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Plastic Bronchitis Following Massive Hemothorax and Hemorrhagic Shock: A Case Report and Mechanistic Insights

Masif Hemotoraks ve Hemorajik Şok Sonrası Gelişen Plastik Bronşit: Olgu Sunumu ve Mekanistik Bulgular

Shengquan Wei, Jinghe Chen, Mingjun Wu

Abstract

Type 1 fibrinous plastic bronchitis (PB) is rare in adults, especially following massive hemothorax. A 62-year-old smoker developed extensive fibrinous bronchial casts after thoracoscopic evacuation of a massive right hemothorax (6,500 mL) with hemorrhagic shock, massive transfusion, and consumptive coagulopathy. Bronchoscopy on postoperative day 4 revealed branching casts in the segmental bronchi. Repeated mechanical extraction, saline lavage, and viscoelastic-guided correction of coagulopathy, without the use of intrabronchial fibrinolytics, led to resolution. Histopathology confirmed Type 1 inflammatory PB. Massive hemorrhage may trigger Type 1 PB through airway epithelial injury, a procoagulant milieu, and neutrophilic inflammation. Prompt bronchoscopic clearance with targeted hemostatic correction may be key when fibrinolysis is deferred because of bleeding risk.

Keywords: *Plastic bronchitis, hemothorax, hemorrhagic shock, coagulopathy, bronchoscopy.*

Öz

Tip 1 fibröz plastik bronşit (PB), özellikle masif hemotoraks sonrasında yetişkinlerde nadir görülmektedir. Altmış iki yaşında sigara içen bir hastada, hemorajik şok, yoğun kan transfüzyonu ve tüketim koagülopatisi ile birlikte sağda masif hemotoraksın (6500 mL) torakoskopik olarak boşaltılmasından sonra yaygın fibröz bronşiyal yapılar ortaya çıktı. Ameliyat sonrası 4. günde yapılan bronkoskopide segmental bronşlarda dallanan yapılar görüldü. Tekrarlanan mekanik ekstraksiyon, intrabronşiyal fibrinolitikler kullanılmadan sadece serum fizyolojik lavajı ve viskoelastik yönlendirmeli koagülasyonun düzeltilmesi, iyileşmeyi sağladı. Histoloji, Tip 1 inflamatuvar PB'yi doğruladı. Masif kanama, hava yolu epitel hasarı, prokoagülan ortam ve nötrofilik inflamasyon Tip 1 PB'i tetikleyebilir. Bu hastalarda, fibrinoliz olmaksızın, hemostatik dengenin düzeltilmesi ile hızlı bronkoskopik temizlik çok önemlidir.

Anahtar Kelimeler: *Plastik bronşit, hemotoraks, hemorajik şok, koagülopati, bronkoskopi.*

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Plastic bronchitis (PB) is a rare condition characterized by the formation and expectoration or bronchoscopic removal of firm, branching endobronchial casts that partially or completely obstruct the tracheobronchial tree (1,2). Two principal phenotypes have been described: Type 1 (inflammatory), consisting of fibrin-rich material with dense cellular infiltrates, and Type 2 (lymphatic/mucinous), which is often acellular and associated with lymphatic abnormalities (1,3). Although PB is most frequently reported in pediatric populations, particularly following Fontan palliation for congenital heart disease, adult cases, though rare, have been described in association with diverse etiologies, including lymphatic disorders, infections, and inflammatory conditions (4,5).

Recent advances in understanding PB pathogenesis have highlighted the potential role of neutrophil extracellular traps (NETs) in stabilizing fibrin matrices and promoting cast persistence within the airway lumen (6). However, the pathophysiologic mechanisms linking systemic hemorrhagic events to the development of Type 1 fibrinous PB remain poorly characterized. Here, we present an adult case of Type 1 PB that developed following massive intrathoracic hemorrhage and hemorrhagic shock, discuss mechanistic considerations grounded in contemporary literature, and describe a multimodal management approach that integrated mechanical clearance, inhaled anti-inflammatory therapy, and hemostatic correction while deferring fibrinolytic therapy because of ongoing bleeding risk.

