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Case Report

Life-Threatening Iatrogenic Pulmonary Hemorrhage Managed with Salvage Anticoagulation-Free Venovenous Extracorporeal Membrane Oxygenation

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SEVERE HEMOPTYSIS REMAINS A significant medical emergency, often requiring a multidisciplinary approach to identify the bleeding source and implement effective treatment. Airway obstruction due to clots secondary to massive hemoptysis is a life-threatening complication, and conventional methods of airway management may fail, necessitating the use of advanced techniques such as extracorporeal membrane oxygenation (ECMO). Recent literature supports the feasibility of venovenous (vv) ECMO as a bridge to definitive therapy in selected cases of refractory hemoptysis.[1]

Atrial fibrillation (AF) ablation, although effective for arrhythmia control, carries a risk of complications, including pulmonary vein stenosis (PVS), thrombosis, and, rarely, atriobronchial fistula. Such complications often occur directly after the procedure or up to 12 months later.[2] When AF ablation was first introduced, initial guidelines recommended routine computed tomography (CT) scan screening for PVS[3]; however, this practice has been largely abandoned over time,[4] owing to a reduction in complications. Overall, PVS occurs in 1.3% to 3.4% of all AF ablation procedures.[2] The intensity of PVS symptoms (dyspnea, chest pain, pleural effusion, hemoptysis, cough, and chronic pulmonary infection) depends on the timing of stenosis, severity of stenosis, number of affected veins, presence or absence of venous collateralization, and patient's physical activity level.[2,5,6] These symptoms are due to focal pulmonary hypertension and pulmonary infarction.[6] This report presents a unique case of life-threatening pulmonary hemorrhage following cryobiopsy of bronchial varices, mimicking neoplasia, secondary to left superior pulmonary vein (LSPV) stenosis and thrombosis occurring 2 years after AF ablation, managed with vvECMO and ultimately necessitating left upper lobectomy.

Case Presentation

This 52-year-old male was referred to the study center with a 2-year history of intermittent hemoptysis episodes with some

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long episode-free intervals. Hemoptysis episodes were mild (traces of blood in sputum every day for several weeks with blood-free intervals of several months). The patient was obese (body mass index, 42.6) at presentation and had a previous history of mixed altered lung function (forced expiratory volume at 1 second, 48% of predicted value), non−ST elevation myocardial infarction (NSTEMI) with preserved ejection fraction, and AF radiofrequency ablation followed by therapeutic anticoagulation. These episodes of hemoptysis became more frequent and severe, justifying medical consultation, although he was free of symptoms following an NSTEMI acute coronary syndrome at 6 months before admission and for which dual antiplatelet therapy was initiated.

The initial workup showed pulmonary nodules. Despite extensive outpatient evaluations, including noncontrast CT scan, positron emission tomography-CT, and fiberoptic bronchoscopy assessments, the source of bleeding remained unidentified, and previously identified pulmonary nodules had disappeared. Bronchoscopy revealed morphologic abnormalities (cobblestone appearance of the bronchial mucosa and an important submucosal vascular lattice; Fig 1) in the lower part of the left stem bronchus and the left bronchial division.

