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Diaphragm Eventration: Radiologic Assessment and Respiratory Implications-An Updated Review

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Abstract

Background: Diaphragm eventration is an abnormal elevation of all or part of a hemidiaphragm caused by thinning and weakness of muscle with preserved anatomic continuity. It may be congenital—due to incomplete myoblast migration and muscularization—or acquired, most commonly from phrenic nerve injury, trauma, thoracic surgery, or neurologic disease. Although often asymptomatic, extensive involvement can impair ventilatory mechanics, reduce lung volumes, and precipitate recurrent infections and failure to thrive in pediatrics.

Aim: To synthesize contemporary concepts in embryology, etiology, epidemiology, imaging diagnosis, functional assessment, management, and multidisciplinary care of diaphragm eventration, emphasizing radiologic differentiation from mimics and respiratory implications across age groups.

Methods: Narrative review of developmental anatomy, pathophysiology (congenital neurogenic muscular aplasia versus acquired denervation atrophy), population patterns, and diagnostic pathways integrating chest radiography, computed tomography (CT), ultrasonography, dynamic fluoroscopic sniff testing, and adjunct MRI; appraisal of pulmonary function testing (PFT) profiles; and comparative discussion of conservative care versus surgical plication (open, VATS, laparoscopic, robotic), perioperative care, and rehabilitation.

Results: Chest radiography suggests diagnosis; CT confirms continuity and delineates morphology; ultrasound provides bedside dynamic assessment (especially in children); fluoroscopic sniff testing differentiates paradoxical motion. Symptomatic patients exhibit restrictive physiology (reduced FVC/FEV_1). Indications for plication include refractory dyspnea, ventilator weaning failure, growth failure, and recurrent atelectasis/pneumonia. Plication improves mechanics and quality of life, with typical FEV_1/FVC gains up to ~30% on follow-up PFTs. Complications arise from underlying mechanics (atelectasis, pneumonia) and from surgery (effusions, DVT, arrhythmias), mitigated by standardized perioperative bundles and rehabilitation.

Conclusion: A radiology-led diagnostic pathway combined with tailored functional assessment supports risk-stratified management. Conservative observation suffices for asymptomatic cases; minimally invasive plication offers durable benefit for selected symptomatic patients when embedded in coordinated, multidisciplinary care.

Keywords: Diaphragm eventration; phrenic nerve; fluoroscopic sniff test; ultrasonography; computed tomography; pulmonary function tests; pediatric; video-assisted thoracoscopic surgery (VATS); plication; respiratory rehabilitation.

Introduction

The diaphragm develops between the fourth and twelfth weeks of embryogenesis, during which the central tendon forms from the anterior septum transversum. This tendon subsequently merges with the pleuroperitoneal folds laterally and the dorsal mesentery of the esophagus centrally, establishing the fundamental structure of the diaphragm. Concurrently, the muscular components arise from peripheral cervical somites at levels C3 to C5, which later migrate to form the diaphragmatic muscle fibers. In its mature form, the diaphragm is a dome-shaped

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structure approximately 2 to 4 mm thick, separating the thoracic and abdominal cavities. The central tendon lies beneath the heart and is fused with the parietal pericardium, while the peripheral muscle fibers attach to the xiphoid process of the sternum, lower ribs, and upper lumbar vertebrae. The crura anchor the diaphragm to the lumbar spine, with the right crus attaching from L1 to L3 and the left crus attaching from L2 to L3 [1]. This arrangement allows the diaphragm to act as the principal muscle of inspiration, contracting to increase thoracic volume and facilitate lung expansion. Motor innervation is provided by the left and right phrenic nerves. Dysfunction or absence of these nerves, whether congenital or acquired, can result in diaphragmatic paralysis. Eventration of the diaphragm refers to abnormal elevation of all or part of a hemidiaphragm due to weakened or absent muscular function while maintaining its anatomical attachments. Congenital eventration arises from incomplete muscular development, whereas acquired forms result from phrenic nerve injury, trauma, or infection.

The functional consequence of diaphragmatic eventration depends on the extent of muscular involvement. Partial elevation may be clinically silent, whereas complete hemidiaphragm involvement can compromise respiratory mechanics, leading to dyspnea, orthopnea, or recurrent pulmonary infections. Radiographic imaging, including chest X-ray and computed tomography, is essential for confirming the diagnosis, delineating the extent of diaphragmatic thinning, and differentiating it from other causes of diaphragmatic elevation such as hernia or paralysis. Management strategies range from conservative observation in asymptomatic cases to surgical plication in patients with significant respiratory compromise, aiming to restore diaphragmatic tension and optimize lung function [2][3][4]. Diaphragmatic eventration illustrates the intersection of developmental anatomy, neural function, and respiratory physiology, emphasizing the importance of early recognition and tailored intervention to preserve pulmonary capacity and overall cardiopulmonary health.

