

Successful non-operative management of acute incarcerated lung herniation after video- assisted thoracoscopic surgery in a sarcopenic patient: a case report

Geng Chen

Shenzhen University of Advanced Technology General Hospital

Yunxi Zhang

Shenzhen University of Advanced Technology General Hospital

Xuanqin Wu

Shenzhen University of Advanced Technology General Hospital

Weimin Luo

weiminluo120@163.com

Shenzhen University of Advanced Technology General Hospital

Case Report

Keywords: Lung hernia, Video-assisted thoracoscopic surgery (VATS), Thoracic sarcopenia, Non-operative management, Intercostal collar effect, Pneumothorax, Case report

Posted Date: October 4th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11028118/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.
[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Background

Port-site intercostal lung hernia (ILH) is an exceedingly rare complication following video-assisted thoracoscopic surgery (VATS), historically managed via urgent surgical reconstruction. The predisposing role of subclinical thoracic sarcopenia, the biomechanical limitations of passive lung reduction, and the feasibility of structured conservative management for acute incarcerated ILH remain poorly characterized.

Case Presentation

A 71-year-old Asian male with preoperatively documented thoracic muscle weakness and subclinical sarcopenia underwent uneventful VATS combined segmentectomy. Five hours following routine chest tube removal on postoperative day 4, a violent paroxysm of coughing triggered an acute intercostal lung hernia with extensive subcutaneous emphysema. An intentional atmospheric pneumothorax maneuver aimed at inducing passive lung retraction paradoxically failed due to an acute "collar effect"—a constricting ring formed by rapid parenchymal edema and reactive intercostal spasm. In the absence of parenchymal ischemia or strangulation, urgent re-exploration was avoided in favor of a structured non-operative protocol: focal bolster counter-pressure, continuous low-pressure pleural water-seal venting, aggressive intrathoracic pressure suppression, and serial dynamic computed tomography (CT) tracking. The incarcerated parenchyma progressively decompressed, achieving complete spontaneous reduction by postoperative day 12 and full anatomical restoration by postoperative day 16 without recurrence.

Conclusions

Subclinical thoracic sarcopenia and post-drain removal cough dynamics can precipitate port-site lung herniation. Incarcerated lung tissue may resist passive pneumothorax collapse due to the intercostal collar effect. A structured, pressure-regulated conservative regimen provides a safe, lung-preserving alternative to redo surgery in carefully selected patients without strangulation.

Background

Intercostal lung hernia (ILH), defined as the protrusion of pulmonary parenchyma beyond the confines of the musculoskeletal thoracic cage through an abnormal defect, is an uncommon clinical entity first classified by Morel-Lavallée in 1847[1]. While the majority of acquired lung hernias stem from high-energy blunt or penetrating thoracic trauma and conventional open thoracotomies, port-site lung herniation following video-assisted thoracoscopic surgery (VATS) is exceedingly rare, primarily due to the minimal access incisions and the avoidance of mechanical rib spreading.

The clinical presentation of post-VATS lung hernia can range from an asymptomatic reducible bulging to acute, life-threatening incarceration and strangulation. Historically, surgical exploration with direct suture closure or prosthetic mesh reinforcement has been regarded as the definitive treatment of choice to prevent parenchymal ischemia. However, in elderly patients with significant comorbidities, re-exploration poses substantial secondary surgical risks. The role and efficacy of structured conservative management for acute incarcerated ILH without strangulation remain poorly characterized, and the precise mechanical interactions governing non-operative reduction are seldom documented[2]. Furthermore, the role of subclinical thoracic sarcopenia[3] and peri-removal cough dynamics as predisposing triggers is frequently under-recognized[4].

Herein, we report an unusual case of acute, incarcerated 4th intercostal space lung hernia occurring hours after chest tube removal following VATS combined segmentectomy in a 71-year-old male with documented thoracic muscular sarcopenia. We detail the paradoxical failure of an intentional atmospheric pneumothorax maneuver to achieve passive reduction due to an intercostal "collar effect," followed by the complete anatomical resolution achieved via a structured non-operative compression and pressure-regulation protocol, chronologically verified by serial dynamic computed tomography (CT). This case report has been reported in accordance with the CARE (CAsE REport) guidelines.

Case Presentation

Patient History and Preoperative Evaluation

A 71-year-old retired Asian male (height: 167 cm; weight: 73 kg; body mass index: 26.18 kg/m²) presented with an asymptomatic solitary pulmonary nodule in the left upper lobe that had been detected six months prior, along with a one-week history of productive cough with purulent sputum and pleuritic chest pain. The patient had a 40 pack-year smoking history and had ceased smoking one month prior to admission. His medical history was notable for a remote inguinal hernia repair and baseline right pulmonary emphysema complicated by bullae. Physical examination revealed generalized soft-tissue laxity and thoracic wall sarcopenia, characterized by prominent redundant skin folds in bilateral axillary regions (subsequently documented photographically during the postoperative period to reflect baseline phenotype, Fig. 1A).

Outpatient unenhanced chest CT on August 8, 2026, demonstrated an irregular, enlarging cavitary nodule in the apicoposterior segment of the left upper lobe (LS1 + 2), suspicious for primary bronchogenic carcinoma. Arterial blood gas analysis on ambient air revealed a pH of 7.446, arterial partial pressure of carbon dioxide (PaCO₂) of 37.5 mmHg, arterial oxygen saturation (SaO₂) of 94.9%, and mild baseline hypoxemia with an arterial partial pressure of oxygen (PaO₂) of 66.7 mmHg (reference: 83.0–108.0 mmHg). The alveolar-arterial oxygen gradient [P(A-a)O₂] was widened to 33.4 mmHg (reference: 5.0–15.0 mmHg), consistent with physiological ventilation-perfusion mismatch secondary to underlying emphysema.

