondriac region. Laboratory examination revealed anemia (erythrocytes – 3.5×10^6/ul, Hb – 105 g/l and neutrophil leukocytosis 10.9×10^9/l).

The thoracic and abdominal radiography, performed by emergency in the clinic, showed the trans diaphragmatic positioning of the intestinal handles in the left hemi thorax, the diaphragmatic hernia having large dimensions than before (fig. 2).

The diagnostic of certainty was established with the help of thoracic CT with dynamic contrast in angiographic regime, which confirmed the subtotal prolapse of the large
Клінічний випадок

Опис

ранічевого пациента 5 років. Попередня рентгеногістологічна картина
відображувала відтік кишечного контенту в легкій кількості.

Посічено, що діафрагма була розширення

і обличчена слизистою.

After a 2-hour preoperative preparation, surgery was
performed, performing a left subcostal laparotomy.

Intraoperatively, a defect of the left hemidiaphragm was
detected (Fig. 4) at the fusion level of the anterior part of
the tendon and the muscular part. Through this defect
the intrathoracic hernia the omentum, colon and small
intestine handles. A portion of the omentum adheres
intimately to the edges of the diaphragmatic defect. After
the reduction of the eviscerated organs in the pleural
cavity it was found that the segment of herniated omen-
tum had severe and trophic inflammatory changes,
which was resected. Cyanotic herniated bowels, on the
surface multiple petechiae. The mesoz with significant
changes of vascular stasis and petechial foci. After de-
bridement of the defect area, the integrity of the
diaphragm was restored with non-absorbable interrupt-
ed sutures. For the purpose of consolidation, a biodegra-
dable acellular biological graft fragment of equine peri-
cardium (Bioteck Heart) was applied. The pleural cavity
was drained by microtoracotomy with tubular drainage,
and the abdominal cavity with a glove blade drainage.
The operation was completed with the restoration of the
anatomical structure of the anterior abdominal wall.

Histologically, in the tissues taken from the margins
of the diaphragmatic defect, a lympho-plasmocytic in-
flammatory process with macrophage elements, more
frequent in the vicinity of the cellulose adipose tissue
islets, and the adhesive fibrillary cellulose tissues (with
predominantly non-connective tissue, predominating in
the adipose tissue) was revealed (Fig. 5A). In some areas
of the tissue plastic area, outbreaks of young unformed
connective tissue were attested which, at times, mimic
the mature granulation tissue, with the accentuation of
proliferative fibrovascular processes. The density of un-
formed fibrillar tissue was variable, in some areas with

and small intestine handles by a defect of 2 cm diameter
centrally located at the level of the left hem diaphragm. The
total atelectasis of the lower lobe of the left lung was deter-
dined with fluid accumulation in the left pleural cavity,
stomach and spleen located orthotopically (fig. 3).

Fig. 3. Patient S, 4 years. Preoperative thoracic and abdominal CT. Subtotal prolapse of the small and large intestine handles
in the cavity of the left hemithorax is visualized by a defect of the left hemidiaphragm with total atelectasis of the lower lobe
of the left lung. Pleural effusion

A

B

C

D

E

F
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lished case (in 1579) of diaphragmatic rupture, diagnosed through the diaphragm following trauma was mentioned at autopsy, to Ambroise Pare. Herniation of the stomach

discases indicates at lesions of tissue contusion and dis-

ordered fibrillar appearance (Fig. 5B). In part, the pro-

cess was circumscribed by adhesive fibroblastic proces-

s. In the non-formed adhesions, marked by mature granulations, there was an increase of the proliferative processes of vascular and fibroblastic, compared with the fibrillar component, frequently associated with the presence of lymphocytes and granulocytes from discrete to moderate. In some areas, excessive vascularization and fibroblastic proliferative-cellular processes with reduced conjunctival fibrillar component have been observed, which mimic angiomatous processes. The lack of hemosiderosis indicates at lesions of tissue contusion and dis-

junction of the hemidiaphragmous resident tissue.

