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Case Report

Laparoscopic repair of eventration of the left dome of the diaphragm: case report with review of literature

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ABSTRACT

Diaphragmatic eventration (DE) is characterized by an abnormal elevation of one or both hemi diaphragms without any actual disruption or discontinuity in the diaphragmatic musculature. This condition results in a structurally intact but functionally impaired diaphragm. The etiology may be either congenital or acquired, thereby allowing for its occurrence across a wide age spectrum-from neonates to elderly individuals. Its diagnosis is usually made and confirmed on imaging. Symptomatic patients of DE require surgery for its repair (open/ laparoscopic/ thoracoscopic/robotic). The authors, herein, present the case of a 51 years old male patient who had symptomatic left sided DE. He underwent a laparoscopic repair and had an uneventful recovery.

Keywords: Acquired, Congenital, Diaphragmatic, Elevation, Eventration, Laparoscopic

INTRODUCTION

Diaphragmatic eventration (DE) can be unilateral or bilateral, with unilateral cases being more commonly encountered. Left-sided eventration is notably more frequent than right-sided involvement.[1] This asymmetry is likely due to the earlier closure of the right pleuroperitoneal canal during embryogenesis and the mechanical support provided by the liver to the right hemidiaphragm.[1] In fact, approximately 88% to 97% of cases involving congenital hemidiaphragmatic agenesis occur on the left side.[1] Epidemiologically, DE in adults has a mean age of presentation around 41.86 years, with a higher incidence observed in males.[1]

CASE REPORT

A 51 years old male patient presented to the surgical out patients department (OPD) with complaint of dyspnoea on exertion and in lying down position since 2 months. The symptom had worsened over the said period with initial dyspnoea only on climbing stairs; but of late, also experienced on walking on flat ground. Also, he gave a typical history of feeling breathless in reclining position, which got better on sitting up. He also complained of dull pain in his left lower chest off and on since the last 15 days. On examination, his pulse was 88 per minute, blood pressure was 140/90 mms of mercury and respiratory rate was 12 per minute. A per abdomen exam was normal. On auscultation of lungs, air entry seemed to be diminished in the left lower zone. A plain X-ray chest postero-anterior view was then done and it revealed an eventration of the left dome of his diaphragm (Figure 1A). A contrast enhanced computed tomography (CECT) scan done thereafter, confirmed the same (Figure 1B). The patient was then counselled to undergo a laparoscopic repair, in view of his symptomatology. After due workup and assessment of fitness, he was taken up for a laparoscopic repair. The patient was in a supine, leg straight and split positionA 1-0 V-loc® was used to plicate the left dome of diaphragm in a continuous fashion (Figure 1 C and D, 2 A-D, 3 A and B). After this was completed, a Titanium mesh was placed optimally in situ, to fortify the suture line; and was suture fixed to the diaphragm Figure 3 C and D). The patient had an uneventful postoperative recovery. He was discharged

from the hospital on postoperative day (POD) 3. On his POD 10 out patients department follow up visit, all his operative wounds had healed well.

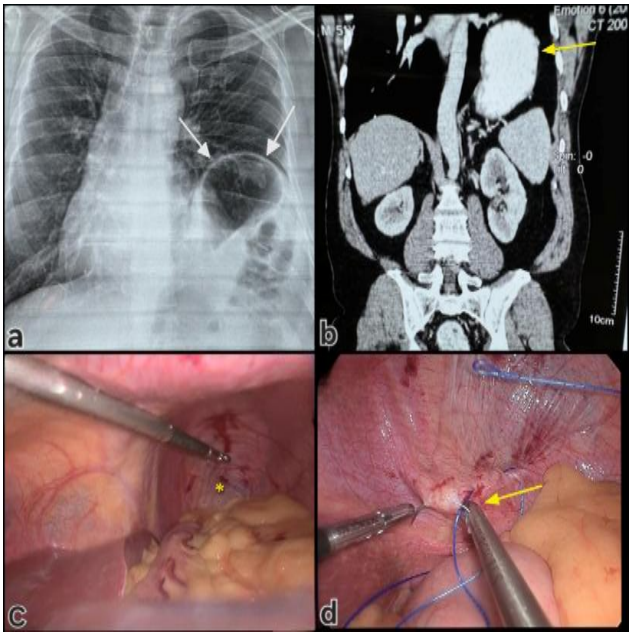

Figure 1 (A-D): (A) Plain X-ray chest PA view showing eventration of left dome of diaphragm (white arrows), (B) CECT chest-abdo showing the same with the contrast filled stomach just under the left dome (yellow arrow), (C) 1st look at laparoscopy showing the 'deep' left sided eventration of diaphragm (yellow asterisk), (D) Initiation of plication (yellow arrow).

