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A Rare Case of Persistent Left Upper Lobe Non-expansion After Resection of a Giant Mediastinal Mature Teratoma Managed With Lobectomy

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Abstract

Giant mediastinal mature teratomas can compress the lungs, airways, and great vessels, although lung re-expansion after tumor removal is not always achieved. A 60-year-old woman with a history of childhood poliomyelitis presented with progressive dyspnea and weakness over 12 days, followed by gasping respiration and decreased consciousness requiring intubation and mechanical ventilation. Admission arterial blood gas analysis showed severe hypercapnic respiratory failure with acidemia (pH 7.258, partial pressure of carbon dioxide ($PaCO_2$) 87.9 mmHg). Computed tomography revealed a 15.76-cm anterior mediastinal mass containing fat and calcification, displacing the heart and great vessels, narrowing the right pulmonary artery, and causing compressive atelectasis of the left lung. Bilateral pulmonary opacities, right lower lobe bronchiectasis, and a sputum culture positive for *Pseudomonas aeruginosa* suggested possible concomitant pulmonary disease. The well-circumscribed tumor was resected through a left hemiclamshell incision with median sternotomy, with no gross evidence of invasion into adjacent structures. Despite intraoperative re-expansion attempts, the left upper lobe remained collapsed, and left upper lobectomy was performed. Histopathology confirmed a mature teratoma, while examination of the resected lobe showed atelectasis without tumor involvement. The patient remained ventilator-dependent after an unsuccessful extubation attempt and later developed severe metabolic and hemodynamic deterioration, including hypoglycemia and refractory hypotension despite maximal vasopressor support. The patient died on postoperative day 8 after further clinical deterioration. This case demonstrates that the left upper lobe may fail to re-expand despite removal of a compressing mediastinal mass and intraoperative re-expansion attempts, even in the absence of tumor involvement on histopathology.

Categories: General Surgery, Oncology, Cardiac/Thoracic/Vascular Surgery
Keywords: adult thoracic surgery, lung resection, mediastinal teratoma, pulmonary atelectasis, severe respiratory failure

Introduction

Mediastinal teratomas are germ cell tumors that commonly arise in the anterior mediastinum and contain tissues derived from multiple embryonic germ layers [1,2]. Mature teratomas are generally benign, and complete surgical resection is the mainstay of treatment [2]. However, giant tumors may compress adjacent intrathoracic structures and cause severe respiratory compromise [1,2].

Pulmonary atelectasis secondary to extrinsic compression may improve following tumor removal, but the degree of lung re-expansion can vary. Published surgical reports describe both successful pulmonary re-expansion after tumor resection and cases requiring concomitant pulmonary resection [1,3,4]. However, the management of a lobe that fails to re-expand after removal of a compressing mediastinal mass without apparent tumor invasion is less clearly described in the available literature. In such cases, intraoperative assessment of lung re-expansion and the underlying cause of non-expansion may inform surgical decision-making.

We report a case of a giant anterior mediastinal mature teratoma in a 60-year-old woman presenting with severe respiratory failure requiring mechanical ventilation. Following tumor removal, the left upper lobe failed to expand, prompting lobectomy. Histopathological examination demonstrated atelectasis without tumor involvement of the resected lung. This case highlights the challenges of intraoperative decision-making when pulmonary re-expansion does not occur after tumor decompression and the underlying mechanism remains uncertain.

Case Presentation

A 60-year-old woman presented to the emergency department with progressive dyspnea and generalized weakness for approximately 12 days, followed by gasping respiration and decreased consciousness requiring endotracheal intubation and mechanical ventilation (Table *1*). Admission arterial blood gas analysis

revealed severe hypercapnic respiratory failure with acidemia (pH 7.258, partial pressure of carbon dioxide ($PaCO_2$) 87.9 mmHg, and partial pressure of oxygen (PaO_2) 60.7 mmHg on a fraction of inspired oxygen (FiO_2) of 0.80). Her medical history included childhood poliomyelitis, with no reported history of hypertension, diabetes mellitus, cardiac disease, or kidney disease.