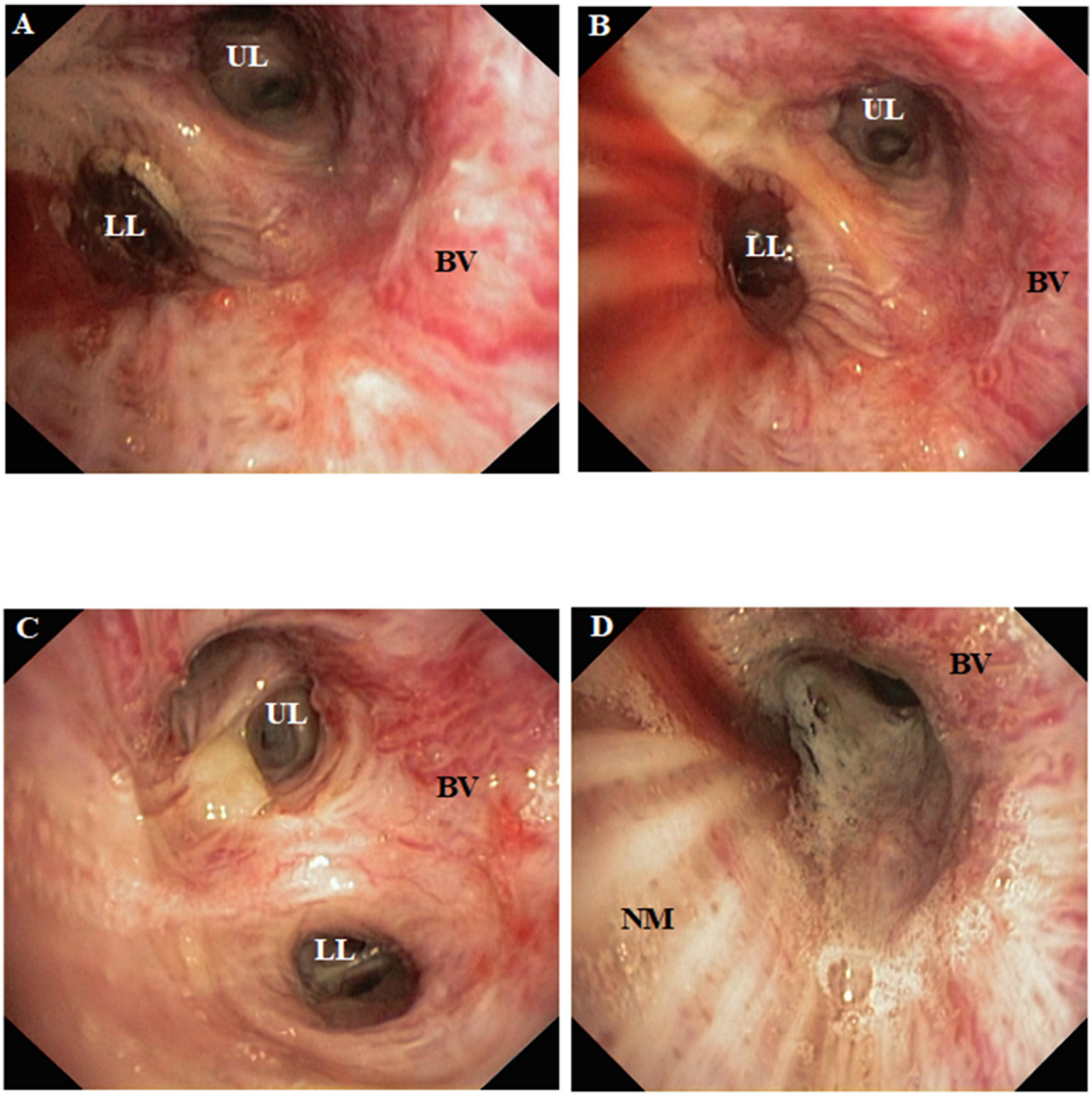

Fig 1. Four endoscopic views of the left main bronchus in the division of the upper lobe (UL) and lower lobe (LL). These images show both left lobe orifices, abnormal mucosa with cobblestone appearance and vascular lattice (BV), and minor bleeding seemingly originating from the lower lobe bronchus. (A, B and C) Endoscopic view of the lower part of the main left stem bronchus and the first bronchial division of the left lung into the upper lobe and lower lobe. Endoscopic images shows the upper left quadrant of the bronchial mucosa, where a reddish region indicates the presence of bronchial varices (BV) or vascular abnormalities. Prominent, dilated submucosal vessels are visible, suggestive of vascular congestion. The mucosal surface exhibits a characteristic cobblestone pattern consistent with these changes. These abnormalities appear to originate from the orifice of the left UL. Minor bleeding can be seen in (A) and (B), seemingly originating from the left LL. (D) Endoscopic view of the left main bronchus in a proximal view showing dissemination of the vascular lattice onto the left main bronchus. NM, normal mucosa; BV, bronchial varices.

Owing to the unavailability of the technique locally, the patient was transferred as an outpatient to a university institution to perform cryobiopsies for concern of malignant lesions. The initial laboratory workup showed a normal coagulation profile as well as a normal hemoglobin level (13.9 g/dL).