Pulmonary function tests showed preserved basic spirometry: forced vital capacity (FVC) was 2.96 L (83.38% of predicted), forced expiratory volume in one second (FEV₁) was 2.29 L (84.81% of predicted), and the FEV₁/FVC ratio was 77.36% (99.59% of predicted). However, specialized respiratory muscle evaluation revealed a disproportionate reduction in peak expiratory flow (PEF: 3.65 L/s, 45.01% of predicted; Z-score: -2.67), alongside decreased maximal inspiratory pressure (MIP: 63.00 cmH₂O, 66.85% of predicted) and maximal expiratory pressure (MEP: 69.75 cmH₂O, 79.80% of predicted)[5]. These findings objectively documented subclinical chest wall muscular weakness and sarcopenia. Following pulmonary prehabilitation with inhaled budesonide/formoterol and oral bromhexine, three-dimensional CT bronchovascular reconstruction was performed for preoperative surgical planning.

Surgical Intervention and Early Recovery (Postoperative Days 0–3)

On August 17, 2026, postoperative day (POD) 0, the patient underwent VATS anatomical combined segmentectomy (left S1 + 2c and S6, necessitated by tumor proximity across the interlobar fissure), accompanied by thoracic adhesiolysis and intercostal nerve blocks under general anesthesia with double-lumen endotracheal intubation. A three-port configuration was utilized: a 1.0-cm observation port at the 8th intercostal space (ICS) along the midaxillary line, a 3.0-cm utility incision at the 4th ICS along the anterior-to-midaxillary line, and an auxiliary port at the 7th ICS along the posterior axillary line. A soft-tissue wound protector was deployed without mechanical rib spreading. The utility incision was closed anatomically: the intercostal muscle layer was approximated with interrupted absorbable sutures, followed by standard closure of the fascia and skin. A 24-French chest tube was positioned via the inferior port and attached to a closed drainage system. Postoperative multimodal analgesia comprised patient-controlled intravenous dezocine (a synthetic opioid partial agonist), intravenous parecoxib (20 mg twice daily), and oral gabapentin (0.1 g three times daily), supplemented by mechanical expectoration and sequential pneumatic compression. Postoperative histopathology confirmed invasive lung adenocarcinoma with direct invasion into the adjacent lobe parenchyma across the fissure (pT3N0M0, Stage IIB).

A routine postoperative chest CT on August 18 (POD 1) confirmed complete resection of the cavitory lesion with expected postoperative changes, dependent bibasilar atelectasis, and minimal left hydropneumothorax, with an intact thoracic wall contour (Fig. 2A). Transient hypoalbuminemia was corrected with intravenous human albumin (10 g of 20% solution) and furosemide (20 mg). By August 20 (POD 3), 24-hour thoracic drainage had decreased to 140 mL with no air leaks.

Acute Pulmonary Herniation and Therapeutic Interventions (Postoperative Day 4)

The left chest tube was routinely removed at approximately 15:00. Approximately five hours following drain removal (at ~ 20:00 on POD 4), triggered by a bout of vigorous paroxysmal coughing, the patient experienced sudden left-sided incision pain, chest tightness, and a dynamic protruding mass over the

4th ICS. Palpation revealed an expansile cough impulse and extensive subcutaneous crepitus extending across the chest wall to the neck.

Emergency unenhanced chest CT performed at 20:40 confirmed an acute intercostal lung hernia: a portion of the left upper pulmonary parenchyma had extruded through the 4th ICS utility incision defect. The hernia neck measured 2.83 cm in transverse diameter, accompanied by marked subcutaneous emphysema dissecting the cervical and chest wall fascia, as well as progressive hydropneumothorax (Fig. 2B). The acute pain and respiratory distress provoked a hypertensive surge (blood pressure peaking at 180/105 mmHg), which was successfully managed with intravenous urapidil, oral nifedipine, and metoprolol.

Initial external manual reduction combined with simple compressive dressings was unsuccessful due to marked local soft-tissue tension and parenchymal engorgement. At approximately 22:20 on POD 4, a bedside tube thoracostomy was performed under local anesthesia using a pigtail catheter connected to a water-seal drainage system. To abolish the positive outward transpulmonary pressure gradient driving the herniation, an intentional, controlled atmospheric pneumothorax was introduced by temporarily venting the catheter to ambient air. This maneuver aimed to induce passive collapse of the ipsilateral lung parenchyma, thereby releasing mechanical tension and facilitating spontaneous retraction of the extruded tissue back into the pleural space. However, this physiological reduction strategy failed: rapid parenchymal edema and reactive intercostal muscular spasm formed a rigid constricting ring (an incarcerated "collar effect"), firmly entrapping the lung segment within the 4th intercostal defect.

Given the absence of strangulation, color changes, or parenchymal infarction, urgent surgical re-exploration was deferred in favor of a structured, non-operative compression protocol:

1. Chest Wall Immobilization: A thick, sterile gauze bolster was contoured directly over the 4th intercostal defect, over which an adjustable elastic thoracic binder was applied to maintain constant apposition against the chest wall without restricting diaphragmatic excursion (Fig. 1B).
2. Continuous Pleural Venting: The re-inserted chest drain was maintained on continuous water seal to evacuate pleural gas and prevent intrapleural positive-pressure spikes during coughing.
3. Control of Intrathoracic Pressure: Continuous intravenous dezocine infusions were reinstated for cough and pain suppression, combined with inhaled bronchodilators, oral bromhexine, and oral lactulose (30 mL) to avoid Valsalva-induced pressure surges during bowel movements. Blood pressure was maintained within normal limits using oral amlodipine (5 mg once daily).