Etiology

Diaphragmatic eventration may present as either a congenital or acquired condition, each with distinct pathophysiologic mechanisms. Congenital eventrations arise from abnormal development of the diaphragmatic musculature and can affect anterior, posterolateral, or medial regions of the hemidiaphragm. During embryogenesis, a defect in myoblast migration to the septum transversum results in the replacement of functional muscle fibers with fibroelastic tissue. This structural deficiency creates a thin, weakened hemidiaphragm, predisposing the affected side to cephalic displacement. The congenital form is frequently associated with other developmental anomalies, including spondylocostal dysostosis, Kabuki syndrome, Beckwith-Wiedemann syndrome, and Poland syndrome. Chromosomal abnormalities and congenital malformations, such as pulmonary hypoplasia, spinal muscular atrophy, intestinal malrotation, and congenital heart defects, have also been linked to diaphragmatic eventration. Additionally, mitochondrial respiratory chain disorders may impair diaphragmatic function in the neonatal period. Certain in utero infections, including fetal rubella and cytomegalovirus, are implicated in rare cases of congenital diaphragmatic weakening [5][6][7]. Acquired diaphragmatic eventrations are more prevalent than congenital cases and typically result from phrenic nerve injury or diaphragmatic muscle atrophy. Traumatic causes include blunt or penetrating thoracic trauma, iatrogenic injury during thoracic surgery, and birth-related trauma. Phrenic nerve dysfunction may also develop secondary to neurologic disorders such as multiple sclerosis or Guillain-Barré syndrome, or from mechanical compression, radiation therapy, or connective tissue disease. Loss of phrenic nerve innervation leads to progressive muscle atrophy, diaphragmatic thinning, and cephalic elevation of the affected hemidiaphragm [2][8]. Eventrations, whether congenital or acquired, are classified anatomically based on the extent and laterality of diaphragmatic involvement. Complete eventration involves the entire hemidiaphragm, whereas partial eventration affects only a segment. Bilateral involvement, though rare, can result in significant respiratory compromise due to the reduction of ventilatory capacity on both sides. Understanding the etiology and anatomical classification of diaphragmatic eventration is essential for accurate diagnosis, prognostication, and planning of appropriate therapeutic interventions, which may range from conservative observation to surgical plication depending on the severity of functional impairment.

Epidemiology

Diaphragmatic eventration is a rare condition, and its true incidence and prevalence are difficult to determine due to the limited number of reported cases and the high rate of asymptomatic presentations. Estimates from case reports and small cohort studies suggest that the condition occurs in less than 0.05% of the general population, although some sources report an incidence of approximately 1 in 10,000 live births [6][9][10]. The condition shows a male predominance, with the left hemidiaphragm being more commonly affected than the right. This laterality may be related to differences in embryologic development, anatomical structure, or diaphragmatic exposure to thoracic and abdominal contents during growth. The actual prevalence of diaphragmatic eventration is likely higher than reported because many individuals remain asymptomatic and are incidentally diagnosed during imaging studies performed for unrelated conditions. In adults, acquired forms may be underrecognized, particularly when phrenic nerve injury or minor

diaphragmatic thinning produces only subtle radiographic findings without significant respiratory compromise. In neonates and pediatric populations, the prevalence may be underestimated because mild congenital eventration may not cause immediate respiratory distress and thus may go undetected unless imaging is performed for another indication. Population-level data are scarce, and most knowledge of diaphragmatic eventration is derived from case series, surgical reports, or retrospective analyses. The rarity of the condition limits the ability to conduct large-scale epidemiologic studies, making it difficult to identify precise risk factors, patterns of presentation, or long-term outcomes. Despite its low incidence, diaphragmatic eventration is clinically significant because of its potential impact on respiratory mechanics, particularly in cases of extensive or bilateral involvement, which may necessitate surgical intervention or close monitoring in affected individuals.

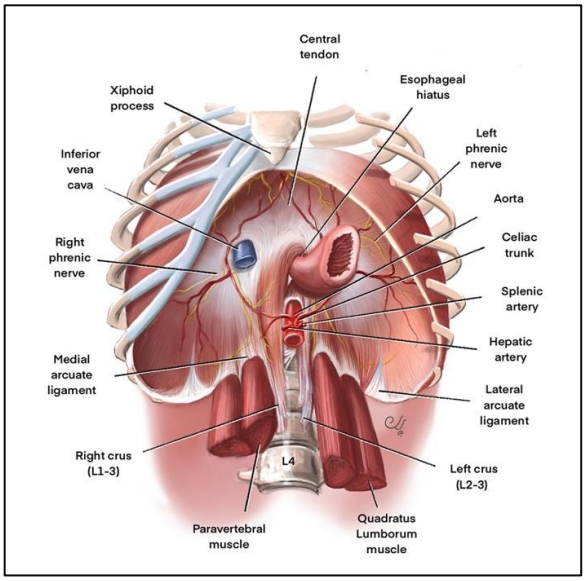

Fig. 1: Normal Diaphragm Anatomy.