Parameter	At admission	Postoperative day 6	Reference Range
Complete blood count			
Hemoglobin (g/dL)	12.3	10.5	12.0-15.0
Hematocrit (%)	42.5	36.1	35.0-49.0
Leukocytes (×10³/µL)	12.7	30.6	4.50-11.50
Platelets (×10³/µL)	197	243	150-450
Arterial blood gas analysis			
pH	7.258	7.093	7.350-7.450
$PaCO_2$ (mmHg)	87.9	60.7	35.0-45.0
PaO_2 (mmHg)	60.7	34.5	83.0-108.0
Oxygen saturation (%)	89.7	62.9	94.0-98.0
HCO_3^- (mmol/L)	38.3	18.1	22.0-26.0
Base excess (mmol/L)	+8.54	−11.43	Not available
Lactate (mmol/L)	5.3	9.2	<1.8
FiO_2 (%)	80	90	Not applicable
Renal function and electrolytes			
BUN (mg/dL)	21	46	8-23
Creatinine (mg/dL)	0.56	1.90	0.51-0.95
Sodium (mmol/L)	138	132	136-145
Potassium (mmol/L)	5.2	6.9	3.5-5.1
Chloride (mmol/L)	97	90	98-107
Liver function			
AST/SGOT (U/L)	55	404	10-35
Glucose metabolism			
Random blood glucose (mg/dL)	183	22	70-140

TABLE 1: Laboratory findings at admission and on postoperative day 6

$PaCO_2$, partial pressure of carbon dioxide; PaO_2, partial pressure of oxygen; HCO_3^-, bicarbonate; FiO_2, fraction of inspired oxygen; BUN, blood urea nitrogen; AST/SGOT, aspartate aminotransferase. Reference ranges were obtained from the institutional laboratory reference intervals. FiO_2 is a ventilatory parameter and has no reference range. The reference range for base excess was not available in the laboratory records.

Chest radiography demonstrated extensive opacification of the left hemithorax with rightward displacement of the mediastinal structures (Figure *1A*). Contrast-enhanced chest computed tomography revealed a heterogeneous anterior mediastinal mass measuring 11.4 × 10.64 × 15.76 cm, containing fat and calcification (Figure *1B*-*1D*). The mass displaced the heart and major vascular structures to the right and narrowed the right pulmonary artery. Compressive atelectasis involved the anterior segments of the left upper and lower lobes. Bilateral ground-glass opacities and consolidations, possibly representing concomitant pulmonary disease, and right lower lobe bronchiectasis were also observed. Microbiological evaluation revealed no growth in blood and urine cultures, while sputum culture grew *Pseudomonas aeruginosa* (susceptible to cefepime, ceftazidime, and ciprofloxacin; intermediate to levofloxacin; resistant to meropenem and piperacillin/tazobactam).

