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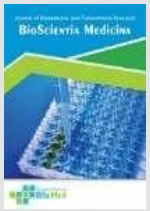

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Delayed Right-Sided Traumatic Diaphragmatic Rupture Complicated by Hepatothorax and Visceral Herniation a Decade Post-Trauma: A Case Report

Mada Aji Prakoso[1*], Albertus Ari Adrianto[2], Parish Budiono[2]

[1]Department of Surgery, Faculty of Medicine, Universitas Diponegoro, Semarang, Indonesia

[2]Department of Digestive Surgery, Dr. Kariadi General Hospital, Semarang, Indonesia

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***Corresponding author:**
Mada Aji Prakoso

E-mail address:
prakosomada@gmail.com

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A B S T R A C T

Background: Traumatic diaphragmatic rupture (TDR) is a rare consequence of high-energy blunt trauma, accounting for less than 1% of all traumatic injuries. Right-sided ruptures are particularly uncommon, representing only 5% to 20% of cases, largely due to the protective anatomical positioning of the liver. Consequently, right-sided injuries are notoriously difficult to detect, often leading to a delayed diagnosis. **Case presentation:** We present the case of a 29-year-old female who presented with progressive dyspnea ten years after sustaining a motor vehicle collision. Her initial injury was managed as a right-sided hemothorax, with the diaphragmatic defect remaining undetected. A decade later, imaging revealed an elevated right hemidiaphragm with massive herniation of the liver, gallbladder, transverse colon, and omentum into the right thoracic cavity. The patient underwent a successful abdomino-thoracotomy with primary repair of a 10 cm x 4 cm defect, reinforced with a prosthetic mesh. She was discharged on postoperative day seven with an uneventful recovery. **Conclusion:** This case emphatically highlights the persistent, lifelong risk of delayed visceral herniation following thoracoabdominal trauma. A high index of suspicion must be maintained for patients presenting with delayed respiratory symptoms, as prompt surgical intervention yields excellent outcomes.

1. Introduction

Traumatic diaphragmatic rupture (TDR) represents a profoundly complex and highly morbid clinical entity within the realm of modern surgical traumatology. Despite the continuous evolution of advanced trauma life support protocols and high-resolution diagnostic imaging, TDR remains a rare occurrence, constituting significantly less than 1% of all traumatic injuries identified in modern trauma centers. However, this statistical rarity belies the critical nature of the injury. The presence of a diaphragmatic tear serves as a definitive marker of catastrophic kinetic energy transfer, indicating that the patient has sustained physical forces capable of disrupting one of the most robust and biologically vital barriers in the human body. To understand the gravity of this injury, one must first appreciate the intricate architectural and physiological functions of the diaphragm itself.[1]

The diaphragm is a paramount musculotendinous septum that serves as the primary engine of ventilation while concurrently maintaining a strict anatomical boundary between two distinctly unique physiological environments: the positive-pressure abdominal cavity and the negative-pressure thoracic cavity.[2] Architecturally, it is composed of a tough central aponeurotic tendon surrounded by radiating

peripheral muscle fibers that anchor to the lower ribs, the sternum, and the lumbar vertebrae. This structural configuration allows the diaphragm to act as a dynamic piston during the respiratory cycle. With each inspiration, the diaphragmatic musculature contracts and descends, increasing the intrathoracic volume to draw air into the lungs, while simultaneously increasing the intra-abdominal pressure. This constant, rhythmic fluctuation creates a permanent trans-diaphragmatic pressure gradient. Under resting conditions, the pressure differential between the abdomen and the thorax ranges from 7 to 20 centimeters of water. However, during maximal respiratory efforts, coughing, or Valsalva maneuvers, this gradient can surge dramatically. In a healthy individual, the intact diaphragm easily withstands these immense pressure shifts. Yet, when the structural integrity of this barrier is compromised by trauma, this very pressure gradient transforms from a life-sustaining physiological mechanism into a relentless pathological force.[3]