Dynamic Radiologic Evolution and Clinical Resolution (Postoperative Days 5–16)

Chest CT on August 22 (POD 5) showed that the pulmonary hernia remained stable without signs of ischemia or strangulation, while soft-tissue emphysema was partially absorbed (Fig. 3A). Laboratory evaluation on August 24 (POD 7) revealed a normal white blood cell count ($7.05 \times 10^9/\text{L}$) with 76.6%

neutrophils, persistent lymphopenia ($0.80 \times 10^9/L$), and mild anemia (red blood cell count: $4.02 \times 10^{12}/L$; hemoglobin: 128 g/L). Elevated levels of fibrinogen (6.10 g/L; reference: 2.0–4.0 g/L) and D-dimer (1.58 $\mu\text{g/mL}$; reference: 0.00–0.55 $\mu\text{g/mL}$) were noted. Serum chemistry indicated persistent hypoalbuminemia (total protein: 56.9 g/L; albumin: 35.3 g/L), mild hypokalemia (3.42 mmol/L), and mild transaminase elevation (ALT: 71.8 U/L; AST: 66.5 U/L) with normal renal function. Repeat CT on POD 7 demonstrated partial retraction of the herniated lung, further regression of subcutaneous emphysema, and residual pneumothorax with approximately 15% lung collapse (Fig. 3B).

Follow-up CT on August 28 (POD 11) showed dynamic fluctuation of the hernia sac during coughing, while subcutaneous emphysema had nearly resolved and pneumothorax remained minimal (Fig. 3C). The chest tube was clamped on August 28 for trial observation. On August 29 (POD 12), follow-up CT confirmed complete spontaneous reduction of the pulmonary parenchyma back into the hemithorax, with restored intercostal integrity and absence of pneumothorax recurrence after clamping (Fig. 3D). The chest catheter was definitively removed later that morning.

A pre-discharge CT on September 2 (POD 16) verified complete restoration of the chest wall architecture, absence of residual herniation, full re-expansion of the left lung, and stable minor bilateral pleural effusions (Fig. 3E). Concurrently, laboratory indices improved (white blood cells: $6.28 \times 10^9/L$; hemoglobin: 132 g/L; serum potassium: 3.76 mmol/L; albumin: 37.8 g/L; ALT: 41.8 U/L). The patient was discharged on POD 16 in good clinical condition, with complete resolution of chest wall pain, absence of an expansile cough impulse, and normal respiratory function. Maintenance medications upon discharge included oral amlodipine (5 mg once daily), loxoprofen (an oral non-steroidal anti-inflammatory drug), and bromhexine. Comprehensive discharge counseling was provided, emphasizing strict avoidance of heavy lifting, Valsalva maneuvers, and vigorous coughing to prevent positive intrathoracic pressure spikes. During a telephone follow-up conducted one week post-discharge, the patient reported being fully ambulatory and asymptomatic, with no recurrence of chest tightness, incisional pain, or palpable bulging at the surgical site. Given his underlying pulmonary emphysema and thoracic wall sarcopenia, a scheduled outpatient review with a repeat low-dose chest CT was planned at 1 and 3 months post-discharge to confirm durable chest wall integrity and monitor for late intercostal dehiscence.

From the patient's perspective, he expressed great satisfaction with avoiding a secondary surgical intervention under general anesthesia, noting that the external compression binder provided immediate comfort and physical reassurance without causing significant breathing restriction.

Discussion

Epidemiology and Port-Site Vulnerability in VATS

ILH, initially classified by Morel-Lavallée in 1847 into congenital and acquired subtypes, represents an uncommon mechanical disruption wherein lung tissue protrudes through an abnormal chest wall defect. More than 80% of acquired ILHs historically arise secondary to high-energy blunt/penetrating thoracic

trauma or conventional posterolateral thoracotomies involving aggressive rib spreading and intercostal nerve/muscle devascularization[1]. In the era of minimally invasive thoracic surgery, port-site lung herniation following VATS remains extraordinarily rare, largely because small access incisions (< 3–4 cm) and the avoidance of mechanical rib retractors preserve structural chest wall rigidity.

Anatomically, the fourth intercostal space along the anterior-to-midaxillary line exhibits inherent structural vulnerability. In this topographic region, the thoracic cage is devoid of robust extrinsic muscular reinforcement: the pectoralis major terminates medially, the latissimus dorsi covers only the posterior thoracic margin, and the anterior reach of the serratus anterior provides relatively thin muscular coverage. Furthermore, the muscular fibers of the external intercostal muscle taper anteriorly and are replaced by the thinner, aponeurotic external intercostal membrane. During anatomical VATS segmentectomy, this fourth intercostal port serves as the primary utility incision. Repeated instrument articulation, torque, and specimen extraction through this port inevitably impose shear stresses and microtrauma on the intercostal tissue borders, rendering this interspace susceptible to acute dehiscence when subjected to subsequent mechanical stress[6].

The "Triple-Hit" Pathogenesis: Sarcopenia, Tissue Frailty, and Cough Dynamics

The development of acute post-VATS lung herniation in our patient can be conceptualized through a "triple-hit" pathophysiologic model combining systemic connective tissue vulnerability, localized sarcopenia, and acute biomechanical trigger forces:

Hit 1: Systemic Connective Tissue Attenuation. The patient exhibited profound axillary skin redundancy and subcutaneous soft-tissue atrophy, coupled with a remote history of inguinal hernia repair. Prior adult inguinal herniation frequently reflects altered systemic collagen turnover (e.g., decreased type I/III collagen ratio) and inherent fascia laxity[7, 8]. This systemic predisposition impaired tissue tensile strength and blunted the intrinsic healing capacity of the sutured intercostal fascia.