Pathophysiology

Diaphragmatic eventration is characterized by an abnormal elevation of one hemidiaphragm, maintaining the structural continuity of the diaphragm without any true defect or herniation. The elevated segment may exhibit a distinct transition from normal diaphragm to the affected area, occasionally assuming a mushroom-like appearance on imaging or during surgical inspection [1]. This elevation results in reduced diaphragmatic contractility and compromised respiratory mechanics, particularly in severe or extensive cases. In congenital diaphragmatic eventration, the pathophysiology is linked to neurogenic muscular aplasia. During embryogenesis, there is incomplete or abnormal migration of myoblasts to the developing diaphragm, leading to a region where the muscular fibers are replaced by thin, fibrous, and aponeurotic tissue. These stretched and scattered muscle fibers are insufficient for normal contractile function, resulting in passive cephalic displacement of the hemidiaphragm. The reduced diaphragmatic strength compromises ventilation, particularly in the basal lung segments, which may predispose neonates to respiratory distress or recurrent pulmonary infections. The extent of functional impairment depends on the size and location of the affected segment, with larger anterior or posterolateral eventrations having a more pronounced impact. In contrast, acquired diaphragmatic eventration arises secondary to phrenic nerve injury or muscular atrophy rather than developmental defects. Trauma, thoracic surgery, or neurologic disorders such as Guillain-Barre syndrome or multiple sclerosis can impair phrenic nerve function, resulting in diaphragmatic paralysis and thinning of muscle fibers. Unlike congenital cases, these patients do not display aponeurotic changes; instead, the elevation occurs as a consequence of muscle denervation and atrophy, leading to diminished contractility and cephalic displacement of the diaphragm. In both congenital and acquired forms, the altered diaphragmatic mechanics can impair ventilation, reduce lung expansion, and increase reliance on accessory respiratory muscles, with clinical severity correlating with the degree of elevation and functional loss [11].

History and Physical

The clinical presentation of diaphragmatic eventration is highly variable, with many patients remaining asymptomatic throughout life. Incidental findings on routine chest imaging often lead to diagnosis in adults, whereas symptomatic cases in neonates or infants may present acutely. In neonates with bilateral congenital diaphragmatic eventration, respiratory compromise can be profound, occasionally resulting in cyanosis and acute respiratory failure. In both pediatric and adult populations, respiratory manifestations can include dyspnea on exertion, orthopnea, tachypnea, and shallow, labored breathing. Adults with diaphragmatic eventration may present with recurrent respiratory infections, chronic productive cough, or localized atelectasis due to impaired lung expansion on the affected side. Chest discomfort or pain is also reported, sometimes associated with arrhythmias or palpitations secondary to altered intrathoracic dynamics. Gastrointestinal manifestations in adults often correlate with increased intraabdominal pressure and may include dyspepsia, dysphagia, gastroesophageal reflux, and epigastric discomfort. Symptoms may be exacerbated during physical exertion, pregnancy, or in the context of fluid accumulation or infection. In infants, gastrointestinal involvement is more prominent, often manifesting as vomiting, bloating, constipation, poor oral intake, and failure to thrive. Severe cases may be complicated by

gastric volvulus. Respiratory distress in neonates may result from mediastinal shift toward the contralateral hemithorax, leading to compromised ventilation. A detailed history should assess for perinatal trauma, prior thoracic surgery, or underlying congenital syndromes associated with diaphragmatic abnormalities [2][11].

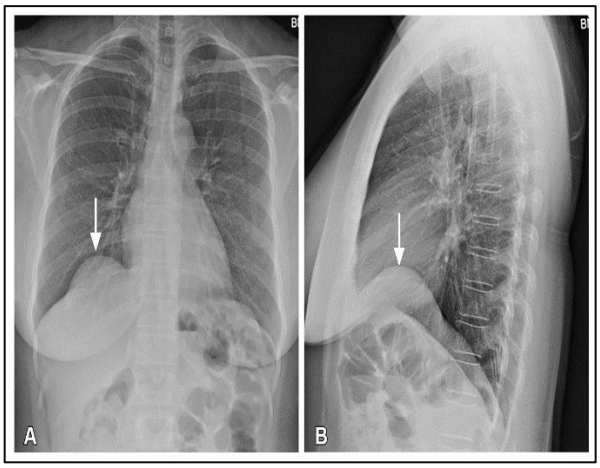

Fig. 2: Diaphragm Eventration.

Physical Examination

Physical findings are diverse and often nonspecific, reflecting the variable functional impairment caused by eventration. Shared findings in both adults and pediatric patients include tachypnea, accessory muscle use during respiration, and paradoxical chest wall motion on the affected side. Unilateral diaphragmatic elevation may result in decreased tactile fremitus, dullness to percussion, and diminished breath sounds. Gastrointestinal manifestations may be suggested by epigastric or periumbilical tenderness, and in severe cases, bowel sounds may be auscultated within the thoracic cavity. In infants with congenital eventration, additional findings may include a scaphoid abdomen, severe epigastric tenderness due to gastric volvulus, and musculoskeletal abnormalities related to birth trauma such as Erb palsy, clavicular fracture, or upper limb posturing with adduction, internal rotation, and forearm pronation. Signs of thoracic trauma should also be assessed. Acquired diaphragmatic eventration in adults often demonstrates evidence of previous blunt or penetrating thoracic trauma. Cardiovascular signs may include tachycardia, arrhythmias, or localized chest wall pain. Gastrointestinal examination may reveal abdominal tenderness due to increased intraabdominal pressure, fluid sequestration, ascites, or pregnancy-related displacement of intraabdominal organs. The variability of physical findings underscores the importance of integrating clinical history with imaging studies to confirm diagnosis and guide management.