CASE

A 62-year-old man with a 40-pack-year smoking history presented with acute right-sided pleuritic chest pain and progressive dyspnea following a forceful coughing episode. The patient reported a fall four days before symptom onset. Initial computed tomography (CT) imaging demonstrated a large right hemothorax with mediastinal shift and passive atelectasis of the right lower lobe (Figure 1). Emergency thoracoscopic evacuation was performed, with an estimated intraoperative blood loss of 6,500 mL.

The postoperative course was complicated by refractory hemorrhagic shock necessitating massive transfusion (12 units of packed red blood cells, 8 units of fresh frozen plasma, and 2 pools of platelets), vasopressor support, and invasive mechanical ventilation. The patient did not experience hemoptysis during the hospital course, although bronchoscopy revealed blood-tinged sputum, findings attributable to the underlying intrapleural bleeding and airway mucosal irritation rather than primary pulmonary hemorrhage.

On postoperative day 4, persistent lobar collapse and worsening hypoxemia (PaO2/FiO2 ratio, 150 mmHg)

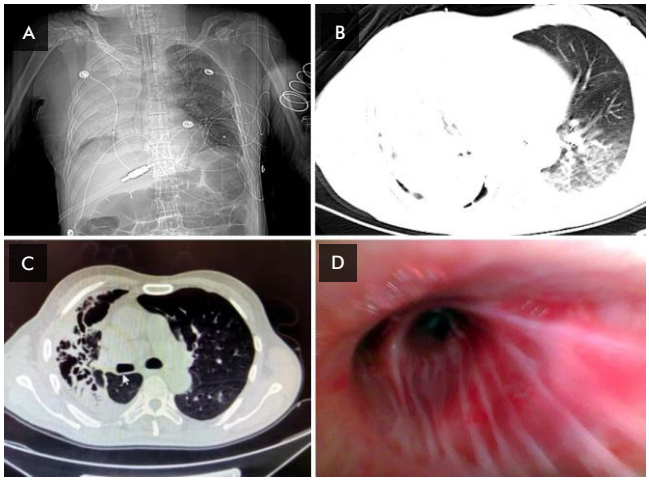


Figure 1: Preoperative chest radiograph showing a large right-sided hemothorax with associated lung compression, leftward mediastinal shift, and passive atelectasis of the right lower lobe (A); postoperative day 3 chest CT (axial view) following thoracoscopic evacuation showing persistent right opacification and cast-related lobar collapse despite pleural drainage (B); postoperative week 5 chest CT (axial view) demonstrating complete resolution of hemothorax, re-expansion of the right lung, and absence of residual pleural fluid (C); bronchoscopic view at week 5 confirming complete clearance of airway casts with restoration of normal bronchial mucosa (D).

prompted urgent flexible bronchoscopy. Endoscopic examination revealed extensive yellowish, focal fibrinous bronchial casts admixed with blood-tinged sputum, with branching casts occluding the posterior segmental bronchi of the right lower lobe and the apical segment of the right upper lobe (Figure 2). The casts were cohesive and rubbery in consistency. Under general anesthesia, mechanical extraction was performed using a cryoprobe and flexible forceps, followed by segmental saline lavage.