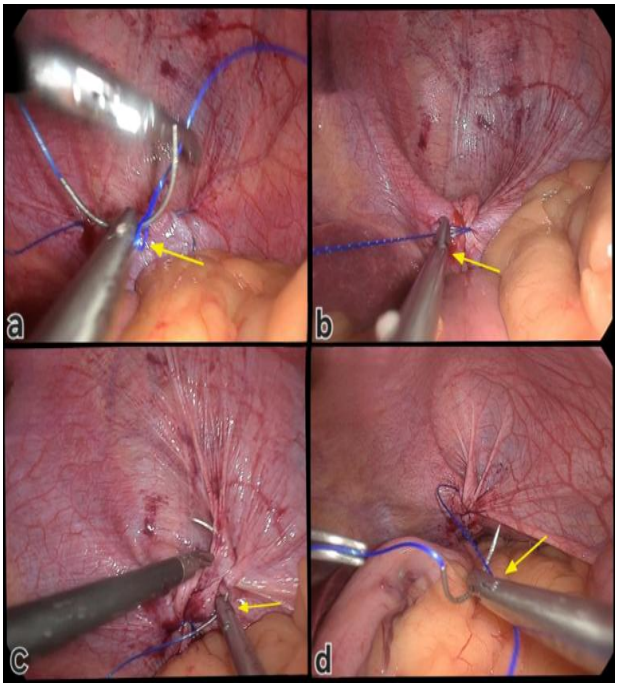

Figure 2 (A-D): Progressive plication of eventration of left dome of diaphragm using no. 1 V-loc (yellow arrows), causing 'flattening' effect.

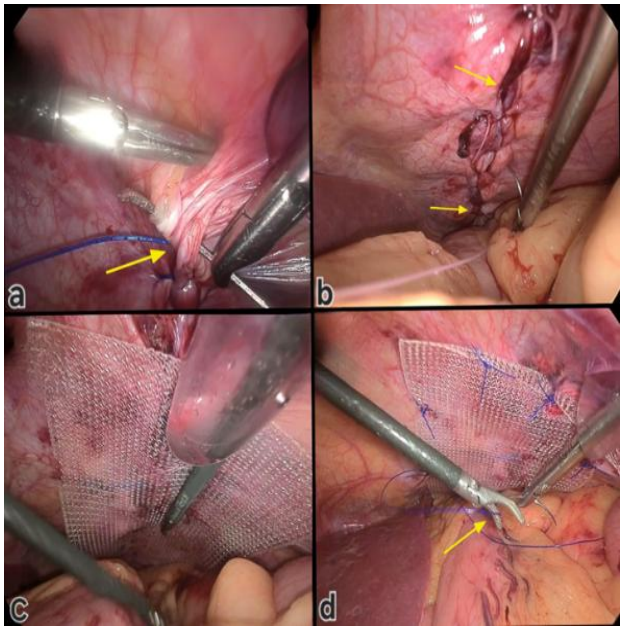

Figure 3 (A-D): (A) Plication of left dome of diaphragm coming to an end (yellow arrow), (B) Completed plication suture line (yellow arrows), (C) Titanium mesh placed optimally in situ over the suture line to buttress it, (D) Mesh being suture fixed to the surrounding diaphragm (yellow arrow).

DISCUSSION

From an embryological standpoint, congenital DE arises due to a defect in the migration of myogenic precursor cells (myoblasts) to the septum transversum during fetal development.[2] This aberration leads to the partial or complete substitution of functional diaphragmatic muscle with fibroelastic tissue. Consequently, the affected hemidiaphragm becomes abnormally thin, weak, and lacks normal contractile ability. The cephalad (upward) displacement of the diaphragm leads to a loss of effective partitioning between the thoracic and abdominal cavities. As a result, the diaphragm fails to function as a competent musculotendinous barrier, especially under conditions of fluctuating intra-abdominal or intrathoracic pressure.[2]

In advanced cases, the diaphragm becomes so attenuated that it resembles a passive membranous sheet incapable of maintaining the necessary pressure gradient between the thoracic and abdominal compartments. This pathophysiological shift results in a phenomenon termed thoracoabdominal compartment syndrome, where increases in pressure within one cavity are readily transmitted to the other, thereby eliminating their functional independence. While two distinct anatomical compartments may still exist, the physiological consequence is the emergence of a singular thoracoabdominal cavity.[3]

Eventration of the diaphragm may also be associated with various congenital anomalies and infectious conditions.