Evaluation

The evaluation of diaphragmatic eventration requires a comprehensive approach that integrates clinical assessment, imaging, and functional testing. Most patients, particularly those with acquired eventration, are asymptomatic, and the condition is frequently discovered incidentally on routine chest imaging. In symptomatic individuals, a careful history and physical examination form the initial step in evaluation, identifying respiratory or gastrointestinal complaints that may suggest functional compromise of the diaphragm. Patients with a history of thoracic trauma, previous surgery, or underlying neuromuscular disorders warrant particular attention, as these factors may predispose to acquired diaphragmatic eventration. Chest radiography remains the first-line imaging modality for diagnosis. Posterior-anterior and lateral views typically reveal elevation of the affected hemidiaphragm, while cardiomediastinal contours remain preserved. The elevated portion of the diaphragm may appear dome-shaped or show a smooth, sharply defined edge. While chest x-ray can suggest the diagnosis, computed tomography (CT) of the thorax offers more precise anatomical delineation, particularly when there is uncertainty or suspicion for concomitant intrathoracic or intra-abdominal pathology. CT imaging allows assessment of diaphragm continuity, thickness, and the sharpness of the eventration margin, helping distinguish eventration from diaphragmatic hernia, in which the attachment sites and continuity of the diaphragm are disrupted [10][12]. Beyond anatomical assessment, functional evaluation is crucial for symptomatic patients or those being considered for surgical intervention. Pulmonary function tests (PFTs) typically reveal a restrictive pattern characterized by reduced forced vital capacity (FVC) and reduced forced expiratory volume in one second (FEV1). The restrictive deficit is more pronounced in bilateral eventration and is often improved following diaphragm plication. Assessment of respiratory muscle strength through maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) further quantifies diaphragmatic performance. MIP is typically reduced in eventration due to compromised diaphragmatic contraction, while MEP is generally preserved, reflecting the selective involvement of inspiratory muscles [12][13].

Dynamic evaluation of diaphragmatic motion can be performed using fluoroscopic sniff testing, which assesses diaphragmatic excursion during rapid inspiratory efforts. In diaphragmatic eventration, a portion of the hemidiaphragm may demonstrate abnormal motion, whereas in true diaphragmatic paralysis, paradoxical movement is often observed. This distinction, while informative, has limited impact on management, as both conditions may require surgical plication if conservative measures fail. In pediatric populations, fluoroscopic sniff testing can be technically challenging, making ultrasonography a preferred alternative for assessing diaphragmatic motion

without radiation exposure [12][14]. In congenital diaphragmatic eventration, prenatal diagnosis is feasible with high-resolution fetal ultrasonography, fetal CT, or magnetic resonance imaging (MRI). However, differentiating eventration from congenital diaphragmatic hernia can be difficult prenatally, as both may present with hypoplastic lungs and abdominal organs occupying a plane similar to the heart. Postnatally, suspicion arises when chest radiographs reveal an elevated hemidiaphragm, often during evaluation for respiratory distress. Confirmatory assessment includes fluoroscopy, dynamic MRI, or ultrasonography to evaluate diaphragmatic motion. Additional testing may be indicated based on the clinical history, such as investigations for underlying infections, neuromuscular disorders, developmental syndromes, or degenerative conditions contributing to the eventration [12]. Overall, evaluation of diaphragmatic eventration integrates anatomical, functional, and clinical assessment. Imaging defines the morphology and extent of diaphragmatic elevation, while functional testing quantifies respiratory compromise. Both congenital and acquired forms require tailored assessment to guide management, monitor progression, and determine candidacy for surgical intervention, ensuring that the underlying etiology and clinical impact are appropriately addressed.

Treatment / Management

The management of diaphragmatic eventration is guided primarily by the underlying etiology, the severity of symptoms, and the patient's functional status. In cases where patients are asymptomatic or present with mild clinical manifestations, conservative or supportive care is often sufficient. This approach focuses on maintaining adequate oxygenation and optimizing nutritional and pulmonary function. In instances of hypoxemia, supplemental oxygen is administered to maintain adequate oxygen saturation, typically via nasal cannula. If this approach is insufficient, nasal continuous positive airway pressure (CPAP) may be required to support respiratory function and prevent atelectasis. In neonates and infants, gastrointestinal symptoms and failure to thrive are common, necessitating careful nutritional support. Enteral feeding via gavage or, in severe cases, parenteral nutrition may be required to ensure sufficient caloric intake, fluid balance, and electrolyte replenishment to meet metabolic demands. Physical therapy and pulmonary rehabilitation may further enhance respiratory mechanics and overall functional capacity in both pediatric and adult patients [10][11]. Surgical intervention is indicated when conservative measures fail or when the severity of diaphragmatic eventration leads to significant functional compromise. Diaphragmatic plication is the standard surgical approach, aiming to mechanically stabilize the weakened hemidiaphragm. The procedure involves creating pleats in the affected hemidiaphragm using U-stitches and anchoring them to reduce diaphragmatic elevation. This maneuver flattens and lowers the hemidiaphragm, thereby increasing intrathoracic volume and allowing for improved lung expansion. Plication can be performed through open thoracotomy, minimally invasive video-assisted thoracoscopic surgery (VATS), laparoscopic techniques, or robotic-assisted surgery. Postoperative management typically includes placement of a thoracostomy tube to facilitate pleural drainage, which is usually maintained until output falls below 200 mL per day. It is important to note that while surgical plication improves lung expansion and reduces symptoms, it does not restore intrinsic contractile function to the affected hemidiaphragm [12][15][16].