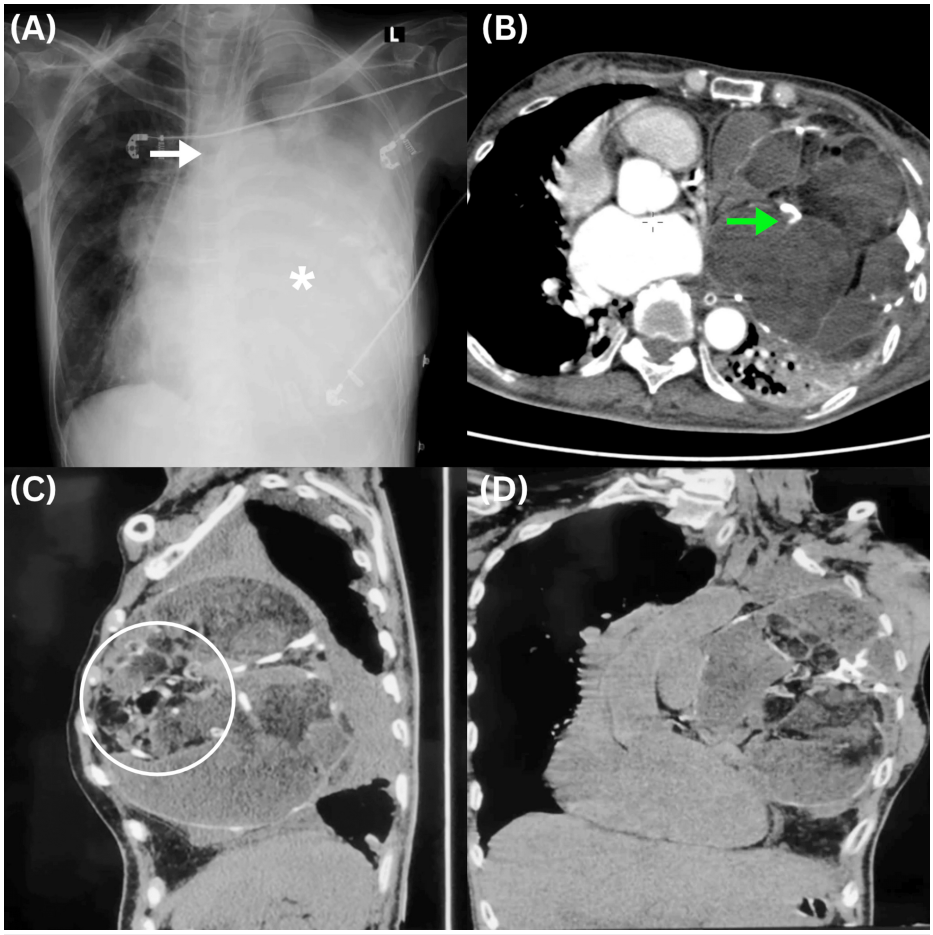


FIGURE 1: Preoperative radiological findings of a giant anterior mediastinal mature teratoma

(A) Chest radiograph demonstrating rightward deviation of the trachea (arrow) and extensive opacification of the left hemithorax (asterisk). (B) Axial contrast-enhanced computed tomography showing a heterogeneous anterior mediastinal mass with a calcified component (green arrow). (C) Sagittal computed tomography reconstruction highlighting the heterogeneous internal components of the mass (circle). (D) Coronal computed tomography reconstruction demonstrating the craniocaudal extent of the mass and compression of adjacent intrathoracic structures.

The patient underwent tumor resection through a left hemiclamshell incision with median sternotomy. Intraoperatively, a well-circumscribed solid mass measuring approximately 10 × 14 × 14 cm was identified, with no gross evidence of surrounding tissue infiltration (Figure *2A*). On sectioning, the mass consisted predominantly of adipose tissue, with hard components resembling bone and scattered strands of hair (Figure *2B*, *2C*). Following tumor resection, the left upper lobe failed to re-expand despite intraoperative re-expansion attempts using mechanical ventilation, prompting left upper lobectomy (Figure *2D*). The tumor, resected lung, and sampled suprasternal lymph nodes were submitted for histopathological examination.