Hit 2: Thoracic Wall Sarcopenia and Respiratory Muscle Weakness. A distinctive feature of this case was the preoperatively documented discrepancy between preserved spirometric volumes (FVC and FEV₁ > 80%) and severely compromised respiratory muscle strength. The patient displayed marked reductions in MIP (66.85% of predicted), MEP (79.80% of predicted), and PEF (45.01% of predicted; Z-score: -2.67). Sarcopenia of the intercostal musculature drastically decreases tissue resistance to suture pull-through. When standard absorbable sutures are tied under tension against atrophic, friable muscle bundles, the suture loops act as "cheese wires," cutting through the sarcopenic muscular fibers rather than maintaining structural apposition[9].

Hit 3: The Acute Positive-Pressure Trigger. The temporal correlation between routine chest drain removal and violent paroxysmal coughing was decisive. During vigorous coughing, physiological studies demonstrate that sudden contraction of the diaphragm and abdominal wall against a closed glottis can

propel intrathoracic and intrapleural pressures up to 100–300 cmH₂O[4]. Following chest tube removal, the pleural space is completely sealed. When the patient suffered an acute paroxysm of coughing, this massive outward transpulmonary pressure gradient directly targeted the recently closed 4th intercostal defect, shearing through the weakened intercostal suture line and acutely extruding the lung parenchyma[10].

Paradoxical Failure of Passive Pneumothorax Reduction: The "Collar Effect"

In acute reducible lung herniation, external manual reduction is typically the first-line intervention. In our case, initial manual pressure failed due to severe localized soft-tissue tension. Theoretically, introducing an intentional, controlled atmospheric pneumothorax via a bedside thoracostomy catheter should abolish the positive transpulmonary outward driving force, inducing passive elastic recoil of the ipsilateral lung and facilitating spontaneous retraction back into the hemithorax.

However, this physiologic reduction maneuver unexpectedly failed, unveiling a mechanical entrapment phenomenon: the "incarcerated collar effect". Once the pulmonary parenchyma extruded through the relatively narrow, rigid 2.83-cm intercostal ring:

1. Venous and Lymphatic Outflow Obstruction: The rigid margins of the adjacent ribs and taut intercostal fascia constricted the base of the herniated lung segment. This triggered rapid capillary engorgement and acute interstitial parenchymal edema, generating an "hourglass" or "mushroom-shaped" configuration where the extrathoracic hernia sac expanded to a volume substantially larger than the rigid neck defect.
2. Reactive Intercostal Muscular Spasm: Acute nociceptive stimulation of the adjacent 4th intercostal neurovascular bundle provoked a persistent, involuntary spasm of the surrounding thoracic musculature, converting the intercostal space into an inelastic constricting ring.
3. Loss of Elastic Recoil Leverage: The edematous, turgid extrathoracic parenchyma lost its intrinsic compliance. Consequently, passive elastic collapse of the intrathoracic lung was insufficient to pull the swollen, locked extrathoracic mass back through the constricting ring.

This pathophysiologic mechanism highlights that in the setting of rapid parenchymal edema, inducing an artificial pneumothorax cannot overcome the mechanical entrapment of the collar effect, and clinicians should be cautious not to rely solely on lung collapse for acute reduction.

The "Four-Pillar" Non-Operative Protocol vs. Surgical Re-Exploration

The management of post-thoracotomy and post-VATS lung hernia remains contentious. Conventional surgical dogma generally favors urgent operative exploration—incorporating primary closure, pericostal approximation, or prosthetic mesh reconstruction—to prevent parenchymal ischemia and gangrene[6,

11]. However, in elderly patients presenting with baseline pulmonary emphysema, thoracic sarcopenia, and tissue frailty, secondary surgery under general anesthesia carries substantial morbidities, including rib devascularization, postoperative chronic pain, prosthesis infection, and prolonged mechanical ventilation.

Emergency surgery remains mandatory for strangulated hernias presenting with systemic sepsis, intractable hemoptysis, complete loss of parenchymal perfusion on contrast-enhanced imaging, or hemodynamic collapse. Conversely, when serial imaging confirms viable lung parenchyma without infarction and the patient remains clinically stable, non-operative management represents an appealing, lung-preserving alternative.

Rather than relying on simple watchful waiting, we devised a structured, physiologically targeted "Four-Pillar" Conservative Protocol:

1. Focal External Counter-Pressure: Applying a contoured bolster secured by an elastic thoracic binder creates sustained inward mechanical resistance. Biomechanically, this neutralizes the outward transmural pressure vector generated during spontaneous ventilation, physically restricting progressive herniation while preserving diaphragmatic excursion[12].
2. Dynamic Pleural Pressure Venting: Maintaining low-pressure water-seal drainage serves as a critical "pop-off" safety valve. During sudden spikes in intra-abdominal or pleural pressure, the water-seal system vents pleural gas, blunting the sudden barotrauma that would otherwise exert outward shear stress against the vulnerable intercostal defect.
3. Systemic Suppression of Intrathoracic Pressure Dynamics: The success of non-operative reduction relies heavily on blunting the drivers of intercostal tension. Aggressive multimodal cough suppression, adequate analgesia to break the vicious cycle of reactive intercostal spasm, and prophylactic measures against Valsalva-induced strain (e.g., scheduled stool softeners) effectively eliminate sudden positive-pressure surges across the healing chest wall.
4. Serial Quantitative Imaging Tracking: Close surveillance via low-dose dynamic CT is indispensable. It provides real-time verification of parenchymal perfusion, objectively monitors the resolution of interstitial edema within the hernia neck, and confirms durable anatomical reduction before chest tube removal.

Over the course of sustained pressure regulation, the parenchymal edema gradually abated, relieving the mechanical lock of the collar effect and permitting spontaneous retraction of the lung tissue without structural compromise.