Indications for surgical plication include persistent respiratory distress unresponsive to conservative management, dyspnea that is attributable solely to diaphragmatic dysfunction rather than comorbid conditions such as heart failure or primary pulmonary disease, and infants exhibiting inadequate nutritional intake or failure to thrive. Other indications include recurrent or life-threatening pneumonia and inability to wean from mechanical ventilation due to diaphragmatic weakness. Postoperative follow-up is essential to monitor recovery, assess pulmonary function, and detect potential complications. Follow-up evaluations often include repeat posterior-anterior and lateral chest radiographs to assess hemidiaphragm position and expansion, pulmonary function tests to evaluate improvements in respiratory mechanics, and quality-of-life assessments, such as the St George Respiratory Questionnaire, which captures patient-reported outcomes regarding physical activity, respiratory symptoms, and psychosocial well-being. The frequency and duration of follow-up are tailored to the individual patient based on the severity of preoperative symptoms, age, and response to surgery. Collectively, these strategies aim to optimize respiratory function, enhance quality of life, and prevent long-term morbidity associated with diaphragmatic eventration.

Differential Diagnosis

When evaluating a patient with suspected diaphragmatic eventration, several conditions must be considered to ensure accurate diagnosis and appropriate management. Diaphragmatic paralysis is one of the primary conditions to differentiate. While both eventration and paralysis can present with an elevated hemidiaphragm on imaging, paralysis typically affects the entire hemidiaphragm and may show paradoxical motion during inspiration, whereas eventration can involve either a portion or the entirety of the hemidiaphragm, often maintaining normal attachments [1]. Phrenic nerve palsy should also be

considered, as it may result from trauma, surgical injury, or neuropathic disease and can mimic the presentation of acquired eventration. Diaphragmatic hernia represents another differential, particularly when the continuity of the diaphragm is disrupted and abdominal contents protrude into the thoracic cavity. Imaging modalities such as chest CT or MRI can aid in differentiating hernias from eventration, as the latter maintains structural continuity of the diaphragm [2]. Pulmonary conditions such as lung consolidation or subpulmonic pleural effusion can also mimic diaphragmatic elevation, and clinical correlation along with imaging is necessary to exclude these causes. Pleural masses, including tumors or localized fibrosis, may create the appearance of diaphragmatic elevation on radiographs and should be ruled out. Traction injuries or iatrogenic damage, particularly following thoracic surgery, may result in diaphragmatic elevation or functional compromise. Additionally, intra-abdominal conditions such as ascites or hepatosplenomegaly can exert upward pressure on the diaphragm, mimicking eventration on imaging studies. Clinicians must consider these possibilities in conjunction with history, physical examination, and radiologic findings to differentiate true diaphragmatic eventration from other causes of apparent diaphragm elevation. Accurate differentiation is crucial, as management strategies differ significantly depending on the underlying etiology [1][2].

Prognosis

The prognosis of diaphragmatic eventration is generally favorable and is largely determined by the extent of diaphragmatic elevation, laterality (unilateral versus bilateral), the presence of coexisting cardiopulmonary disease, and the patient's age and functional reserve. Many adults—particularly those with modest, unilateral eventration—remain asymptomatic for years and require no intervention beyond periodic clinical follow-up. In infants and young children, the clinical course is more variable: mild congenital eventration may be discovered incidentally, whereas extensive elevation can present with respiratory distress, feeding difficulty, recurrent lower respiratory infections, and failure to thrive. In such pediatric cases, early recognition and timely referral to a center experienced in pediatric thoracic care can be decisive in improving growth and developmental trajectories. For symptomatic patients, especially those with exertional dyspnea, recurrent atelectasis or pneumonitis, or ventilator dependence after cardiac or thoracic surgery, diaphragm plication offers durable relief. Contemporary series demonstrate substantive improvements in exercise tolerance, dyspnea scores, and pulmonary function after plication, with gains in forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) reported up to approximately 30% on follow-up pulmonary function testing [16][17]. These improvements typically emerge within weeks to months as lung re-expansion occurs and paradoxical or ineffective diaphragmatic motion is eliminated, thereby restoring more efficient mechanics to the thoracic cage. Minimally invasive approaches (thoracoscopic or robotic) are associated with shorter hospital stays, reduced postoperative pain, and earlier mobilization when compared with traditional open thoracotomy, while maintaining comparable functional gains in appropriately selected patients.

Long-term outcomes are influenced by modifiable cofactors. Obesity, chronic obstructive pulmonary disease (COPD), severe kyphoscoliosis, and uncontrolled gastroesophageal reflux can blunt postoperative gains or perpetuate symptoms, whereas pulmonary rehabilitation, vaccination against influenza and pneumococcus, smoking cessation, and optimal treatment of comorbidities enhance durability of benefit. Recurrence after a well-constructed plication is uncommon; when present, it is usually related to suture failure, progressive tissue laxity, or new phrenic neuropathy. In bilateral eventration or in patients with neuromuscular disease, expectations should be calibrated to more modest improvements, and noninvasive ventilatory support (eg, nocturnal CPAP/BiPAP) may remain necessary. Overall, with careful patient selection, disciplined perioperative care, and targeted rehabilitation, quality of life and functional capacity improve meaningfully for the majority of symptomatic patients undergoing plication, while asymptomatic individuals can often be safely managed with observation and risk-factor optimization.