The overwhelming majority of TDR cases encountered in clinical practice—accounting for approximately 72% to 94% of all documented incidences—are secondary to severe blunt force trauma. These injuries are predominantly the result of high-velocity motor vehicle collisions, falls from significant heights, or severe crush injuries involving heavy machinery.[4] The biomechanics underlying blunt diaphragmatic rupture are governed by the principles of fluid dynamics within a closed biological cylinder. Upon impact, the abdominal cavity acts like a fluid-filled compartment. A sudden, violent deceleration force or a direct crushing impact against the abdominal wall—such as a steering wheel impacting the epigastrium or a seatbelt restraining the lower abdomen during a high-speed crash—causes a massive and instantaneous spike in intra-abdominal pressure. Following Pascal's principle, this kinetic energy is transmitted equally in all directions throughout the peritoneal cavity. Because the pelvic floor and the muscular abdominal walls are highly resistant to sudden stretching, the kinetic energy inevitably seeks the path of least resistance. The diaphragm, stretched tightly across the superior aspect of the abdominal cavity, absorbs the brunt of this explosive upward force. The sudden distension tears the diaphragmatic muscle fibers, most frequently at the posterolateral aspects where the embryonic pleuroperitoneal membranes fuse, resulting in a large, radial defect.

Despite the violence of the inciting mechanism, a significant and enduring challenge in the clinical management of TDR is the alarmingly high rate of delayed or missed diagnosis.[5] Contemporary surgical literature suggests a troubling reality: up to 66% of all diaphragmatic injuries may not be evident during the initial trauma evaluation. This diagnostic failure rate is even more pronounced in the chaotic environment of the emergency department, where it can soar to an estimated 90%. The reasons for this profound diagnostic oversight are multifactorial and deeply rooted in the realities of polytrauma management. Patients who sustain sufficient force to rupture their diaphragm rarely present with an isolated injury. They typically arrive in the trauma bay with a constellation of severe, immediately life-threatening conditions, including traumatic brain injuries, hemorrhagic shock, pelvic ring fractures, splenic lacerations, or massive hemothoraces. The acute management rightly focuses on securing the airway, controlling massive hemorrhage, and stabilizing the hemodynamics. Consequently, the subtle respiratory or gastrointestinal signs of a diaphragmatic tear are often completely obscured by the dominating clinical picture of these concurrent injuries.

Furthermore, the initial diagnostic modalities utilized in the acute trauma setting are notoriously insensitive for detecting diaphragmatic integrity. The standard supine anteroposterior chest radiograph, while indispensable for identifying tension pneumothorax or massive hemothorax, lacks the spatial resolution and proper anatomical orientation to clearly visualize the diaphragmatic contour. Even focused assessment with sonography for trauma (FAST), which is excellent for detecting intra-

abdominal hemorrhage, does not reliably assess the continuity of the diaphragmatic muscle. Unless there is overt and massive herniation of hollow viscera into the thoracic cavity at the time of the initial scan, the injury remains hidden in plain sight.[6]

The diagnostic complexity reaches its absolute zenith when addressing right-sided traumatic diaphragmatic ruptures. Anatomically and epidemiologically, the right hemidiaphragm presents an exceptional challenge, accounting for a mere 5% to 20% of all documented TDR cases. This significant left-to-right disparity is not due to a difference in structural strength between the two sides, but rather to the formidable presence of the liver.[7] The right lobe of the liver is a massive, dense, parenchymal organ that occupies the entire right upper quadrant of the abdomen. Its superior surface is intimately attached to the inferior surface of the right hemidiaphragm via the coronary ligaments and the bare area. This anatomical configuration provides a profound protective buffering effect. When a sudden spike in intra-abdominal pressure occurs, the sheer mass of the liver diffuses the kinetic energy over a larger surface area, often protecting the right diaphragmatic muscle from the bursting forces that would easily rupture the left side.

However, when the traumatic force is so catastrophic that a right-sided defect does occur, the liver paradoxically becomes the clinician's greatest diagnostic adversary. Following the rupture, the liver itself acts as an immediate biological plug. It is drawn upward into the muscular defect by the negative intrathoracic pressure, effectively tamponading the tear. This temporary occlusion provides a false seal. By blocking the defect, the liver prevents the immediate upward migration of air-filled, hollow abdominal viscera like the stomach or the colon. Consequently, the initial trauma chest radiograph or even early computed tomography scans may appear deceptively normal, showing only a slight elevation of the right hemidiaphragm that is easily dismissed as poor inspiratory effort or simple atelectasis from a concurrent pulmonary contusion.