During the procedure, the patient developed massive intrabronchial bleeding originating from the biopsy site. He was promptly intubated in the operating room and then transferred to the intensive care unit (ICU) for further management. In the ICU, a bronchial blocker was deployed in the left main bronchus to prevent contralateral lung flooding; however, persistent bleeding led to near-complete airway obstruction by clots (Fig 2). Adequate ventilation of the patient became subsequently impossible, resulting in oxygen saturation dropping to 50% and necessitating emergent vvECMO implantation (Fig 3) with cannulation in the femorojugular conformation using 21 Fr and 25 Fr Medtronic cannulas and a 1.81 m^2 A.L. ONE ECMO oxygenator. The circuit was primed without anticoagulation, and the vvECMO ran without anticoagulation therapy. Histology of the biopsy specimens remained inconclusive but did not demonstrate active neoplasia.

The course of vvECMO was complicated by multiple episodes of ventilator-associated pneumonia caused by *Serratia marcescens*, which were treated with broad-spectrum antibiotics. In addition, the oxygenator membrane experienced severe and repeated dysfunction, demonstrated by low postmembrane arterial partial pressure of oxygen (PaO_2), owing to the high flow rate (6.3 L/minute) required to adequately oxygenate this morbidly obese patient with high cardiac output. This necessitated several oxygenator and total circuit replacements and ultimately was managed by the implantation of dual oxygenators in parallel (Fig 4), which significantly improved membrane performance and durability. This approach allowed successful maintenance of vvECMO for a total duration of 5 weeks despite the absence of anticoagulation therapy.

During this 5-week course of vvECMO, repeated bronchoscopies were performed in an attempt to remove obstructive clots; however, bleeding persisted despite these measures. Bronchial embolization was attempted but proved infeasible owing to an inability to catheterize the target vessels and to concerns about inadvertent embolization of nontarget arteries supplying the spinal cord. Contrast-enhanced CT raised the suspicion of LSPV stenosis and thrombosis as complications of the prior AF ablation (Fig 5). A review of the preablation magnetic resonance imaging scans which revealed patent pulmonary veins. This suspicion of LSPV thrombosis was confirmed at day 22 after ICU admission by angiopneumographic imaging with direct catheterization of the left pulmonary artery showing abnormal vascularization of the left upper lobe (very thin left superior pulmonary artery and vein, confirming LSPV