Complications

Complications of diaphragmatic eventration arise from impaired ventilatory mechanics, altered intrathoracic and intra-abdominal pressure relationships, and susceptibility to infection. A chronically elevated, thinned, and poorly contractile hemidiaphragm reduces ipsilateral lung volume, predisposes to basal atelectasis, and disrupts ventilation–perfusion matching. Over time, this can culminate in acute or chronic respiratory failure, especially in patients with limited cardiopulmonary reserve, underlying parenchymal disease, or superimposed infections. Pneumonitis and pneumonia are common downstream consequences of impaired cough mechanics and mucus clearance. In infants and small children, the combined burden of increased work of breathing, early satiety from cephalad displacement of abdominal viscera, and recurrent intercurrent illness can lead to nutritional deficiency and failure to thrive. Mechanical shifts in the mediastinum during episodes of acute decompensation can provoke cardiac arrhythmias, particularly in patients with preexisting conduction abnormalities or structural heart disease. Gastrointestinal sequelae—such as early satiety, bloating, reflux, and constipation—are frequent clinical accompaniments that exacerbate dyspnea by further elevating intra-abdominal pressure. While true

gastric volvulus is rare in eventration compared with diaphragmatic hernia, vigilance is warranted for severe, refractory gastrointestinal symptoms. Surgical correction with diaphragm plication has its own risk profile. Perioperative complications include pneumonia, pleural effusions, prolonged air leak, postoperative pain with splinting, deep venous thrombosis (DVT), and cardiopulmonary events such as supraventricular arrhythmias or ischemia in susceptible individuals. On the abdominal side, rapid reduction of chronically displaced viscera can, in rare cases, contribute to abdominal compartment syndrome, heralded by rising intra-abdominal pressure, oliguria, and escalating ventilatory pressures. Other recognized risks include wound infection, postoperative ileus, hemothorax, and, infrequently, phrenic nerve injury (more relevant to surgical procedures in the neck or mediastinum than to standard plication planes). Late complications encompass symptom recurrence from plication loosening or suture failure, persistent or recurrent pleural effusion, and less commonly chronic neuropathic pain along intercostal distributions. Mitigation strategies are multidisciplinary: prehabilitation and smoking cessation reduce pulmonary complications; perioperative lung-protective strategies and incentive spirometry counter atelectasis; early mobilization and pharmacologic prophylaxis reduce thromboembolic events; and thoughtful intraoperative technique—including nonabsorbable suture selection and tension-balanced plication—lowers recurrence risk. In pediatric populations, nutrition optimization, aspiration precautions, and close growth monitoring limit downstream morbidity. Ultimately, the overall risk–benefit balance favors plication in appropriately chosen symptomatic patients, whereas conservative management is preferable for asymptomatic or minimally symptomatic individuals.

Postoperative and Rehabilitation Care

Postoperative management following diaphragm plication (or observation after decompensation in nonoperative cases) is best delivered within a structured pathway that anticipates respiratory, hemodynamic, and abdominal physiology changes. Immediate care is often provided in a high-acuity or intensive care unit (ICU) setting, particularly for patients who required preoperative or intraoperative mechanical ventilation, have significant comorbidities, or underwent extensive plication. A planned, early trial of extubation is prioritized once gas exchange, hemodynamics, and protective airway reflexes are adequate, recognizing that plication improves mechanics but surgical pain and altered chest wall compliance can transiently increase work of breathing. Post-extubation support may include high-flow nasal cannula or noninvasive ventilation (CPAP/BiPAP) as a bridge, especially overnight. Because operative restoration of diaphragmatic contour can acutely redistribute abdominal contents caudally, intra-abdominal pressure (IAP) monitoring is recommended in high-risk patients (eg, those with tense abdomen, ascites, large visceral reduction, or oliguria). Rising IAP—manifesting as escalating ventilator pressures, reduced urine output, abdominal tenderness, or increased lactate—should prompt evaluation for abdominal compartment syndrome, a surgical emergency requiring urgent decompression. Concurrently, standardized pulmonary hygiene bundles—incentive spirometry, chest physiotherapy, early mobilization, and adequate analgesia—are essential to prevent atelectasis and pneumonia. Multimodal analgesia (acetaminophen, NSAIDs where appropriate, limited opioids) and regional techniques (paravertebral blocks, intercostal nerve blocks, or epidural analgesia for open cases) improve cough mechanics, reduce splinting, and hasten ambulation.

Other key elements include meticulous fluid management to avoid pulmonary edema, judicious diuresis in the presence of pleural effusions, and routine chest imaging to confirm lung re-expansion and exclude significant pneumothorax or hemothorax. Chest tube management (when used) follows standard criteria for removal once output is acceptable and no air leak persists. Venous thromboembolism prophylaxis combines pharmacologic agents (unless contraindicated) with sequential compression devices and early ambulation. Nutritional support—enteral feeding advancement in pediatrics or dietitian-guided plans in adults—addresses preoperative deficits and supports healing. Respiratory therapy and pulmonary rehabilitation begin in the inpatient setting and continue after discharge, focusing on diaphragmatic breathing retraining, graded aerobic conditioning, and inspiratory muscle endurance. A typical surveillance program includes clinic follow-up at 2–4 weeks for wound review and symptom assessment, then formal pulmonary function tests (PFTs) at approximately 8–12 weeks and again at 6–12 months to quantify functional gains. Patients should receive clear activity guidance, with avoidance of heavy lifting and valsalva maneuvers for 4–6 weeks to protect the repair, while encouraging progressive walking targets (eg, step counts) to reduce deconditioning. Education on vaccination (influenza, pneumococcal when indicated), smoking cessation, sleep apnea screening, and reflux management completes the rehabilitation package, enhancing durability of benefit and reducing readmissions.