If this right-sided injury is left unrecognized during the acute hospitalization phase, the patient unknowingly enters a treacherous latent period. The natural history of a missed TDR was classically described by Grimes in 1974, who categorized the pathology into three distinct chronological phases: the acute phase at the time of injury, the latent or interval phase, and the obstructive phase. The latent phase can last for weeks, months, or remarkably, even years. During this period, the patient may remain entirely asymptomatic or experience only vague, intermittent symptoms such as mild postprandial fullness or atypical chest discomfort. However, beneath the surface, a relentless biomechanical process is underway. Because the diaphragm is in constant motion, a traumatic defect cannot spontaneously heal. The persistent, unidirectional physiological pressure gradient—exacerbated by everyday activities that increase intra-abdominal pressure—gradually and inexorably forces the liver further through the right-sided defect. The fibrotic margins of the unhealed tear are slowly stretched, dilating the opening millimeter by millimeter.[8]

Over time, the protective tamponade effect of the liver fails. As the defect becomes sufficiently large, the liver undergoes massive herniation into the right hemithorax, creating a condition known as hepatothorax. This migration inevitably pulls other mobile intra-abdominal structures upward along the path of least resistance. The transverse colon, the gallbladder, and large volumes of omentum are subsequently drawn through the dilated defect and into the thoracic cavity. This delayed visceral herniation triggers a cascade of potentially fatal physiological disruptions. Within the rigid confines of the rib cage, the herniated abdominal mass exerts severe compressive forces on the adjacent pulmonary parenchyma. This leads to chronic compression atelectasis, a profound reduction in functional residual capacity, and severe ventilation-perfusion mismatching, ultimately manifesting as progressive, debilitating dyspnea. Concurrently, the gastrointestinal structures that have migrated into the

thorax face an even deadlier threat. The hollow loops of the bowel must traverse the narrow, unyielding fibrotic ring of the diaphragmatic defect. This anatomical chokepoint puts the herniated viscera at an exceptionally high risk for incarceration. The venous and lymphatic drainage of the bowel is easily compromised by the tight muscular ring, leading to rapid tissue edema. This swelling further constricts the arterial blood supply, initiating a vicious cycle of ischemia, necrosis, and bowel perforation. If the condition progresses to this obstructive and strangulating phase, the patient faces catastrophic physiological collapse, with mortality rates rising steeply.[9]

While the acute presentation of traumatic diaphragmatic rupture is thoroughly documented within the surgical literature, reports detailing the intricate pathophysiological progression of right-sided ruptures spanning a latency period of a full decade are exceedingly rare. Such cases present a unique opportunity to observe the extreme limits of human physiological adaptation and the long-term consequences of trans-diaphragmatic pressure dynamics. Understanding this insidious chronicity is paramount for any clinician managing patients with a remote history of major trauma.[10] Therefore, the aim of this study is to present a highly unusual case of a right-sided TDR diagnosed ten years post-injury, presenting with massive hepatothorax and multi-organ herniation. The novelty of this manuscript lies in its comprehensive exploration of the long-term pathophysiological pressure dynamics that facilitate delayed herniation, providing a critical framework for trauma surgeons and clinicians evaluating patients with chronic respiratory symptoms and a remote history of blunt trauma.

2. Case Presentation

In accordance with the highest standards of medical ethics and the principles established in the Declaration of Helsinki, written informed consent was obtained directly from the patient for the publication of this case report and any accompanying radiological or intraoperative images. The patient was comprehensively counseled regarding the nature of the publication, understanding that her clinical history, diagnostic findings, and surgical outcomes would be disseminated within the scientific literature for educational and academic purposes. Strict confidentiality protocols were maintained throughout the preparation of this manuscript to protect the patient's identity. All identifiable markers have been thoroughly anonymized or omitted. The patient was explicitly informed that while her anonymity is prioritized, absolute anonymity cannot be completely guaranteed in medical literature. She voluntarily agreed to these terms without coercion or financial incentive. Furthermore, formal ethical approval was waived by the local Institutional Review Board, as is standard protocol for retrospective, anonymized, single-patient reports that do not involve experimental interventions or deviations from established standards of care.