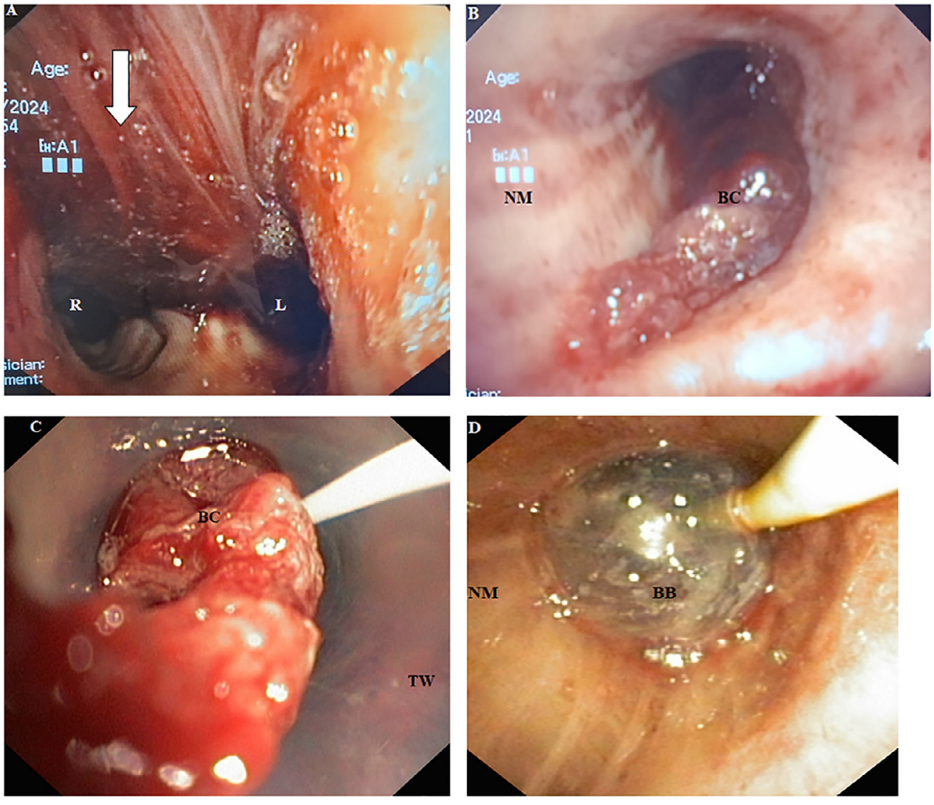

Fig 2. Endoscopic view of near-complete airway obstruction due to massive clotting and bronchial blockers in place. (A) Endoscopic view of the lower part of the trachea and carina (first bronchial division) showing a blot clot through both the left (L) and right (R) bronchi and posterior wall of the trachea. The *arrow* indicates the posterior wall of the trachea with a blood clot. (B) Endoscopic view of the left main bronchus with a blood clot completely obstructing the light of the left main bronchus. NM, normal mucosa; BC, blood clot. (C) Endoscopic view of the orotracheal tube with a blood clot completely obstructing its light. TW, tube wall; BC, blood clot. (D) Endoscopic view of the left main bronchus with the bronchial blocker (BB) in place. NM, normal mucosa.

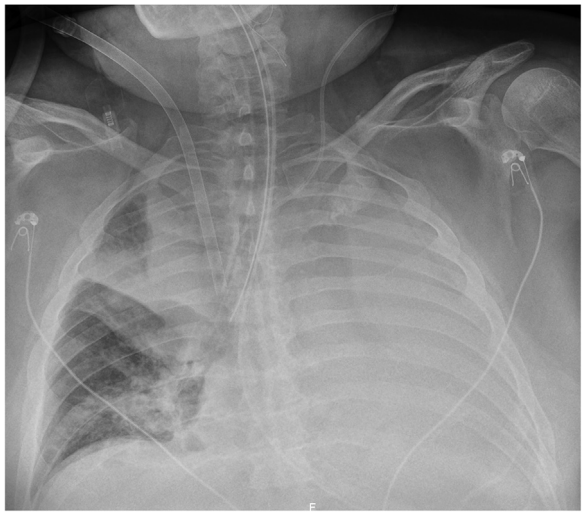

Fig 3. Chest X-ray showing selective intubation, venovenous extracorporeal membrane oxygenation cannulas in the right internal jugular vein, and diffuse atelectasis of the right lung multiples lobes and complete atelectasis of the left lung.

thrombosis), as well as a completely condensed left upper pulmonary lobe (Fig 6). A transesophageal examination analyzed the LSPV at 1.8 mm from the arteriovenous junction. The diameter of the LSPV at its arteriovenous junction was 12.4 mm, and the flow was laminar with a maximum velocity of 0.62 m/s, which did not raise a suspicion of stenosis at that level. The endobronchial mucosal abnormalities observed at the end of the left main bronchus were attributed to mucosal venous congestion caused by venous pulmonary hypertension due to impaired venous drainage of the left superior pulmonary artery.

Fig 4. Venovenous extracorporeal membrane oxygenation circuit showing parallel dual oxygenators (*arrows*).

After a multidisciplinary approach, the patient underwent surgical left upper lobectomy to control the source of bleeding. The major preoperative concern was the risk of left pneumonectomy in the event of perioperative refractory bleeding in this patient with prior severely reduced lung function. In the ICU, postoperative management was complicated by hemorrhagic shock due to ECMO-induced coagulopathy and platelet dysfunction.[7] The patient's condition rapidly improved after surgery, and the final fiberoptic bronchoscopies showed complete regression of vascular abnormalities (Fig 7), confirming the diagnosis of hemoptysis-induced by LSPV stenosis, as well as the absence of residual bleeding. This allowed for vvECMO removal at 5 weeks after cannulation and 3 days after surgery. Owing to obesity, the patient remained mildly hypercapnic but was successfully extubated 11 days after surgery. Extubation was delayed primarily because of a drug-induced prolonged coma; however, he was ultimately discharged from the ICU in stable condition 62 days after admission.

Discussion

This case highlights the complexity of managing life-threatening hemoptysis with airway compromise. Massive intrabronchial bleeding poses unique challenges, as traditional airway control techniques may fail in the presence of clot formation and persistent hemorrhage. In this scenario, vvECMO served as a life-saving measure by providing adequate oxygenation and decarboxylation while definitive diagnosis and treatment were pursued.