Preventive Nuances and Clinical Takeaways

This case yields several critical preventive insights for thoracic surgeons performing VATS procedures in vulnerable cohorts:

Identification of Latent Sarcopenia: Normal spirometric values (FEV₁, FVC) can mask underlying chest wall sarcopenia. The routine integration of respiratory muscle parameters (PEF, MIP, MEP) or CT-derived

pectoralis/intercostal muscle cross-sectional area indices should be considered to identify patients with fragile chest wall integrity preoperatively[5, 13].

Reinforced Port Closure Technique: In patients with advanced age, emphysema, history of hernias, or low MEP, simple intercostal muscle approximation using fine, rapidly absorbable sutures is inadequate. We advocate the use of heavy, slowly absorbable (e.g., 0 or 1 – 0 polydioxanone) or non-absorbable braided sutures placed in a figure-of-eight or full-thickness pericostal configuration around adjacent ribs. This transfers mechanical stress directly to the bony rib cage rather than friable muscular tissue[14]. Rigorous approximation of the anterior serratus fascia must be prioritized to establish a second-layer anatomical barrier.

Peri-Drain Removal Pressure Prophylaxis: Chest tube removal represents a high-risk transition point. Preemptive antitussive administration, continuous analgesia, and patient coaching to avoid post-drain removal coughing paroxysms or straining are essential maneuvers to safeguard the fresh intercostal closure line against sudden barotrauma.

Conclusions

Port-site intercostal lung herniation following VATS is an elusive but severe mechanical complication, predominantly driven by the convergence of subclinical chest wall sarcopenia, connective tissue frailty, and acute post-drain removal coughing paroxysms. Clinicians must recognize that incarcerated lung tissue can resist passive collapse during induced pneumothorax due to the intercostal "collar effect"—characterized by parenchymal edema and reactive muscular spasm.

In carefully selected, hemodynamically stable patients without evidence of strangulation or ischemia, a structured "four-pillar" conservative regimen combining localized bolster compression, pleural water-seal venting, strict intrathoracic pressure suppression, and serial CT monitoring provides a safe, effective, and lung-preserving alternative to urgent redo surgery. For high-risk patients with documented respiratory muscle weakness, preoperative sarcopenia screening and tension-free, pericostal port closure techniques are strongly recommended to prevent this acute complication.

Abbreviations

VATS

Video-assisted thoracoscopic surgery

ILH

Intercostal lung hernia

ICS

Intercostal space

POD

Postoperative day

CT

Computed tomography
PFT
Pulmonary function test
FVC
Forced vital capacity
FEV₁
Forced expiratory volume in 1 second
PEF
Peak expiratory flow
MIP
Maximal inspiratory pressure
MEP
Maximal expiratory pressure.

Declarations

Ethics approval and consent to participate

Ethical approval was waived by the Institutional Review Board of Shenzhen University of Advanced Technology General Hospital for this single case report. Written informed consent was obtained from the patient.

Clinical trial number

Clinical trial number: not applicable.

Consent to publish

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

Funding

The authors declare that they received no specific funding for this study.

Author Contribution

Geng Chen conceived the study and drafted the manuscript. Xuanqin Wu performed the surgical and non-operative interventions. Yunxi Zhang collected radiologic data and participated in clinical care. Weimin Luo critically revised the manuscript. All authors read and approved the final manuscript.

Acknowledgements

Not applicable.

Data Availability

All data generated or analysed during this study are included in this published article.

References

1. Weissberg D, Refaely Y. Hernia of the lung. *Ann Thorac Surg.* 2002;74(6):1963–6.
2. Ugolini S, Abdelghafar M, Vokkri E, Sharkey AJ, Fontaine E, Voltolini L, Morabito A, Ugolini D, Granato F. Case Report: Spontaneous lung intercostal hernia series and literature review. *Front Surg.* 2023;9:1091727.
3. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, Cooper C, Landi F, Rolland Y, Sayer AA, et al. Sarcopenia: revised European consensus on definition and diagnosis. *Age Ageing.* 2019;48(1):16–31.
4. Fontana GA, Lavorini F. Cough motor mechanisms. *Respir Physiol Neurobiol.* 2006;152(3):266–81.
5. Laveneziana P, Albuquerque A, Aliverti A, Babb T, Barreiro E, Dres M, Dubé B-P, Fauroux B, Gea J, Guenette JA et al. ERS statement on respiratory muscle testing at rest and during exercise. *Eur Respir J* 2019, 53(6).
6. Batıhan G, Yıldız D, Ceylan KC. A rare complication of video-assisted thoracoscopic surgery: lung herniation retrospective case series of three patients and review of the literature. *Wideochir Inne Tech Maloinwazyjne.* 2019;15(1):215–9.
7. Henriksen NA, Yadete DH, Sorensen LT, Agren MS, Jorgensen LN. Connective tissue alteration in abdominal wall hernia. *Br J Surg.* 2011;98(2):210–9.
8. Henriksen NA. Systemic and local collagen turnover in hernia patients. *Dan Med J* 2016, 63(7).
9. Khansa I, Janis JE. The 4 Principles of Complex Abdominal Wall Reconstruction. *Plast Reconstr Surg Glob Open.* 2019;7(12):e2549.
10. Ema T, Funai K, Kawase A, Oiwa H, Iizuka S, Shiiya N. Incarceration hernia of the lung after video-assisted thoracic surgery requiring emergency operation: a case report. *J Thorac Dis.* 2018;10(7):E541–3.

11. Temes RT, Talbot WA, Green DP, Wernly JA. Herniation of the lung after video-assisted thoracic surgery. *Ann Thorac Surg.* 2001;72(2):606–7.