A 29-year-old female presented to the outpatient pulmonary clinic complaining of progressive dyspnea on exertion, which had insidiously worsened over the past six months (Table 1a). She also reported intermittent, non-specific right upper quadrant abdominal fullness and early satiety. A detailed review of her past medical history revealed a severe motor vehicle collision ten years prior. Following that accident, she was evaluated at an outside peripheral hospital. Medical records from that admission documented blunt thoracoabdominal trauma resulting in a right hemothorax and pulmonary contusion. Her initial chest radiograph at the time confirmed the hemothorax but showed no apparent elevation of the right hemidiaphragm or overt signs of visceral herniation. She was treated with the placement of a right-sided tube thoracostomy, which drained approximately 200 cc of hemorrhagic fluid. The underlying diaphragmatic defect was not identified during this initial hospitalization. Upon current physical examination, the patient was tachypneic with a respiratory rate of 24 breaths per minute. Auscultation of the chest revealed

significantly diminished breath sounds over the right middle and lower lung fields, accompanied by dullness to percussion. Notably, faint bowel sounds were appreciated upon auscultation of the right lower thorax.

Table 1a. Summary of Clinical Findings on Admission (Patient History & Physical Examination)

Clinical Domain	Specific Finding / Value	Clinical Interpretation
PATIENT HISTORY & DEMOGRAPHICS		
Age & Gender	29 years, Female	Demographic baseline
Chief Complaint	Progressive dyspnea on exertion over the past 6 months	Respiratory compromise
Associated Symptoms	Intermittent right upper quadrant abdominal fullness, early satiety	Gastrointestinal compression
Mechanism of Injury	Severe motor vehicle collision 10 years prior; initially treated with right tube thoracostomy	Delayed presentation of high-energy blunt trauma
PHYSICAL EXAMINATION		
Vital Signs	Respiratory Rate: **24 breaths/min**	Tachypnea
Pulmonary Auscultation	Diminished breath sounds over the right middle and lower lung fields	Loss of right lung volume
Thoracic Percussion	Dullness to percussion over the right lower thorax	Presence of solid organs/fluid in pleural space
Thoracic Auscultation (Pathognomonic)	Faint bowel sounds appreciated in the right lower thorax	Intrathoracic visceral herniation

To provide a comprehensive clinical picture, extensive laboratory workup and arterial blood gas (ABG) analyses were conducted prior to surgical intervention. The results demonstrated mild chronic hypoxemia and slight hepatic enzyme elevation, consistent with chronic hepatic compression and atelectasis (Table 1b). A standing posterior-anterior chest radiograph revealed a significantly elevated right hemidiaphragm with opacification of the right lower lung zone (Figure 1A). To delineate the anatomy, a contrast-enhanced thoracoabdominal computed tomography (CT) scan was performed. The axial, coronal, and sagittal reconstructions demonstrated a massive defect in the right hemidiaphragm (Figure 1B-D). Through this defect, there was extensive herniation of the right hepatic lobe (hepatothorax), the intact gallbladder, loops of the transverse colon, and a large volume of omentum into the right thoracic cavity. The CT clearly displayed the classic collar sign—a waist-like constriction of the herniated viscera at the site of the diaphragmatic tear—as well as the dependent viscera sign, confirming the loss of posterior structural support.