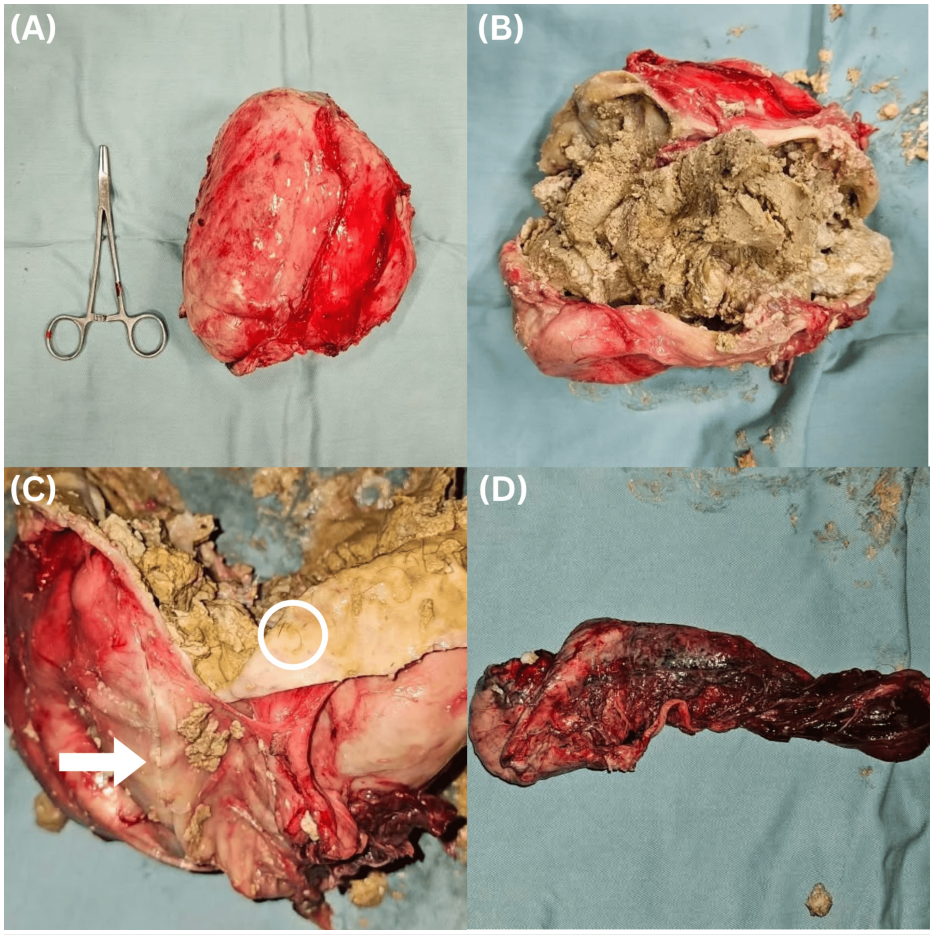


FIGURE 2: Intraoperative and gross pathological findings of the giant mediastinal mature teratoma

(A) External appearance of the resected mediastinal mass. (B) Cut surface showing predominantly adipose material with areas of hard tissue and hair. (C) Opened specimen showing hard components resembling bone (arrow) and strands of hair (circle). (D) Resected left upper lobe specimen following lobectomy.

Histopathological examination confirmed a mature teratoma containing ectodermal derivatives (squamous epithelium, hair follicles, and sebaceous glands), mesodermal derivatives (adipose tissue, cartilage, bone, and bone marrow), and endodermal derivatives (respiratory epithelium) (Figure *3A*-*3D*). The resected left upper lobe showed atelectasis without tumor involvement (Figure *3E*-*3F*), while the sampled suprasternal lymph nodes showed sinus histiocytosis without evidence of tumor.