The feasibility of vvECMO in cases of severe hemoptysis has been described in the literature through isolated case reports, but its application remains limited by the need for specialized expertise and equipment, as well as the potential for complications such as coagulopathy and membrane dysfunction, as were encountered in this patient. Nevertheless, vvECMO even in the absence of anticoagulation is a promising approach,[8,9] contrary to American Thoracic Society and Extracorporeal Life Support Organization guidelines,[7,10,11] which do not recommend vvECMO use in the event of contraindications for anticoagulant therapy. However, the emerging literature tends to support the safety of limited or no anticoagulation therapy during vvECMO, and this case further demonstrates the feasibility and safety of this approach.[12] In this morbidly obese patient, repeated and severe membrane dysfunction was more likely related to the high blood flow required to ensure adequate oxygenation rather than to membrane microthromboses resulting from the absence of anticoagulation. This membrane dysfunction was attributed to a shunt effect within the ECMO circuit, with the high flow through the oxygenators reducing transit time within the membrane, resulting in insufficient oxygenation. This phenomenon is favored by the use of a small surface membrane (1.81 m^2; A.L.ONE ECMO oxygenator), chosen to reduce activation of the inflammatory response and the coagulation cascade. This situation

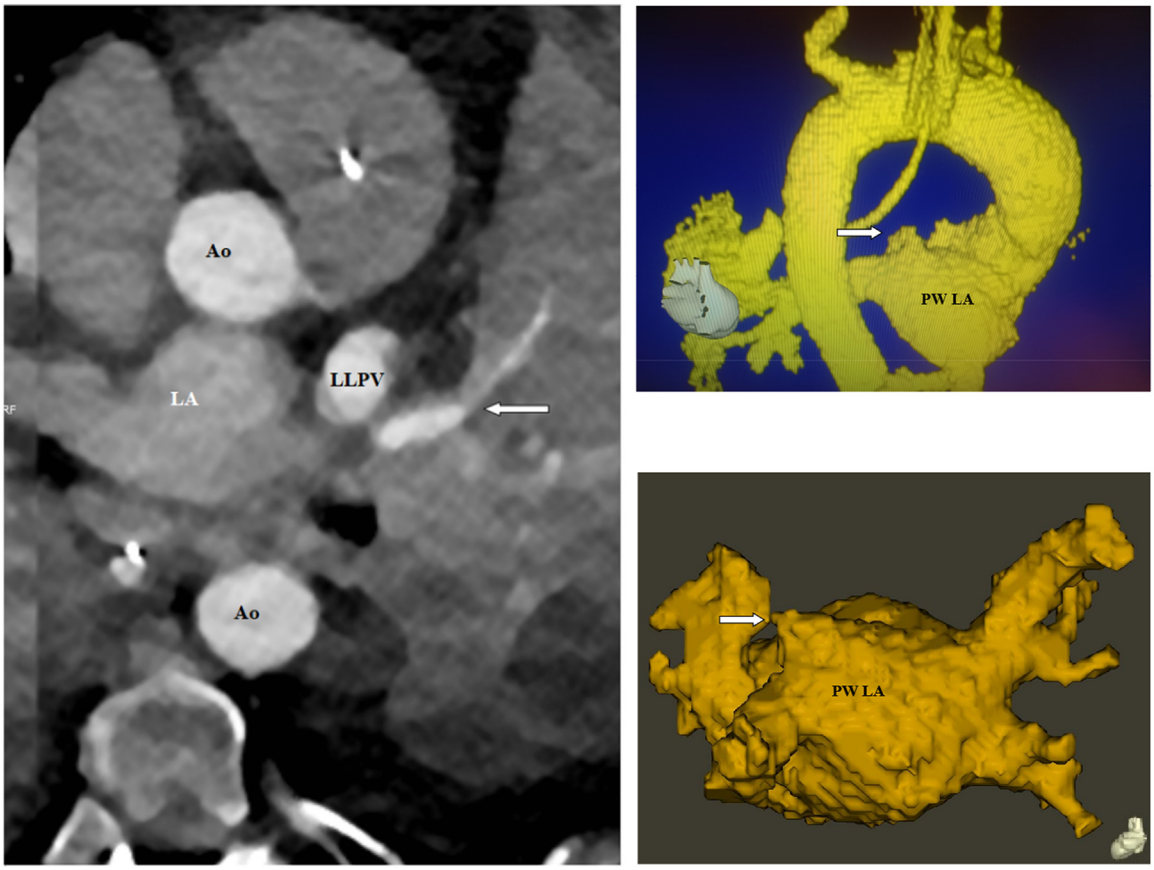

Fig 5. (A) Contrast-enhanced computed tomography scan with pulmonary artery catheterization showing upper left pulmonary vein stenosis (*arrow*). Ao, aorta; LA, left atrium; LLPV, left lower pulmonary vein. (B) 3-dimensional reconstruction of the posterior wall of the left atrium (PW LA) showing absence of the left upper pulmonary vein (*arrow*).

was managed by changing the circuit and adding 2 oxygenators (surface of 1.2 m^2 each; Liva Nova, EOS) in parallel to increase transit time and gas exchange, allowing for adequate postmembrane PaO_2 and prolonged membrane efficacy. During the use of dual parallel oxygenators (a period of 4 weeks and 3 days), flow measurements in each oxygenator showed approximately 50% of the total ECMO blood flow per oxygenator. This situation has been described in several case reports of obese patients during the COVID-19 pandemic.[13] Therefore, in such cases (a morbidly obese patient with high cardiac output), in which maintaining high ECMO blood flow is crucial to allow sufficient organ oxygenation, the use of dual oxygenators might be an option to prevent dysfunction of the ECMO membrane, offering a simpler solution compared to the use of dual parallel circuits.[14]