Consultations

Optimal care for diaphragmatic eventration hinges on early involvement of an interprofessional team tailored to the patient's age, symptom severity, and comorbid conditions. A thoracic surgeon (or pediatric/thoracic surgeon in children) evaluates

candidacy for plication, selects the operative approach (thoracoscopic, robotic, or open), and delineates perioperative risks in light of anatomy and physiology. An adult or pediatric pulmonologist provides baseline and follow-up PFTs, oversees pulmonary hygiene strategies, and co-manages conditions such as asthma, COPD, or bronchiectasis that can compound postoperative risk. In higher-acuity cases, an intensivist coordinates ventilatory weaning, sedation and delirium prevention, and hemodynamic optimization, ensuring early extubation trials align with respiratory mechanics and gas exchange. A respiratory therapist is indispensable for protocolized incentive spirometry, airway clearance adjuncts, and bedside coaching in breathing techniques. Pulmonary rehabilitation specialists and physical therapists design individualized conditioning plans that begin at the bedside and progress through outpatient phases, with objective milestones (eg, six-minute walk distance) to document gains. For infants and children, a nutritionist/dietitian addresses caloric density, feeding strategies, and growth monitoring, while a speech and swallow therapist may be consulted if aspiration risk or dysphagia is suspected. Given the interplay of intrathoracic pressure shifts, cardiology input may be appropriate in patients with structural heart disease, pulmonary hypertension, or arrhythmias, and anesthesiology consultation assists with risk stratification and analgesia planning in complex cases. Additional resources extend the safety net. Pain management teams guide multimodal and regional analgesia to reduce opioid exposure. Social workers and case managers identify barriers to follow-up, coordinate home health services (eg, incentive spirometer coaching, oxygen needs), and secure equipment for patients with limited access. In select scenarios—such as cervical trauma or suspected phrenic neuropathy—neurology or physiatry input can clarify prognosis and adjunctive therapies. Clear role delineation and shared documentation across these consultants ensure seamless transitions from inpatient to outpatient settings, shorten recovery timelines, and reduce preventable readmissions.

Patient Education

Because many cases of diaphragm eventration are discovered incidentally and remain asymptomatic, patient (and caregiver) education focuses on symptom recognition, risk-reduction behaviors, and clarity about when to seek medical attention. Patients should be taught to recognize warning signs of decompensation—worsening shortness of breath at rest or with exertion, tachypnea, orthopnea, new wheeze or cough, chest discomfort, fever, or purulent sputum—prompting early evaluation to avert pneumonia or respiratory failure. In infants and children, caregivers should monitor feeding tolerance, weight gain, and increased work of breathing (retractions, nasal flaring), as well as recurrent respiratory infections that may signal the need for reassessment. Daily practices that reduce symptoms are practical and effective. Breathing exercises (diaphragmatic and pursed-lip breathing) and postural strategies (sleeping with head-of-bed elevation, side-lying on the unaffected side if more comfortable) can improve mechanics and comfort. Patients should avoid heavy meals and late-evening large fluid intake that distend the abdomen and exacerbate dyspnea; small, frequent meals can be helpful, particularly in pediatric or frail patients. Preventive health measures—including influenza and pneumococcal vaccinations where indicated, smoking cessation, and good hand hygiene—reduce infection risk. For those with reflux, anti-reflux strategies (meal timing, head-of-bed elevation, pharmacotherapy as directed) may mitigate nocturnal symptoms and microaspiration. For individuals scheduled for plication, preoperative counseling should set expectations regarding hospital course, likely need for chest drainage, pain management, and the importance of early mobilization and incentive spirometry to prevent atelectasis. Postoperative instructions should specify wound care, signs of infection, activity restrictions (typically no heavy lifting or strenuous core engagement for 4–6 weeks), and follow-up timelines for clinic review and PFTs. A printed or digital action plan helps patients know whom to contact for symptom escalation. Finally, clarifying the distinction between eventration (attenuated, elevated but intact diaphragm) and diaphragmatic hernia (true defect with herniation of abdominal contents) reduces anxiety and aligns expectations, reinforcing that many patients with eventration can be safely observed and that surgery, when required, is aimed at restoring mechanics rather than repairing a hole.

Other Issues

- Definition and etiology: Diaphragm eventration is an abnormal elevation of part or all of the hemidiaphragm due to a thinned, weakened muscular segment with intact continuity. Etiology is congenital (incomplete myoblast migration and muscularization) or acquired, most commonly following phrenic nerve injury from cardiothoracic surgery, cervical trauma, or neuropathies; acquired causes are more frequent in adults.
- Clinical spectrum: Most patients are asymptomatic; symptomatic individuals report exertional dyspnea, orthopnea, early satiety, and, in pediatrics, feeding difficulty and recurrent infections. Physical exam may reveal decreased breath sounds at the base and paradoxical abdominal motion.
- Imaging and physiology: Diagnosis is confirmed with chest radiography (elevated hemidiaphragm), ultrasonography (M-mode

excursion; tissue thickness), CT for anatomy, and fluoroscopic sniff testing demonstrating diminished or paradoxical motion. Ultrasound is particularly useful at the bedside and avoids radiation. Consider dynamic MRI in complex or equivocal cases.