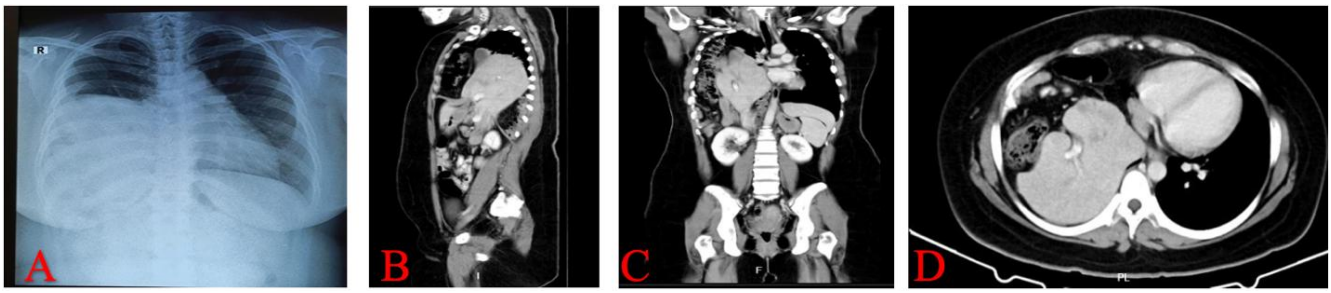

Figure 1. Radiograph and Thoracoabdominal CT Scan imaging. (A) Chest X-ray with elevated right hemidiaphragm; (B) Sagittal, (C) Coronal, and (D) Axial of thoracoabdominal CT scan show hepatothorax, with herniated transverse colon and omentum to the thoracic cavity.

Table 1b. Summary of Clinical Findings on Admission (Laboratory & Radiographic Imaging)

Clinical Domain	Specific Finding / Value	Clinical Interpretation
LABORATORY & BLOOD GAS ANALYSIS		
Hemoglobin (Hb)	11.2 g/dL (Ref: 12.0 - 15.5 g/dL)	Mild anemia
Alanine Aminotransferase (ALT)	68 U/L (Ref: 7 - 56 U/L)	Mild hepatic compression
Aspartate Aminotransferase (AST)	55 U/L (Ref: 10 - 40 U/L)	Mild hepatic compression
Arterial pH	7.38 (Ref: 7.35 - 7.45)	Normal acid-base balance
PaO2 (Room Air)	72 mmHg (Ref: 75 - 100 mmHg)	Mild chronic hypoxemia
PaCO2	42 mmHg (Ref: 35 - 45 mmHg)	Adequate ventilation clearance
RADIOGRAPHIC IMAGING (CT-SCAN & X-RAY)		
Chest Radiograph (PA View)	Significantly elevated right hemidiaphragm with opacification of the right lower lung zone	Suspicion of diaphragmatic eventration or rupture
Thoracoabdominal CT-scan	10 cm x 4 cm posterolateral defect in right hemidiaphragm; herniation of right hepatic lobe, intact gallbladder, transverse colon, and omentum	Massive multiorgan hepatothorax
Specific Radiological Signs	Positive "collar sign" and positive "dependent viscera sign"	Confirmatory for diaphragmatic rupture

Given the risk of visceral incarceration and worsening respiratory compromise, elective surgical intervention was scheduled (Table 2). An abdomino-thoracotomy approach was selected to provide adequate exposure for both visceral reduction and intrathoracic adhesiolysis. Intraoperatively, dense, chronic fibrotic adhesions were encountered between the herniated liver, the transverse colon, and the right pulmonary pleura. Following detailed adhesiolysis, the herniated viscera were safely reduced back into the peritoneal cavity. The diaphragmatic defect was identified posterolaterally, measuring approximately 10 cm in length and 4 cm in width, with thickened, fibrotic margins indicative of chronic injury. A primary tension-free repair was initially performed using interrupted, non-absorbable polypropylene sutures. Due to the substantial size of the defect and the chronic retraction of the surrounding diaphragmatic muscle, the primary repair was reinforced with a non-absorbable prosthetic mesh (dual-mesh) anchored with secure tacks and sutures to bridge and strengthen the remaining weakened musculature.

The patient's postoperative course was remarkably uneventful. A chest radiograph obtained on postoperative day two demonstrated a normalized contour of the right hemidiaphragm and complete re-expansion of the right lung. She tolerated diet advancement well, experienced a return of normal bowel function, and was discharged on postoperative day seven. Outpatient follow-up at three and six months revealed complete resolution of her dyspnea and no radiographic evidence of hernia recurrence.

3. Discussion

Traumatic diaphragmatic rupture stands as a definitive and reliable physiological marker of severe, high-kinetic polytrauma. Historically, patients presenting with this profound injury profile demonstrate markedly elevated Injury Severity Scores, reflecting the massive transfer of mechanical energy required to violently disrupt the thick musculotendinous continuity of the human diaphragm. In the acute clinical setting immediately following a traumatic event, the patient's physiological trajectory is predominantly dictated by concurrent, catastrophic organ damage.[11] The presence of profound intra-abdominal hemorrhage, extensive pelvic ring fractures, and devastating traumatic neurologic injuries typically drives early mortality rates, which range from 13.2 percent to 21 percent. In these acute scenarios, the diaphragmatic defect itself is rarely the primary cause of immediate death.