Congenital syndromes linked to this abnormality include spondylocostaldysostosis, Kabuki syndrome, Beckwith-Wiedemann syndrome, Poland syndrome, chromosomal abnormalities, pulmonary hypoplasia, congenital heart defects, and spinal muscular atrophy.[2] Furthermore, mitochondrial respiratory chain disorders have been implicated in neonatal diaphragmatic dysfunction.[2] Infectious etiologies, including in utero rubella or cytomegalovirus (CMV) infections, have also been documented as potential contributors.[2]

Acquired cases are more commonly observed and typically result from direct or indirect damage to the phrenic nerve or from muscular atrophy of the diaphragm.[2] Causative factors may include blunt or penetrating thoracoabdominal trauma, birth-related injuries, and iatrogenic insults during thoracic surgeries.[2] Phrenic nerve impairment can also arise secondary to systemic diseases such as multiple sclerosis, Guillain-Barré syndrome, radiation-induced neuropathy, nerve root compression, or connective tissue disorders.[2]

Most individuals with DE remain asymptomatic, and the condition is frequently discovered incidentally during radiological evaluation for unrelated complaints. However, symptomatic manifestations can vary widely depending on the severity of diaphragm dysfunction and the degree of organ displacement. The most common clinical feature is dyspnea, particularly on exertion, attributed to impaired diaphragmatic excursion leading to ventilation-perfusion mismatch in the lower lung zones. Patients may also exhibit features of restrictive lung disease, including reduced pulmonary volumes and mild to moderate hypoxemia, especially in the supine position.[4]

The diagnostic work-up begins with chest radiography, which typically reveals an elevated hemidiaphragm while maintaining intact cardiomediastinal borders. Both posteroanterior and lateral views are recommended to accurately assess the extent and nature of diaphragmatic elevation. Ultrasound of the diaphragm can help evaluate its motion, often demonstrating minimal or paradoxical movement-the latter being characterized by an upward shift during inspiration and downward motion during expiration, which is the inverse of the normal diaphragmatic movement.[5]

In cases where the diagnosis remains uncertain or when concomitant intrathoracic or intra-abdominal pathology is suspected, computed tomography (CT) of the chest and upper abdomen is advised. CT imaging allows for precise visualization of the diaphragm's contour and thickness, and typically shows a sharp edge along the elevated diaphragm in cases of eventration. Importantly, CT can also help distinguish DE from a diaphragmatic hernia, the latter being associated with a disruption of the diaphragmatic attachment or the presence of abdominal contents within the thoracic cavity.[2]

Functional assessment with pulmonary function tests (PFTs) may be beneficial in symptomatic patients or when there is uncertainty regarding the clinical significance of radiologic findings. PFTs often reveal reduced vital capacity and forced expiratory volume, consistent with a restrictive ventilatory pattern. In ambiguous cases, fluoroscopic evaluation, commonly known as the “sniff test,” can provide additional insights. In this dynamic imaging test, paradoxical upward movement of the affected hemidiaphragm during sniffing (a quick nasal inhalation) indicates diaphragmatic paralysis, which helps distinguish between true muscular weakness and neuropathic paralysis, such as that caused by phrenic nerve injury.[2]

The management of DE individualized based on the age of the patient, presence and severity of symptoms, underlying etiology, and associated anomalies. Asymptomatic individuals, particularly those with incidental findings, may be managed conservatively through observation and periodic follow-up.[3] However, surgical intervention becomes necessary in symptomatic patients, especially those with progressive respiratory compromise, persistent gastrointestinal symptoms, failure of conservative therapy, or risk of visceral herniation and compromised pulmonary expansion. Additionally, recurrent respiratory infections due to poor diaphragmatic excursion and reduced quality of life are also indications for surgical repair.[6] DE shows different management patterns in pediatric and adult population. In children, the condition is more likely to be symptomatic, and published series report that majority patients ultimately undergo surgical plication, with large cohort demonstrating operative intervention approximately 60-85% particularly in those with respiratory or feeding compromise.[7,8] In contrast DE in adult is rare and frequently asymptomatic consequently no definitive population based surgical rate has been established as the literature largely consist of small surgical case series. In adults diaphragmatic plication is reserved for symptomatic patients.[9]

The gold standard surgical treatment is diaphragmatic plication, which involves folding and suturing the redundant diaphragmatic tissue to flatten and stabilize it, thereby increasing intrathoracic volume and improving lung function. The procedure aims to restore more normal diaphragmatic contour and prevent paradoxical movement, ultimately enhancing ventilatory efficiency and alleviating symptoms.[3]

Contemporary literature strongly supports use of minimally invasive approaches-either laparoscopic or thoracoscopic techniques for diaphragmatic plication. These approaches offer numerous advantages, including reduced postoperative pain, shorter hospital stays, faster recovery, and fewer complications, particularly in pediatric and elderly populations. The laparoscopic approach is particularly favored because it provides excellent access to both hemidiaphragms and allows for

concurrent evaluation of intra-abdominal organs, which is beneficial when gastrointestinal symptoms coexist.

Overall, diaphragmatic plication is considered a safe and effective intervention with favorable long-term outcomes, offering significant improvements in symptom relief, pulmonary function, and quality of life.

CONCLUSION

As seen in this report, laparoscopic repair of DE is feasible, safe and yields good long term results; provided the presence of requisite advanced laparoscopic skills are ably complemented by an advanced setup.

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