Complications of AF ablation, particularly PVS and thrombosis, are rare but potentially devastating. PVS typically manifests within 1 year after the procedure, which may lead to a delay in diagnosis. In a retrospective study assessing risk factors for secondary PVS, Samuel et al[15] found that it might be favorized by diabetes mellitus. In the present patient, the prolonged onset of symptoms could be attributed to his low level of physical activity, as symptom intensity is often correlated with physical exertion.[2] This lack of activity might explain the gap between the patient's acute coronary syndrome and the development of hemoptysis, as he had ceased all physical activity for 6 months before admission owing to his acute coronary syndrome.

The cobblestone appearance and vascular abnormalities observed during fiberoptic bronchoscopy (Fig 1) were similar to those described by Matsumoto et al[16] and raised a concern for neoplastic etiology. Ultimately, these morphologic changes were attributed to congestive mucosa caused by obstruction of the LSPV, known as bronchial varices. Cryobiopsies performed in this area resulted in massive and repeated bleeding due to the elevated venous pressure.

Bronchoscopic features of this pathology (presence of cobblestone appearance altogether with abnormal vascularity and suspected dilated submucosal vein) are not well characterized but should alert the clinician to a history of previous AF ablation. The cornerstone of the diagnosis relies on pulmonary angiography with 3-dimensional reconstruction,[5] which was performed in the present patient through direct pulmonary artery catheterization allowing visualization of pulmonary arterial and venous perfusion. Additionally, transesophageal examination of the pulmonary veins could detect PVS induced by AF ablation with high sensitivity and specificity, using a pulmonary vein peak velocity cutoff of >1 m/s,[17] which was not the case in this patient owing to more distal stenosis (as shown on Fig 6), complicating the diagnosis. A multidisciplinary approach, involving pulmonologists, intensivists,

Fig 6. Contrast-enhanced computed tomography scan with pulmonary artery (PA) catheterization showing a condensed left upper pulmonary lobe (UPL) with a very thin left upper PA (*arrow*). The right lung (RL) shows diffuse pulmonary infiltrates.