TABLE 2. SUMMARY OF DIAGNOSIS, TREATMENT, FOLLOW-UP, AND OUTCOME

Clinical Phase	Specific Parameters	Detailed Description & Findings
1. DEFINITIVE DIAGNOSIS		
Preoperative Assessment	Primary Diagnosis	Delayed right-sided Traumatic Diaphragmatic Rupture (TDR) with massive multiorgan hepatothorax.
	Anatomical Defect	• Location: Posterolateral aspect of the right hemidiaphragm. • Dimensions: 10 cm in length x 4 cm in width. • Herniated Contents: Right hepatic lobe, intact gallbladder, transverse colon, and large volume of omentum.
2. SURGICAL INTERVENTION		
Operative Management	Surgical Approach	**Open Abdomino-thoracotomy** chosen to provide optimal dual-cavity exposure for safe organ mobilization.
	Intraoperative Procedures	• Extensive adhesiolysis of dense, chronic fibrotic attachments between herniated liver, transverse colon, and right pulmonary pleura. • Safe reduction of all herniated viscera back into the peritoneal cavity under direct vision.
	Defect Repair Technique	• Primary tension-free closure utilizing interrupted non-absorbable polypropylene sutures. • **Mesh Augmentation:** Reinforcement with a non-absorbable prosthetic dual-mesh to bridge the weakened musculature, anchored securely with tacks and sutures.
3. INPATIENT & OUTPATIENT FOLLOW-UP		
Postoperative Monitoring	Inpatient Course	• Postoperative Day 1 & 2: Chest radiographs confirmed normalized right hemidiaphragm contour and complete right lung re-expansion. • Patient tolerated diet advancement and experienced return of normal bowel function. • Discharged home on **Postoperative Day 7**.
	Outpatient Surveillance	Routine clinical evaluations conducted at 3 months and 6 months post-discharge.
4. FINAL CLINICAL OUTCOME		
Overall Prognosis	Patient Status	• **Complete resolution of progressive dyspnea and early satiety.** • Excellent respiratory mechanics restored. • No clinical or radiographic evidence of hernia recurrence at the 6-month follow-up interval.

However, when the initial diaphragmatic tear escapes early diagnostic detection due to the overshadowing severity of these associated injuries, the patient unknowingly transitions into the chronic phase of a missed rupture. This delayed presentation introduces a profoundly distinct and complex set of physiological, anatomical, and diagnostic challenges that demand rigorous clinical analysis. The central pathophysiological mechanism orchestrating the delayed presentation of traumatic diaphragmatic rupture is fundamentally driven by intrinsic trans-diaphragmatic pressure gradients (Figure 2). To understand this progression, one must examine the baseline respiratory mechanics of the human body. Under normal physiological conditions, the respiratory cycle creates a perpetual and dynamic pressure differential, wherein the intra-abdominal pressure consistently exceeds the intra-thoracic pressure by approximately 7 to 20 centimeters of water. This physiological baseline gradient is not a static measurement; it undergoes drastic magnification during routine activities requiring Valsalva maneuvers, forceful coughing, straining, or the heavy lifting of objects.[12]

When a traumatic defect occurs, particularly on the right side of the diaphragm, the immediate post-injury phase frequently does not feature the overt herniation of abdominal contents. This is largely due to regional anatomy.[13] The massive, dense right lobe of the liver acts as a formidable anatomical barrier against the right hemidiaphragm. Following the sudden musculotendinous rupture, the natural negative intra-thoracic pressure immediately draws the superior surface of the liver upward against the defect. The hepatic parenchyma temporarily plugs the tear, effectively acting as a biological seal that prevents the immediate upward migration of more mobile, air-filled hollow viscera. Consequently, the natural history of a missed rupture enters a latent or interval phase, often deceiving initial clinical evaluations.