- Differential diagnosis: Distinguish from diaphragmatic paralysis (complete loss of motion), subpulmonic effusion, lower lobe atelectasis, eventration versus hernia, and diaphragmatic rupture post trauma. Clinical context and dynamic imaging resolve ambiguity.
- Management: Asymptomatic or mildly symptomatic cases are managed conservatively with observation, vaccination, pulmonary hygiene, reflux control, and risk-factor modification. Surgical plication is indicated for significant symptoms, recurrent infection/atelectasis, ventilator dependence attributable to eventration, or growth failure in children. Thoracoscopic or robotic approaches are preferred when feasible; nonabsorbable sutures and tension-balanced rows reduce recurrence. Mesh reinforcement is rarely required.
- Outcomes: Most symptomatic patients experience improved dyspnea, exercise capacity, and PFT gains (FEV_1/FVC increases up to ~30% in follow-up testing) with sustained quality-of-life benefits [16][17]. Set expectations conservatively in bilateral disease or neuromuscular disorders.

These pearls underscore a pragmatic approach: confirm diagnosis with dynamic imaging, reserve surgery for clearly symptomatic or complicated cases, and pair intervention with structured rehabilitation to maximize durable benefit.

Enhancing Healthcare Team Outcomes

High-reliability care for diaphragm eventration depends on interprofessional coordination from presentation through rehabilitation. Emergency and primary care clinicians should maintain a diagnostic index of suspicion when evaluating unexplained dyspnea, asymmetric basal opacities, or recurrent pneumonia; standardized imaging pathways (portable CXR → targeted ultrasound → CT/fluoro sniff in select cases) shorten time to diagnosis. Clear referral triggers to thoracic surgery and pulmonology—such as symptomatic limitation, failure to wean from mechanical ventilation, recurrent atelectasis, pediatric growth faltering, or significant radiographic elevation—ensure timely specialty input. In the inpatient setting, daily multidisciplinary rounds that include surgeons, intensivists, respiratory therapists, nursing, pharmacists, and physical/occupational therapists align goals: ventilator weaning plans, analgesia optimization with multimodal regimens, DVT prophylaxis, chest tube management, and mobilization targets. Respiratory therapy operationalizes pulmonary hygiene and teaches breathing strategies; physical therapy progresses ambulation and endurance; pharmacy minimizes sedatives that impair ventilation, assures appropriate antimicrobial use for infections, and manages anticoagulation; dietitians address caloric needs and reflux-sparing meal patterns; and social work/case management coordinates home resources (eg, incentive spirometers, oxygen, transportation) and addresses barriers that jeopardize follow-up.

Quality improvement is strengthened by adopting order sets and care bundles (extubation readiness criteria, IS frequency, early mobilization milestones), tracking process and outcome metrics (time to diagnosis, ventilator days, pneumonia rates, readmissions, patient-reported dyspnea scores), and conducting morbidity and mortality reviews for complications or readmissions. Telehealth or nurse-led calls within 72 hours of discharge identify early warning signs, reinforce inhaled therapies and IS use, and troubleshoot pain control. Education for patients and caregivers—delivered in plain language and culturally attuned—enhances self-management and reduces unnecessary ED utilization. By embedding these practices, teams improve patient safety, reduce medical errors, and achieve better functional outcomes with shorter lengths of stay and lower readmission rates. Ultimately, it is the consistency of this coordinated approach—rather than any single intervention—that translates evidence-based care into reliable, patient-centered results in the management of diaphragm eventration.

Conclusion:

Diaphragm eventration bridges developmental anatomy, neural integrity, and respiratory mechanics, producing a clinical spectrum that ranges from incidental radiographic findings to substantial ventilatory compromise. Accurate, timely diagnosis hinges on a radiology-centric algorithm: chest radiography for detection; CT to confirm diaphragmatic continuity and define morphology; and dynamic modalities—ultrasound or fluoroscopic sniff testing—to characterize motion. Functional corroboration with PFTs typically reveals a restrictive pattern whose severity parallels anatomic involvement and clinical symptoms. Management should be individualized. Asymptomatic or mildly symptomatic patients benefit from conservative strategies—vaccination, pulmonary hygiene, reflux control, and observation—while clearly symptomatic patients with dyspnea attributable to eventration, recurrent atelectasis or infection, failure to thrive, or difficulty weaning from ventilation are appropriate candidates for plication. Minimally invasive approaches (thoracoscopic/robotic) provide reliable

symptom relief, earlier mobilization, and sustained improvements in lung volumes (often ~30% gains in FEV_1/FVC), particularly when coupled with structured postoperative rehabilitation and meticulous analgesia. Outcomes are optimized by an interprofessional model that aligns thoracic surgery, pulmonology, anesthesia/critical care, respiratory therapy, nutrition, and rehabilitation, supported by standardized bundles (extubation readiness criteria, incentive spirometry frequency, early mobilization, VTE prophylaxis) and clear follow-up plans for imaging and PFTs. With judicious patient selection, disciplined perioperative care, and attention to modifiable comorbidities, long-term quality of life and functional capacity improve for most symptomatic patients, while conservative surveillance remains safe for the majority who are asymptomatic.

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