12. Lee MN, Surry LT, Ferraro DM. Aggravation and deliberation over lung herniation. *BMJ Case Rep* 2019, 12(5).

13. McDonald M-LN, Diaz AA, Ross JC, San Jose Estepar R, Zhou L, Regan EA, Eckbo E, Muralidhar N, Come CE, Cho MH, et al. Quantitative computed tomography measures of pectoralis muscle area and disease severity in chronic obstructive pulmonary disease. A cross-sectional study. *Ann Am Thorac Soc.* 2014;11(3):326–34.

14. Athanassiadi K, Bagaev E, Simon A, Haverich A. Lung herniation: a rare complication in minimally invasive cardiothoracic surgery. *Eur J Cardio-thoracic Surgery: Official J Eur Association Cardio-thoracic Surg.* 2008;33(5):774–6.

Table

Table 1. Chronological Timeline of Clinical Events, Laboratory Findings, and Interventions

Date / Timeline	Clinical Status & Events	Diagnostic & Laboratory Findings	Key Therapeutic Interventions
2026-08-12 (Pre-op)	Preoperative functional evaluation	PFT: Normal baseline spirometry (FVC 83.38%, FEV ₁ 84.81%); marked reductions in PEF (45.01%), MIP (66.85%), and MEP (79.80%).	Pulmonary prehabilitation: inhaled budesonide/formoterol, oral bromhexine.
2026-08-13 (Pre-op)	Baseline gas exchange workup	Arterial blood gas: pH 7.446, PaO ₂ 66.7 mmHg, PaCO ₂ 37.5 mmHg, P(A-a)O ₂ 33.4 mmHg.	Optimization of underlying emphysema and expectoration.
2026-08-17 (POD 0)	VATS combined segmentectomy (left S1+2c and S6), 4th ICS utility port (3.0 cm).	Histopathology: invasive adenocarcinoma (pT3N0M0, Stage IIB).	Placement of 24F chest drain; multimodal analgesia (IV dezocine PCA, parecoxib, gabapentin).
2026-08-18 (POD 1)	Hemodynamically stable	Chest CT: Postoperative baseline; dependent atelectasis, minimal hydropneumothorax.	Early ambulation, respiratory physiotherapy, pneumatic compression.
2026-08-20 (POD 3)	Transient hypoalbuminemia	Serum albumin decreased; 24-hour drain volume: 140 mL without air leak.	IV human albumin (10 g of 20% solution) + furosemide (20 mg).
2026-08-21 (POD 4, ~15:00)	Routine drain removal criteria met	Stable vital signs, bilateral lung aeration.	Left thoracic drain pulled routinely.
2026-08-21 (POD 4, ~20:00)	Violent coughing, sudden incision bulge, sharp chest pain, dyspnea.	Emergency CT: Acute 4th ICS lung hernia (hernia neck: 2.83 cm), extensive cervicothoracic subcutaneous emphysema.	Antihypertensive regimen: IV urapidil, oral nifedipine, and metoprolol.
2026-08-21 (POD 4, ~22:20)	Failed manual reduction due to tissue tension and parenchymal edema.	Incarceration confirmed; no signs of parenchymal ischemia or necrosis.	Pigtail catheter thoracostomy placed; induced pneumothorax attempted; converted to rigid pad + elastic binder.
2026-08-22 (POD 5)	Significant pain relief; binder maintained in situ.	Chest CT: Hernia sac stationary; mild reduction of subcutaneous air.	Maintained continuous compression; commenced oral amlodipine (5 mg qd).
2026-08-24 (POD 7)	Gradual symptomatic improvement	WBC $7.05 \times 10^9/L$, FIB 6.10 g/L, D-Dimer 1.58 µg/mL, Alb 35.3 g/L. CT: Partial hernia reduction; ~15% residual pneumothorax.	Sustained thoracic compression, oral lactulose for bowel regularity, cough suppression.

2026-08-28 (POD 11)	Incision soft, no cough impulse; stable respiration.	Chest CT: Subcutaneous emphysema absorbed; minor dynamic hernia sac.	Left chest drain clamped for trial observation.
2026-08-29 (POD 12)	Asymptomatic after clamping trial	Chest CT: Complete spontaneous hernia reduction; resolved pneumothorax.	Definitive removal of chest drain.
2026-09-02 (POD 16)	Fully ambulatory, no clinical recurrence.	Pre-discharge CT: Intact chest wall architecture without hernia. Normal WBC, Hgb 132 g/L, Alb 37.8 g/L.	Discharged home with oral amlodipine, loxoprofen, and bromhexine.

Abbreviations: PFT, pulmonary function test; FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 second; PEF, peak expiratory flow; MIP, maximal inspiratory pressure; MEP, maximal expiratory pressure; POD, postoperative day; VATS, video-assisted thoracoscopic surgery; ICS, intercostal space; PCA, patient-controlled analgesia; CT, computed tomography; IV, intravenous; WBC, white blood cell; FIB, fibrinogen; Alb, albumin; Hgb, hemoglobin.