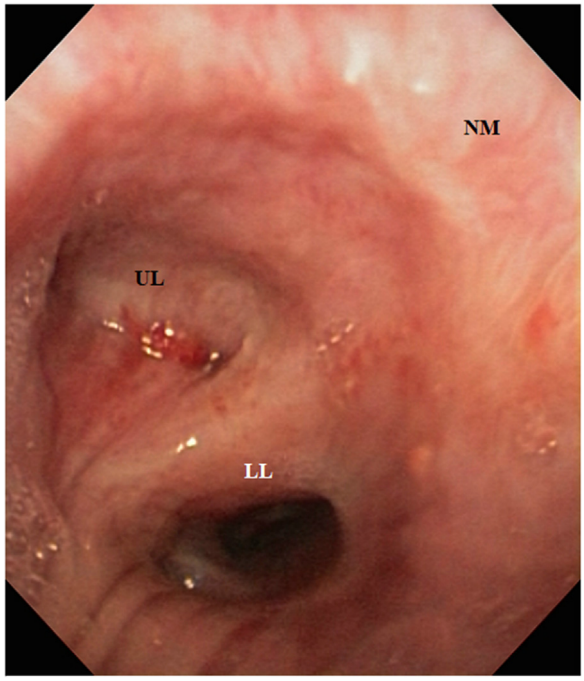

Fig 7. Endocsopic view of the last fiberoptic bronchoscopy (similar view as in Fig 1) showing the suture of the upper left pulmonary bronchus and regression of vascular abnormalities where the vascular lattice was formerly seen. UL, upper lobe; LL, lower lobe; NM, normal mucosa.

cardiologists, and thoracic surgeons, is essential for accurate diagnosis and successful management.

There is no consensus in the literature regarding the definitive management of such cases among such techniques as pulmonary vein stenting,[4,18-20] dilation,[4,19] and oral sirolimus.[4,21] However, conservative treatment is associated with a high frequency of restenosis[22] and thus should be reserved for cases of acute-onset PVS. Bronchial embolization has been described in several case reports,[16,21] with ineffective results owing to the mechanism of bleeding. Chronic PVS and total pulmonary vein occlusion may cause irreversible intrapulmonary vascular changes. The intimal hyperplasia and medial thickening of both pulmonary veins and arteries lead to pulmonary vascular remodeling, which can result in severe pulmonary hypertension.[5,23] Resection of the involved lung or lobe may be more appropriate in this clinical scenario.[24,25]

This case report also illustrates the challenges and complexities of managing hemoptysis and achieving a definitive diagnosis, particularly when caused by rare complications such as PVS following AF ablation. This may lead to unnecessary investigation, resulting in catastrophic complications, high cost and resource utilization. The case emphasizes the need for multidisciplinary collaboration, particularly among radiologists, pulmonologists, and cardiologists, for achieving a successful diagnosis, treatment, and outcome. PVS is often underrecognized by pulmonologists, who typically are the first to encounter these patients presenting with pulmonary

symptoms. Thus, PVS should be suspected in cases of unusual pulmonary symptoms and endoscopic findings, especially with hemoptysis, following prior AF ablation—even with late-onset symptoms.

This appears to be the first reported case of life-threatening iatrogenic pulmonary hemorrhage following cryobyopsies due to bronchial varices secondary to AF ablation managed by prolonged anticoagulation-free vvECMO. Unlike the case reported by Ren et al,[26] here vvECMO was initiated preemptively before thoracic surgery to prevent complications. This report also contributes to the growing body of evidence supporting vvECMO as a viable option for salvage therapy in severe hemoptysis even in the absence of anticoagulation therapy.

Conclusion

This case highlights the complex diagnosis and management of life-threatening pulmonary hemorrhage with airway compromise in an obese patient, necessitating prolonged anticoagulation-free vvECMO and dual oxygenators. It underscores the importance of multidisciplinary collaboration, particularly among radiologists, pulmonologists, and cardiologists, in recognizing secondary PVS after AF ablation. Further research is needed to refine protocols for vvECMO use in severe hemoptysis and to better understand long-term risks of AF ablation.

Declaration of competing interest

All authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Aurélien Gonze: Writing – review & editing, Writing – original draft, Validation, Supervision, Methodology, Investigation, Data curation, Conceptualization. **Thomas Neff:** Writing – review & editing, Validation, Supervision, Investigation. **Sebahat Ocak:** Writing – review & editing, Validation, Supervision. **Benoit Robaye:** Writing – review & editing, Validation, Methodology, Investigation. **Benoit Rondelet:** Supervision. **Martial Gillet:** Investigation. **Pierre Bulpa:** Writing – review & editing, Validation, Supervision, Methodology, Investigation, Conceptualization. **Isabelle Michaux:** Writing – review & editing, Validation, Supervision, Methodology, Conceptualization. **Arnaud Robert:** Writing – review & editing, Writing – original draft, Validation, Supervision, Methodology, Investigation, Data curation, Conceptualization.

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