Figure 2. The pathophysiology of delayed presentation.

This insidious latency period can persist for weeks, months, or, as vividly demonstrated in this exceptional case, span an entire decade. Throughout this extended chronological timeline, the unyielding, unidirectional trans-diaphragmatic pressure gradient exerts continuous mechanical stress on the fibrotic margins of the initial defect. Because the diaphragm is in constant rhythmic motion, the injured muscle

fibers cannot spontaneously heal. Instead, the relentless positive abdominal pressure gradually forces the hepatic mass further upward into the thoracic cavity, progressively stretching and dilating the muscular tear. Over a ten-year span, this slow biomechanical process expanded the defect to a substantial 10 centimeters by 4 centimeters. This significant dilation ultimately created a wide, permissive anatomical pathway for the subsequent migration of less structurally fixed organs, specifically allowing the transverse colon, the intact gallbladder, and large volumes of omentum to be drawn upward.[14]

Once definitive multiorgan visceral herniation transpires, the delicate equilibrium of negative intra-thoracic pressure is completely compromised.[15] The sheer intrathoracic volume now occupied by the herniated abdominal mass exerts direct, severe compressive forces on the adjacent right pulmonary parenchyma. This anatomical displacement leads directly to progressive compression atelectasis, a profound reduction in functional vital capacity, and severe ventilation-perfusion mismatching within the right lung architecture. Over time, the chronic hypoxic state can induce localized pulmonary vasoconstriction and increase right ventricular strain. This complex cardiopulmonary cascade directly accounts for the patient's progressive dyspnea on exertion and the chronic hypoxemia observed during the initial clinical evaluation.

Furthermore, the delayed herniation of hollow viscera introduces a critical, life-threatening threat to the gastrointestinal system. As the transverse colon and omentum pass through the rigid, fibrotic ring of the chronic diaphragmatic defect, the anatomical chokepoint renders them highly susceptible to twisting, kinking, and structural entrapment. This mechanical constriction initially limits the low-pressure venous return of the bowel wall, leading to rapid tissue edema and third-spacing of fluids. As the bowel wall swells within the confined space of the defect, the higher-pressure arterial supply is subsequently compromised. This vascular occlusion predisposes the patient to a rapid sequence of catastrophic complications, including profound bowel ischemia, complete mechanical obstruction, and subsequent transmural perforation. If the herniation progresses from simple displacement to acute incarceration and strangulation, the ensuing systemic toxicity, sepsis, and physiological shock can drive mortality rates to approach 30 percent, fiercely underscoring the lethal potential of this delayed pathology.[16]

This case profoundly underscores the clinical reality that right-sided traumatic diaphragmatic rupture represents an exceptional and highly evasive diagnostic challenge. The clinical presentation is frequently subacute, presenting with vague symptoms that fail to point directly to a specific anatomical defect. Initial radiographic indicators on standard posterior-anterior plain chest films are notoriously ambiguous and lack spatial resolution.[17] The resulting images can easily mimic the appearance of mere pleural effusions, right lower lobe atelectasis, elevated hemidiaphragm from phrenic nerve palsy, or simple diaphragmatic eventration. Furthermore, relying purely on physical examination is inherently flawed. The classical pathognomonic sign originally described by Bowditch—the distinct presence of bowel sounds clearly audible upon auscultation of the thoracic cavity—is manifested in only a small minority of patients. This renders isolated clinical examination an unreliable and wholly insufficient diagnostic tool for ruling out a rupture.

Consequently, high-resolution multi-detector CT-scan imaging, specifically utilizing coronal and sagittal multiplanar reconstructions, serves as the absolute gold standard for establishing a definitive diagnosis. Achieving a high degree of diagnostic accuracy requires the careful identification of subtle yet highly specific radiological markers. The identification of the collar sign—a waist-like constriction of the herniated viscera at the precise anatomical site of the diaphragmatic tear—and the dependent viscera sign are absolutely imperative for confirming the loss of diaphragmatic continuity. Specifically for right-sided defects, recognizing a distinctive hump deformity of

the liver as it herniates upward through the muscular defect serves as the radiological equivalent of the collar sign and is highly specific for confirming the diagnosis prior to any surgical intervention.[18]