Figures

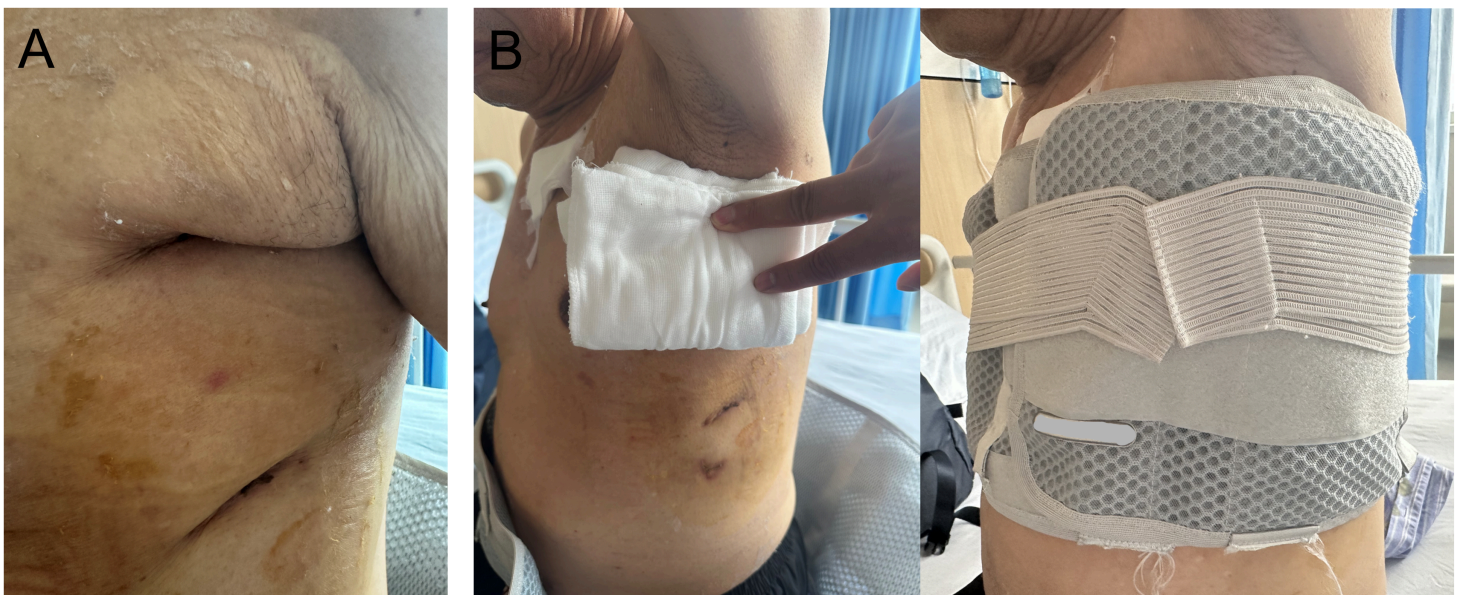


Figure 1

Clinical predispositions and conservative stabilization regimen.

(A) Postoperative clinical photograph illustrating pronounced bilateral axillary skin redundancy and subcutaneous tissue atrophy, representative of baseline systemic connective tissue attenuation and thoracic sarcopenia in this 71-year-old male.

(B) Conservative compression management: sterile contoured gauze bolster positioned directly over the 4th intercostal utility defect, secured with an adjustable elastic thoracic binder to maintain continuous localized counter-pressure.

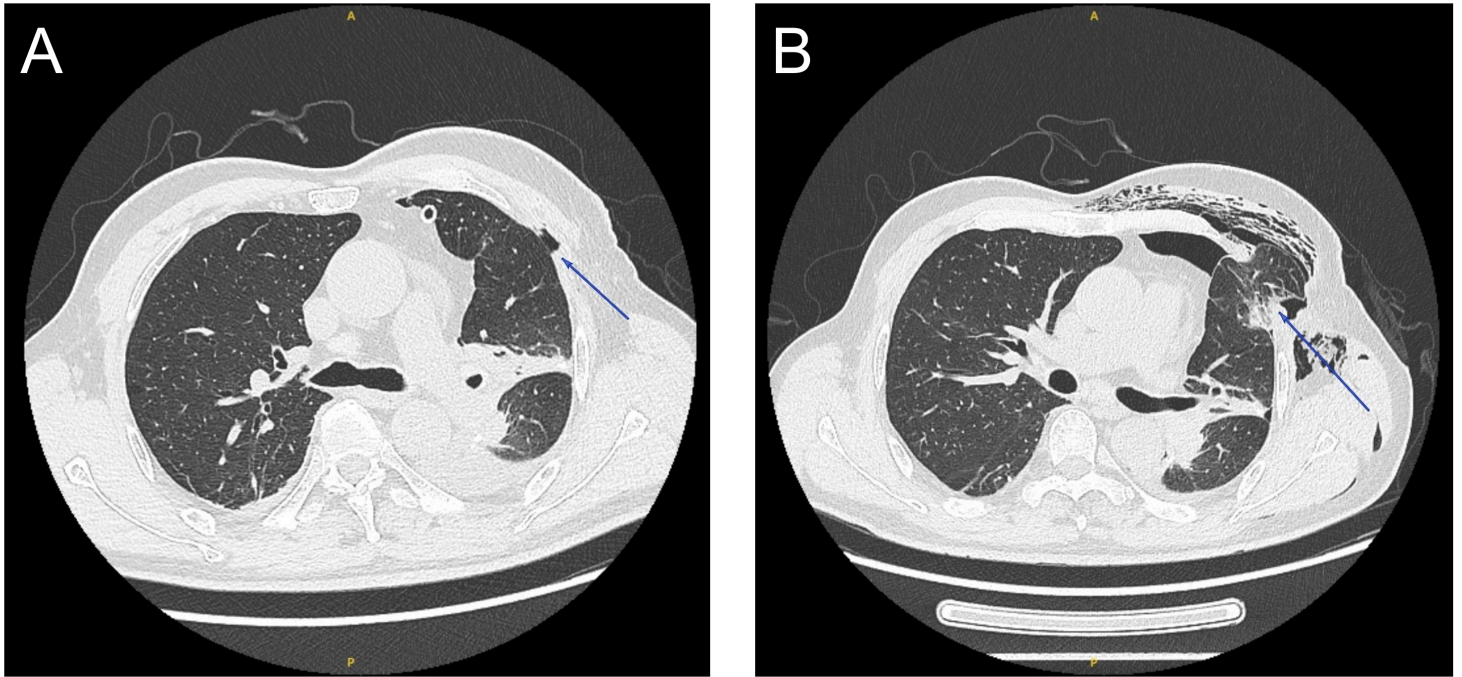


Figure 2

Radiologic presentation of acute intercostal lung herniation.