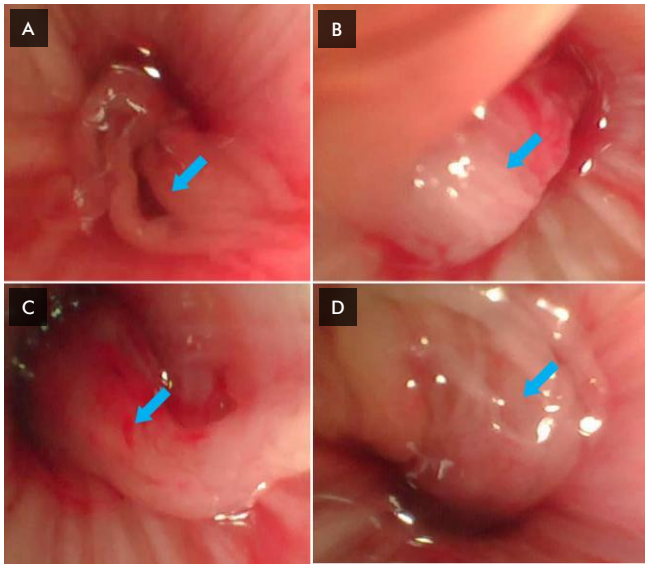


Figure 2: Endobronchial casts extracted by bronchoscopy. Representative photographs of fibrinous bronchial casts retrieved during serial bronchoscopic procedures on postoperative days 4–12. The casts exhibited characteristic yellowish-tan coloration, branching morphology reflecting segmental airway architecture, and rubbery consistency. Multiple sub-branches corresponding to distal bronchi were visible.

Given the inflammatory nature of the casts and the patient's smoking history, adjunctive inhaled therapy was initiated, comprising budesonide 1 mg nebulized twice daily and salbutamol 2.5 mg with 2 mL of normal saline nebulized twice daily. Serial bronchoscopic procedures were performed every other day to address recurrent cast formation, with the gross cast yield progressively declining after each session. By week 2, the airway fibrinous material was markedly reduced; however, the patient declined continued nebulized therapy. Bronchoscopic clearance was continued three times weekly during weeks 3–5 until airway casts were no longer visible endoscopically (Figure 1D).

Laboratory evaluation revealed a platelet nadir of 26 $\times$ 10^9/L, fibrinogen $<$0.8 g/L, and an international normalized ratio (INR) of 2.3, consistent with consumptive coagulopathy. Rotational thromboelastometry (ROTEM)-guided hemostatic interventions, including fibrinogen repletion with cryoprecipitate, platelet transfusion, and prothrombin complex concentrate as indicated, were instituted to correct systemic coagulopathy.

Histopathologic examination of the extracted casts demonstrated dense interlacing fibrin strands with abundant neutrophilic infiltration and negligible mucin content, confirming the diagnosis of Type 1 inflammatory PB (Figure 3).

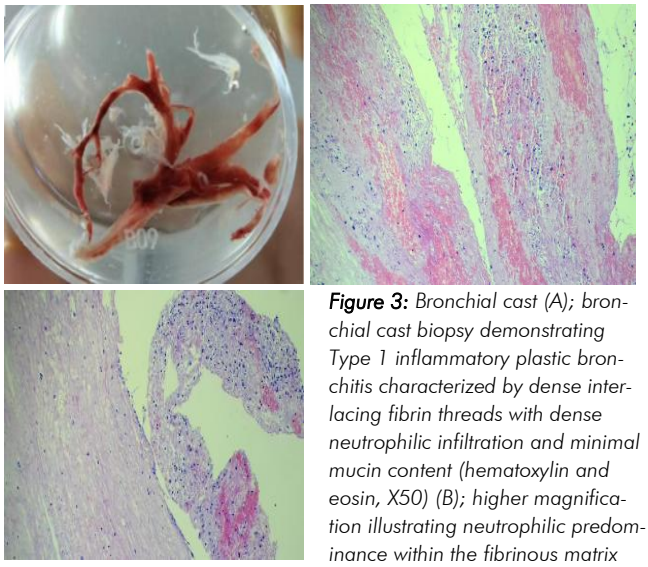


Figure 3: Bronchial cast (A); bronchial cast biopsy demonstrating Type 1 inflammatory plastic bronchitis characterized by dense interlacing fibrin threads with dense neutrophilic infiltration and minimal mucin content (hematoxylin and eosin, X50) (B); higher magnification illustrating neutrophilic predominance within the fibrinous matrix (H&E, X100) (C).

Bronchoalveolar lavage cultures and multiplex viral polymerase chain reaction (PCR) testing were negative for pathogenic organisms. Histopathologic examination of the right pleural lesion (parietal pleura surgical specimen) showed edematous fibroadipose tissue with focal lymphocytic infiltration, extensive subpleural hemorrhage, and fibrin deposition on the pleural surface, consistent with hemorrhagic pleuritis; acid-fast staining was negative for mycobacteria (Figure 4).