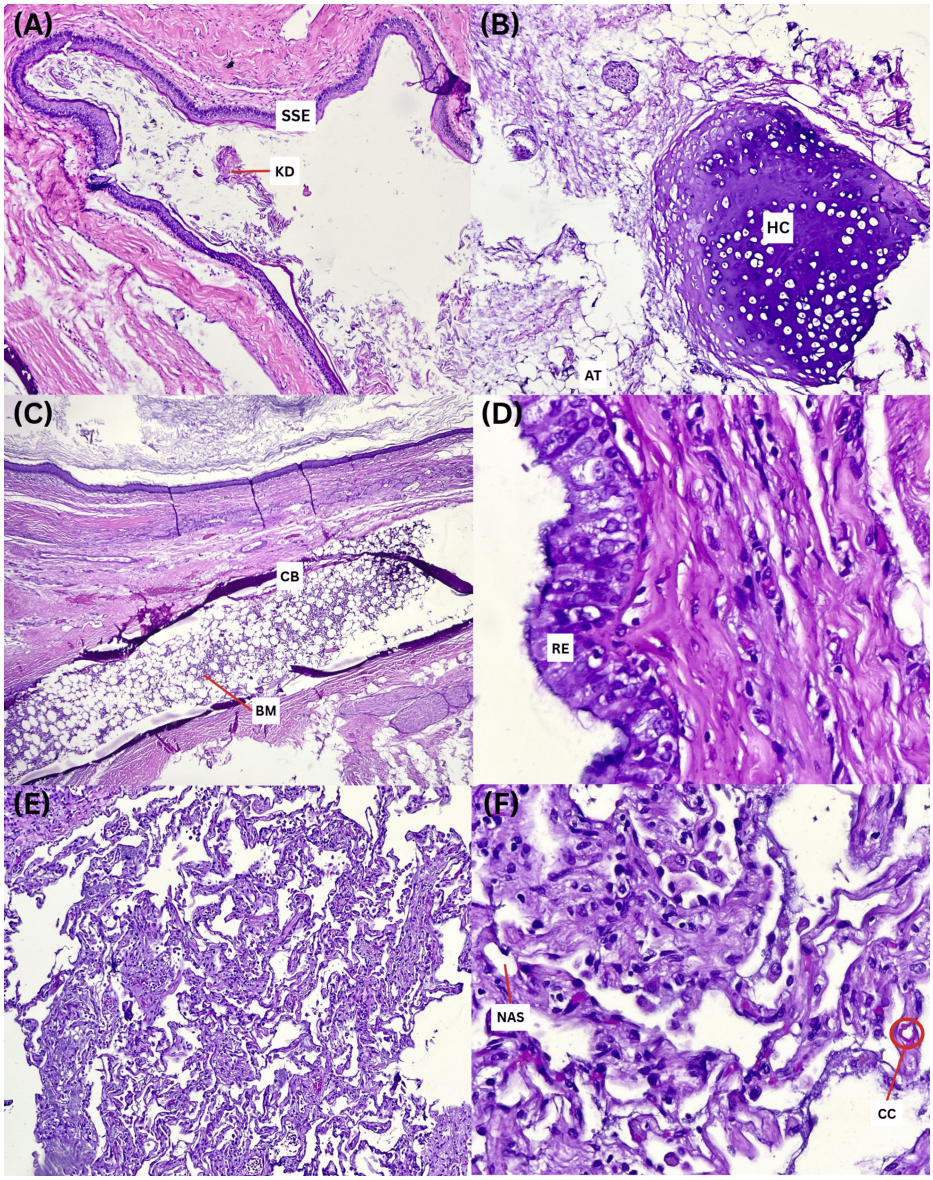


FIGURE 3: Histopathological findings of the mediastinal tumor and resected lung specimen

(A) Stratified squamous epithelium (SSE) with keratin debris (KD), consistent with ectodermal differentiation (H&E, ×100). (B) Hyaline cartilage (HC) and adipose tissue (AT), representing mesodermal components (H&E, ×100). (C) Compact bone (CB) and bone marrow (BM), representing mesodermal components (H&E, ×40). (D) Epithelial lining consistent with respiratory epithelium (RE), representing endodermal differentiation (H&E, ×400). (E) Low-magnification view of the resected lung tissue showing the alveolar architecture (H&E, ×100). (F) Thickened alveolar septa, narrowed alveolar spaces (NAS), and congested capillary (CC) in the resected lung tissue (H&E, ×400).

Postoperative chest radiography on postoperative day 1 demonstrated persistent extensive opacification of the left hemithorax, with additional bilateral pulmonary opacities (Figure *4*). An attempted extubation was unsuccessful, necessitating reintubation. The subsequent clinical course was complicated by severe respiratory and metabolic derangements (Table *1*), including profound hypoglycemia and refractory hypotension despite maximal vasopressor support. The patient died on postoperative day 8.