(A) Axial chest computed tomography (CT) on postoperative day (POD) 1 demonstrating normal baseline postoperative changes, intact chest wall contour, and minimal residual hydropneumothorax (blue arrow).

(B) Emergency unenhanced axial chest CT on POD 4 following vigorous coughing, illustrating acute extrusion of the left upper lobe parenchyma through the 4th intercostal space defect (blue arrow; neck measuring 2.83 cm in transverse diameter), accompanied by widespread subcutaneous emphysema dissecting the thoracic and cervical fascia.

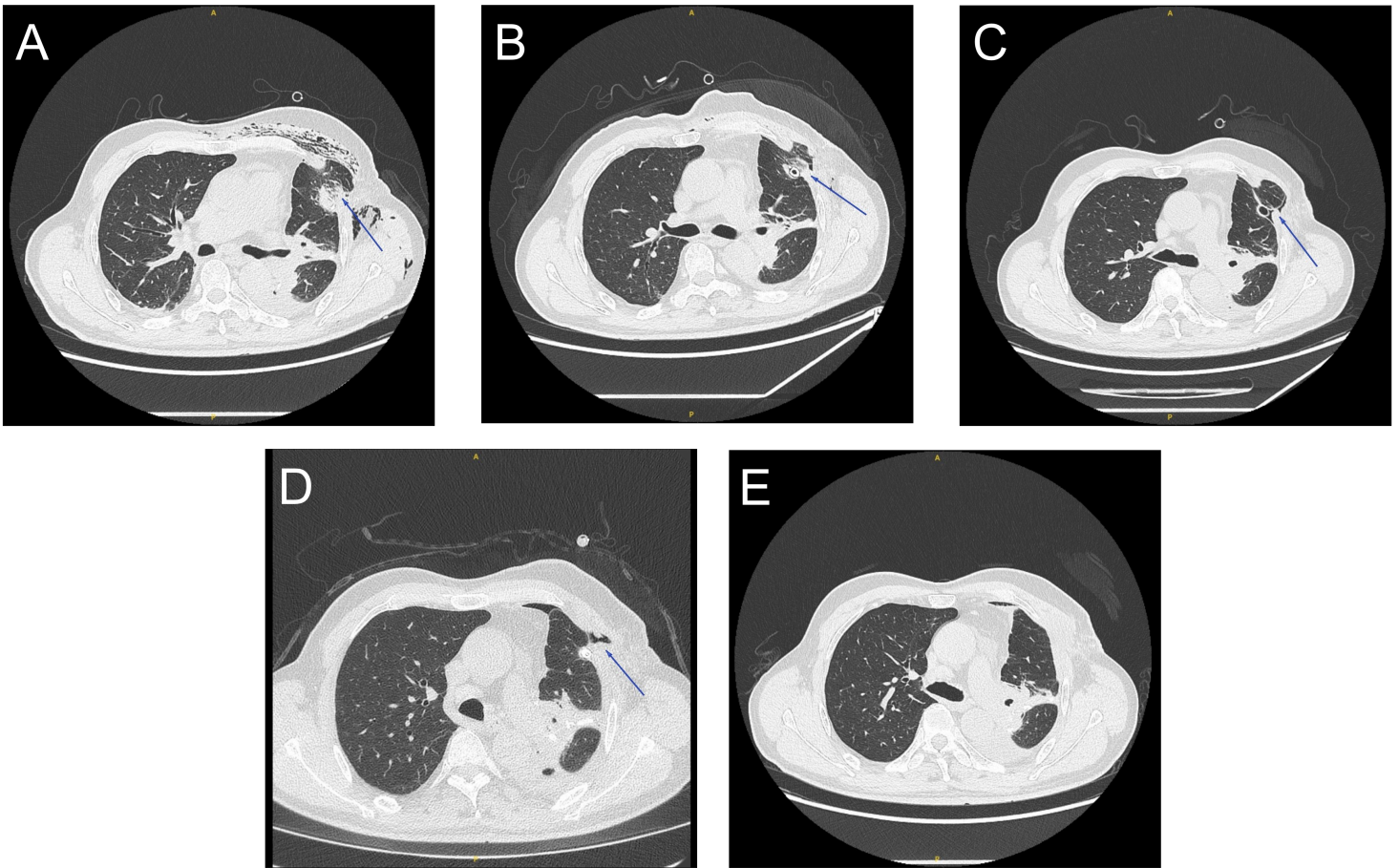


Figure 3

Serial computed tomography tracking spontaneous anatomical hernia reduction.

(A) POD 5: Stabilization of the herniated lung segment without evidence of parenchymal ischemia or strangulation, alongside early regression of subcutaneous gas.

(B) POD 7: Partial spontaneous retraction of the lung parenchyma under continuous external compression and water-seal drainage (residual left pneumothorax ~15%).

(C) POD 11: Dynamic fluctuation of the minor residual hernia sac during respiration, with near-total absorption of subcutaneous emphysema.

(D) POD 12: Complete spontaneous anatomical reduction of the herniated lung tissue back into the hemithorax with restored intercostal integrity (blue arrow), allowing permanent removal of the drainage catheter.

(E) Pre-discharge CT on POD 16: Complete resolution of subcutaneous emphysema, full re-expansion of the left lung parenchyma, and intact architecture of the left 4th intercostal space without recurrence.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [CAREchecklist.pdf](#)