Addressing a delayed traumatic diaphragmatic rupture surgically introduces anatomical and technical complexities that far exceed those encountered during immediate acute repairs. The primary operative hurdle is the inevitable formation of dense, chronic intrathoracic adhesions that rigidly bind the herniated abdominal organs to the delicate pleura, the pericardium, and the pulmonary parenchyma.[19] While minimally invasive laparoscopic or thoracoscopic surgical techniques are increasingly gaining traction for certain uncomplicated presentations, the presence of a massive, multi-organ hepatothorax heavily bound by ten years of fibrotic scar tissue definitively mandated an open abdomino-thoracotomy approach in our case.

This extensive dual-cavity operative approach is critical for ensuring patient safety. It provides unparalleled spatial exposure, allowing for the precise, deliberate mobilization of the liver and fragile bowel segments under direct, unimpeded vision. This vastly minimizes the perilous risk of inducing iatrogenic hepatic hemorrhage, biliary tract injury, or transmural bowel perforation during the arduous process of separating highly vascularized adhesions. Following the successful reduction of the viscera back into the peritoneal cavity, managing the defect itself requires specialized reconstructive techniques. In chronic scenarios spanning a decade, the torn edges of the diaphragmatic muscle have severely retracted, atrophied, and become densely fibrotic. Attempting a simple primary closure utilizing only direct suturing would invariably place the compromised tissue under extreme physiological tension, practically guaranteeing structural failure and immediate recurrence upon the resumption of spontaneous breathing. Therefore, the primary repair must be robustly augmented with a non-absorbable prosthetic dual-mesh. This mesh acts to bridge the substantial gap, distributing the mechanical load of respiration evenly and firmly anchoring the repair to surrounding healthy chest wall structures to accommodate the permanent anatomical distortion.

The primary limitation inherent in this study is its foundational structure as a single retrospective case report. This specific epidemiological framework inherently limits the broad statistical generalizability of the chosen surgical approach and the observed pathophysiological timeline to the wider, highly heterogeneous trauma population. Additionally, while the mid-term follow-up period conclusively demonstrated an excellent initial recovery and a complete return to baseline pulmonary function, extensive long-term surveillance remains absolutely necessary. Extended clinical and radiographic observation is crucial to accurately evaluate the enduring structural durability of the prosthetic mesh repair over subsequent decades and to definitively monitor against the insidious threat of late-stage hernia recurrence.[20] Moving forward, the scientific and surgical communities should direct future research efforts toward the establishment and utilization of multi-center, prospective trauma registries. These comprehensive international databases are essential for identifying predictive clinical biomarkers of delayed herniation and for rigorously evaluating the long-term efficacy, morbidity profiles, and safety margins of minimally invasive surgical approaches versus traditional open mesh repairs in patients presenting with chronic, large-scale diaphragmatic defects.

4. Conclusion

Traumatic diaphragmatic rupture, particularly when involving right-sided injuries that culminate in massive hepatothorax, represents a profound and enduring diagnostic dilemma that necessitates lifelong clinical vigilance. Because the immense, solid mass of the liver can temporarily occlude the traumatic defect immediately following the impact, gradual multi-organ herniation can unfold entirely silently. This insidious biomechanical process often manifests months or, as highlighted here, even years later, bringing with it

potentially fatal respiratory and gastrointestinal consequences that can rapidly devolve into systemic shock. This specific case explicitly and powerfully demonstrates that a documented clinical history of severe blunt thoracoabdominal trauma must never be entirely discounted by treating physicians, regardless of the sheer volume of time that has elapsed since the initial traumatic event occurred.

A remarkably high index of suspicion must be persistently maintained for any patient presenting with unexplained, progressive respiratory distress, subacute abdominal complaints, and a remote history of significant kinetic trauma. Achieving a timely diagnosis through advanced multi-planar imaging with a CT-scan, followed immediately by precise, highly tailored surgical intervention based on the chronicity and physical dimensions of the defect, is the only definitive pathway to halt the progression of the disease. Addressing the anatomical failure completely and securely prevents catastrophic visceral strangulation, restores baseline respiratory mechanics, and ultimately yields excellent, durable long-term outcomes for the patient.

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