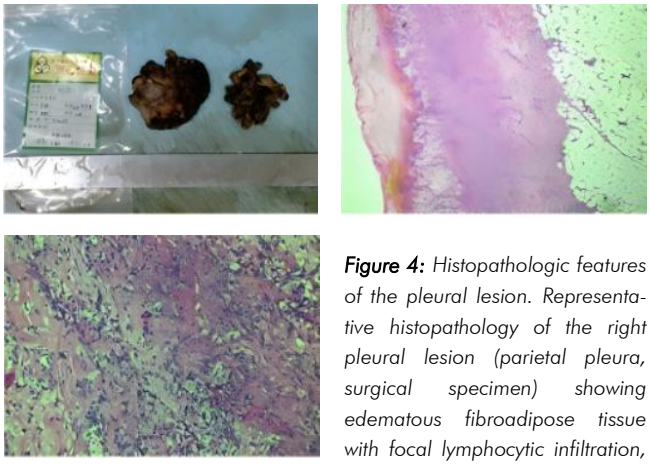


Figure 4: Histopathologic features of the pleural lesion. Representative histopathology of the right pleural lesion (parietal pleura, surgical specimen) showing edematous fibroadipose tissue with focal lymphocytic infiltration, extensive subpleural hemorrhage, and fibrin deposition on the pleural surface (hematoxylin and eosin stain, X50) (A,B); acid-fast staining negative for mycobacteria (C).

Given the potential association between PB and hemodynamic perturbations, transthoracic echocardiography was performed and revealed normal biventricular systolic function, no valvular abnormalities, and no evidence of pulmonary hypertension or right heart dysfunction. These findings excluded a significant cardiac contribution to cast formation in this case.

In view of the ongoing systemic bleeding risk, intrabronchial fibrinolytic therapy was withheld. Management focused on four key elements: (a) repeated mechanical clearance to restore and maintain airway patency, (b) segmental saline lavage to facilitate removal of residual fibrinous material, (c) inhaled corticosteroids and bronchodilators to attenuate airway inflammation and hyperresponsiveness, and (d) protocol-driven correction of systemic hemostatic derangements guided by viscoelastic testing.

Cast production diminished progressively, with substantial reduction by week 2 and complete clearance by week 5. Oxygenation improved (PaO2/FiO2 ratio, 500 mmHg), vasopressors were successfully weaned by day 12, and the patient was extubated on day 16. He was discharged to a rehabilitation facility on day 24 and remained free of recurrent respiratory symptoms at the three-month follow-up. Pulmonary function testing was not completed due to patient-related factors.

DISCUSSION

This case documents the occurrence of adult Type 1 fibrinous PB following massive intrathoracic hemorrhage and hemorrhagic shock, a scenario that illustrates how systemic hemorrhagic events and associated coagulopathy can create a profibrinogenic intrabronchial environment. Our multimodal management approach, combining serial bronchoscopic mechanical clearance, inhaled anti-inflammatory therapy, and targeted correction of

systemic hemostasis while deferring airway fibrinolytic therapy, proved successful and aligns with contemporary management strategies reported in the literature (4,7,8).

The pathophysiology of PB in this context likely involves three convergent processes. First, extensive intrathoracic bleeding and ischemia-reperfusion injury to the bronchial epithelium permit the ingress of fibrinogen-rich plasma into the airway spaces, with local coagulation activation favoring fibrin polymerization and deposition within the bronchial lumen. The patient's 40-pack-year smoking history may have predisposed him to preexisting airway epithelial vulnerability, potentiating fibrinogen leakage and impairing normal mucociliary clearance mechanisms. Second, systemic consumptive coagulopathy and transfusion-related hemostatic perturbations may create a regional procoagulant milieu, with an imbalance between coagulation and fibrinolysis, while stasis within poorly ventilated or collapsed lung segments facilitates cast consolidation. Third, the dense neutrophilic infiltrate observed histologically suggests neutrophil-mediated scaffold formation through the release of proteases and neutrophil extracellular traps (NETs), which have been implicated in stabilizing fibrin matrices and rendering casts resistant to mechanical disruption in other vascular contexts (6). Although specific immunohistochemical staining for NET markers was not performed, representing a limitation of this report, the histologic findings provide a plausible substrate for this mechanism, which warrants validation in future studies employing dedicated NET detection methodologies.