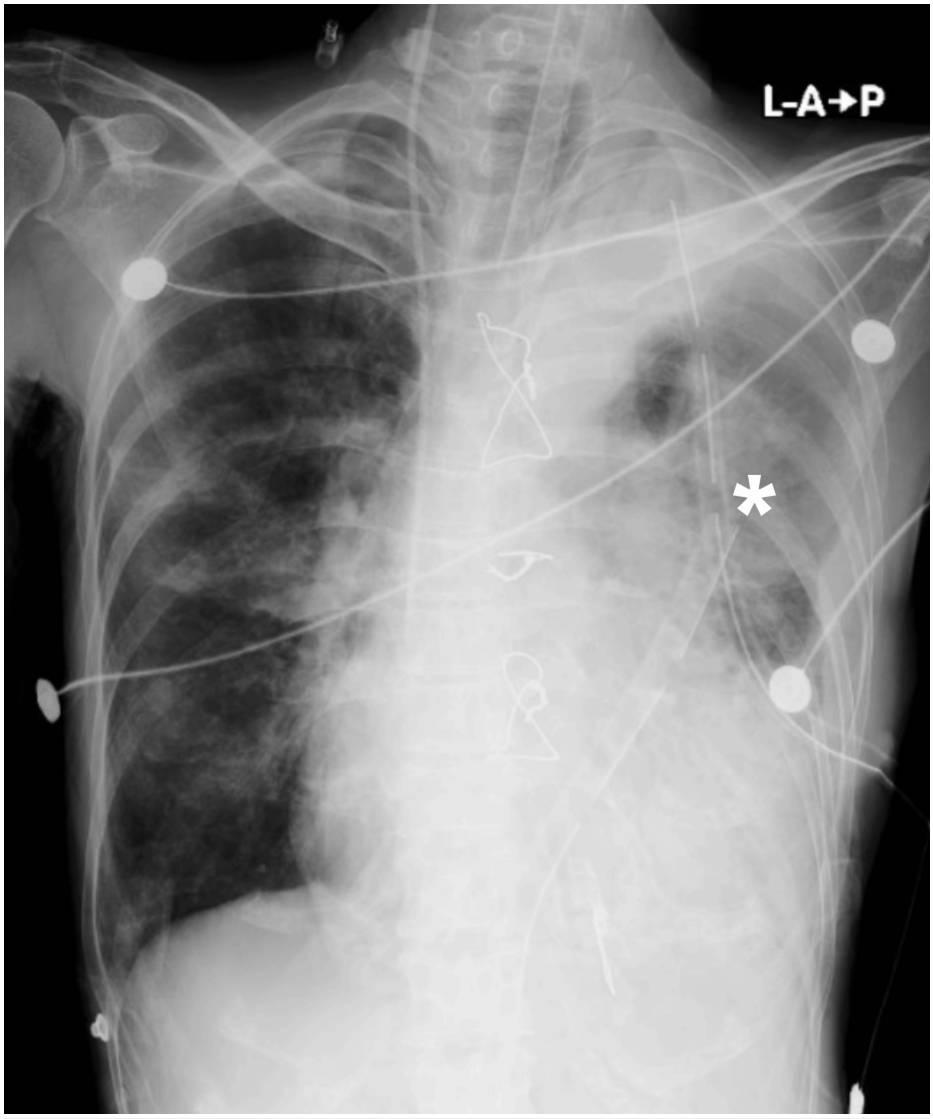


FIGURE 4: Postoperative chest radiograph on postoperative day 1 following left upper lobectomy

The asterisk indicates persistent extensive opacification of the left hemithorax, with additional bilateral pulmonary opacities.

Discussion

This case highlights the challenge of managing a giant mediastinal mature teratoma in a patient presenting with severe respiratory failure. Following tumor removal, the left upper lobe (LUL) failed to re-expand despite intraoperative re-expansion attempts, and left upper lobectomy was subsequently performed. Histopathological examination demonstrated atelectasis without tumor involvement. This finding raises an important clinical question regarding why a previously compressed pulmonary lobe may fail to re-expand after removal of the compressing mass. Although the available findings establish the occurrence of non-expansion, they do not identify its underlying mechanism.

The patient's respiratory failure may have been acute-on-chronic. Admission arterial blood gas analysis demonstrated severe hypercapnia and acidemia (pH 7.258, $PaCO_2$ 87.9 mmHg), accompanied by hypoxemia (PaO_2 60.7 mmHg on an FiO_2 of 0.80). The elevated bicarbonate level (38.3 mmol/L) and positive base excess (+8.54) suggest metabolic compensation, supporting the possibility of pre-existing hypoventilation rather than an entirely acute process developing over 12 days. Her history of childhood poliomyelitis is also relevant, as residual respiratory muscle weakness can contribute to hypoventilation in post-polio patients [5]. Nevertheless, no neuromuscular assessment was available, and the contribution of previous poliomyelitis to her respiratory failure remains uncertain.

The patient's respiratory failure was also likely influenced by structural and pulmonary factors. Computed

tomography showed substantial displacement of the mediastinal structures, narrowing of the right pulmonary artery, and compressive atelectasis involving the anterior segment of the left upper lobe and the left lower lobe. Large mediastinal masses can impair respiratory function through compression of adjacent structures, although the functional consequences of right pulmonary artery narrowing were not directly assessed in this patient [1,2]. Bilateral pulmonary opacities and consolidation, together with right lower lobe bronchiectasis and a sputum culture positive for *Pseudomonas aeruginosa*, suggest possible concurrent pulmonary disease. However, the culture result alone does not establish active infection or its contribution to respiratory failure.

The patient's pre-existing pulmonary abnormalities may have further reduced her pulmonary reserve. Bilateral pulmonary opacities, right lower lobe bronchiectasis, and left-sided compressive atelectasis may have limited her respiratory capacity before surgery. Following left upper lobectomy, the additional loss of lung parenchyma may have further reduced her available respiratory reserve, potentially contributing to persistent ventilator dependence and postoperative respiratory deterioration. However, the extent to which lobectomy contributed to the subsequent clinical deterioration cannot be determined from the available data.

Previous reports illustrate the variable clinical presentation and surgical management of mediastinal mature teratomas. Some lesions may remain asymptomatic, whereas others cause symptoms through compression of adjacent structures [2,4]. Zisis et al. described a giant teratoma associated with total left lung atelectasis in which the collapsed lung re-expanded after tumor removal without pulmonary resection [3]. This contrasts with the present case, in which the LUL failed to re-expand after tumor removal.