Under the protection of a conservative antibacterial treatment and hydroelectrolyte rebalancing, the patient was recovered without complications, being discharged at the 10th postoperative day in a good general condition. Radiologically, on the 9th day after the intervention, a pulmonary drawing without pneumonic opacities was visualized, the contour of the left hemidiaphragm being well highlighted (fig. 6).

Discussions

The first description of the traumatic lesions of the diaphragm belongs to Sennertus (1541), and the first described case (in 1579) of diaphragmatic rupture, diagnosed at autopsy, to Ambroise Pare. Herniation of the stomach through the diaphragm following trauma was mentioned by Ambroise Pare in 1580. In 1853, H.I. Bowditch published the first case of HDT diagnosed with death, establishing five diagnostic criteria. In 1886, Riolfi performed the first successful intervention of a penetrating diaphragm injury, and Walker, in 1899, successfully reduced a diaphragmatic hernia and repaired the diaphragm [7].

Traumatic lesions of the diaphragm are caused by thoraco-abdominal traumatic injuries (75%) or pen-

trating (25%), the rate of severe injuries being 44–94%. In children, HDTs are more commonly located (90%) on the left side [9]. HDTs are considered to be the consequence of the sudden increase of the pleuropertoneal pressure gradient and occur in the weak areas along the
embryonic fusion points. Migration of intraperitoneal organs into the pleural space, with compression of the lung and displacement of mediastinum, compromises cardiovascular and respiratory functions. Thus, dysfunction of a hemidiaphragm leads to a reduction of lung function by 25–50% [7].

Three evolutionary phases of the rupture of the diaphragm are described: 1) the acute phase – at the time of the traumatic injury of the diaphragm; 2) phase two – transient herniation of the viscerum; 3) third phase – development of complications such as strangulation and the appearance of symptoms of intestinal obstruction, pulmonary compression, mortality rate increasing up to 20–80% [4].

Clinical symptoms in HDT are dominated by respiratory distress (86%), abdominal pain (17%), shortness of breath on the affected side (73%), some patients experiencing chest pain, vomiting.

It is considered that only 25–50% of traumatic lesions of the diaphragm are diagnosed by initial chest radiography [4]. According to reports, this method allows the detection of pathognomonic or suggestive signs in 70–85% of cases, while CT of the chest and abdomen confirms the diagnosis, with a sensitivity of up to 82% and a specificity of 87% [8]. Radiological signs suggestive of HDT include: elevated hemidiaphragm, distorted and obscured diaphragmatic margins, presence of intestinal shadows or air levels above the diaphragm, pleural collection, pulmonary collapse, and contralateral displacement of mediastinum [1].

HDT treatment is surgical, by abdominal or thoracic approach, and involves the replacement of the herniated organs under the diaphragm, drainage of the pleural cavity and repair of the diaphragmatic defect. Surgical repair is necessary even for minor defects, which will not heal spontaneously, and the thoraco-abdominal pressure gradient favors the enlargement of the defect with herniation of the abdominal organs [3]. Reconstruction of the diaphragmatic defect can be performed with nonabsorbable sutures interrupted in one or two layers [6]. In some cases, the use of synthetic or biological meshes can be used in the reconstruction of post-traumatic diaphragmatic defects [2].

Some authors consider that laparoscopy is a safe procedure in resolving cases of traumatic diaphragmatic hernias in patients with stable hemodynamics [5]. In the treatment of HDT, it is also possible to use video-assisted thoracoscopic surgery for both diagnostic and treatment purposes, careful selection of patients being essential for the success of this method [10].

Thus, in young children HDT is a major challenge that can occur as a result of traumas, «insignificant» at first sight, overlooked by parents. These can evolve quietly, the clinical symptomatology developing suddenly after certain periods of time, which requires the collection of a thorough anamnestic history. Preoperative diagnosis of HDT in young children is quite difficult, thoracic CT with dynamic contrast in angiographic regime being an effective method in establishing the diagnosis with certainty. The results of the histological examination indicate that the mechanism development of diaphragmatic defect in children may occur as a result of tissue contusion injury and subsequent disruption of the resident hemi diaphragmatic tissue. In the surgical reconstruction of traumatic diaphragmatic defects. The authors declare no conflicts of interests.

References