The management of PB requires a tailored approach based on cast pathology and clinical context. Ntiamoah et al. (4) proposed a diagnostic and therapeutic algorithm that emphasizes supportive care, bronchoscopic extraction, and targeted pharmacologic interventions according to cast type. For inflammatory (Type 1) casts, inhaled corticosteroids and bronchodilators may be beneficial, particularly in patients with underlying airway hyperresponsiveness or allergic predisposition. In the present case, the combination of budesonide and salbutamol was employed as adjunctive therapy to address both the inflammatory component of cast formation and potential bronchospastic elements related to the patient's smoking history. This approach was consistent with the observation that conservative medical management, including inhaled therapies, provides symptomatic relief and facilitates cast resolution in Type 1 PB (4,9).

Flexible bronchoscopy remains essential for diagnosis, enabling direct visualization and therapeutic removal of casts, although repeated procedures may be required to achieve and maintain airway patency (7,8). Mechanical extraction using forceps, cryoprobes, or snares, combined with segmental saline lavage, represents a pragmatic first-line strategy when systemic hemorrhagic risk precludes pharmacologic intervention. Intrabronchial fibrinolytic agents have demonstrated benefit in selected case reports, particularly in pediatric populations, but carry nontrivial hemorrhagic risk and are generally reserved for patients in whom bleeding risk is acceptably low (2,10). When systemic bleeding risk is present, targeted hemostatic correction guided by viscoelastic testing may reduce ongoing intrabronchial fibrin formation and support bronchoscopic clearance efforts, as observed in this case.

Clinicians should consider PB in the differential diagnosis of adults with refractory lobar collapse or unexplained hypoxemia following massive hemothorax or chest trauma. The present case highlights several key management principles: prompt bronchoscopic evaluation, serial mechanical clearance as needed, adjunctive inhaled anti-inflammatory therapy where appropriate, and correction of systemic coagulation abnormalities with the aid of viscoelastic testing. Deferring fibrinolytic therapy was considered safe and potentially advisable in the setting of ongoing bleeding risk, provided that mechanical clearance was thorough, hemostasis was optimized, and anti-inflammatory therapy was instituted. Future studies should systematically measure airway fibrinolytic activity and inflammatory mediators, including NET components, to better define the molecular mechanisms underlying cast formation and persistence.

Limitations

This is a single case report and cannot establish causality. Owing to technological and equipment-related constraints, airway fibrinolytic biomarker measurements and dedicated lymphatic imaging were not performed. Specific immunohistochemical staining for NET markers, such as citrullinated histone H3 and myeloperoxidase, was also not performed. Consequently, the proposed NET-mediated mechanism remains speculative despite the dense neutrophilic infiltrate observed histologically.

CONCLUSION

This case demonstrates that Type 1 fibrinous plastic bronchitis may complicate massive intrathoracic hemorrhage and hemorrhagic shock in adults. A pathophysiologic triad—airway epithelial injury with blood exposure, a local procoagulant microenvironment, and neutrophil-driven scaffold formation—likely underlay cast formation in this case. Early bronchoscopic diagnosis, repeated mechanical clearance, adjunctive inhaled corticosteroid and bronchodilator therapy, and targeted correction of systemic coagulopathy represent effective management strategies and may obviate the need for fibrinolytic thera-

py when bleeding risk is elevated. Increased awareness of this rare complication following hemorrhagic events may facilitate prompt diagnosis and improve patient outcomes.

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CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - S.W., J.C., M.W.; Planning and Design - S.W., J.C., M.W.; Supervision - S.W., J.C., M.W.; Funding - J.C., M.W.; Materials - J.C., M.W.; Data Collection and/or Processing - S.W., J.C., M.W.; Analysis and/or Interpretation - S.W., J.C., M.W.; Literature Review - S.W., J.C.; Writing - S.W., J.C.; Critical Review - S.W., J.C., M.W.

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