Pulmonary resections have also been reported in surgical series of mediastinal mature teratomas. Tian et al. reported five lobectomies and nine wedge resections among 108 patients [6]. Zhang et al. reported nine pulmonary resections in a series of 101 patients, including two lobectomies in the context of severe adhesions and loss of lobar function [1]. Anushree and Shanti also reported right middle lobectomy for atelectasis attributed to previous CT-guided aspiration [4]. However, the reports we identified do not establish whether failure of re-expansion alone is sufficient to justify lobectomy. In the present case, no gross tumor infiltration was documented, and histopathology showed atelectasis without tumor involvement. Whether lung preservation would have been feasible remains uncertain.

Several mechanisms may explain the failure of a pulmonary lobe to re-expand after decompression. Compression atelectasis occurs when external pressure promotes alveolar collapse, whereas airway obstruction may produce resorptive atelectasis through absorption of gas distal to the obstruction [7,8]. Acute lobar atelectasis may have potentially reversible contributors, including airway obstruction and retained secretions, while recruitment maneuvers must be considered in the context of respiratory mechanics and the patient's clinical condition [9,10].

Intraoperative attempts to re-expand the left upper lobe were made using mechanical ventilation. However, the specific techniques, duration, and detailed findings that informed the decision to perform lobectomy were not available for review. Bronchoscopy was not performed, so an endobronchial cause could not be directly assessed.

Histopathological examination of the resected left upper lobe showed narrowing of some alveolar lumina, relatively thickened alveolar septa, and vascular congestion, without tumor involvement. These findings were consistent with atelectatic changes but did not establish the underlying mechanism of persistent non-expansion. Whether the non-expansion resulted from prolonged compression, pre-existing parenchymal changes, or other factors remains uncertain.

The postoperative course was complicated by continued ventilator dependence following an unsuccessful extubation attempt and subsequent severe physiological deterioration. By postoperative day 6, the patient had acidemia, hypoxemia, hyperlactatemia, hyperkalemia, hypoglycemia, worsening renal and hepatic biochemical indices, and hypotension refractory to maximal vasopressor support. These findings indicate critical systemic illness but do not establish a single underlying cause. Potential contributors include ongoing respiratory dysfunction, infection or sepsis, cardiovascular complications, and other forms of postoperative organ dysfunction; none can be confirmed as the cause of death from the available information. Ka Sha et al. reported a case of cardiac arrest associated with resection of a giant mediastinal tumor and discussed potential perioperative cardiopulmonary mechanisms [11]. This report illustrates the potential for serious perioperative deterioration in patients undergoing resection of giant mediastinal tumors, but it does not establish the mechanism of deterioration in the present case. No further postoperative imaging beyond the chest radiograph obtained on postoperative day 1 (Figure *4*) was available for review.

This report is limited by incomplete details of the intraoperative assessment, the unknown duration of preoperative lobar collapse, the absence of a documented neuromuscular evaluation, and uncertainty regarding the contribution of concurrent pulmonary abnormalities to respiratory failure. The lack of further postoperative imaging and subsequent clinical and laboratory data, as well as the absence of an autopsy,

limited our ability to determine the precise mechanism of postoperative deterioration and the cause of death. As a single case, this report cannot establish the frequency or predictors of failure of pulmonary re-expansion after mediastinal tumor resection. Nevertheless, it highlights the importance of evaluating lobar non-expansion as a distinct intraoperative problem rather than assuming that removal of the compressing mass will necessarily restore lung aeration. Depending on the operative findings, clinical considerations may include identifying potentially reversible causes such as retained secretions or airway distortion, assessing the response to re-expansion attempts, and evaluating pulmonary perfusion and viability before deciding whether further lung-preserving measures or resection are appropriate. These considerations are general clinical implications, not evidence that all such assessments were performed in this patient. The mechanism of non-expansion and the feasibility of lung preservation remain unresolved.

Conclusions

Removal of a giant mediastinal mature teratoma does not necessarily restore pulmonary expansion. In this case, the left upper lobe failed to re-expand despite intraoperative attempts, and lobectomy was subsequently performed. Histopathology demonstrated atelectasis without tumor involvement, although the mechanism of non-expansion remained uncertain. This case highlights the importance of assessing lobar re-expansion after tumor decompression when making intraoperative decisions about pulmonary resection.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Nadila Yang

Drafting of the manuscript: Nadila Yang

Concept and design: Ihsanul Amal

Critical review of the manuscript for important intellectual content: Ihsanul Amal

Supervision: Ihsanul Amal

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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