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Catheter-based Endovascular Angioplasty for Fibrosing Mediastinitis-associated Pulmonary Vein Stenosis

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Abstract

Fibrosing mediastinitis (FM) is a rare but severe disease characterized by abnormal proliferation of fibrous tissue within the mediastinum. Pulmonary vein stenosis (PVS) is a life-threatening complication of FM, with a poor prognosis if not properly treated. Currently, catheter-based endovascular angioplasty is an alternative for patients suffering from PVS and has demonstrated significant short-term efficacy. The main steps of this technique include evaluating the structures involved in the mediastinum *via* computed tomography pulmonary angiography (CTPA), establishing transseptal access, performing selective pulmonary venography, conducting balloon angioplasty and stent implantation for the pulmonary veins, monitoring hemodynamic parameters before, during, and after endovascular angioplasty, and providing post-intervention care and routine follow-up. This study discusses the formulation of interventional strategies based on the characteristics of compressed pulmonary vessels and adjacent structures in CTPA, as well as the possible periprocedural complications and corresponding management strategies. This article primarily presents a step-by-step demonstration of catheter-based endovascular angioplasty.

Introduction

Fibrosing mediastinitis (FM), also known as sclerosing mediastinitis and mediastinal fibrosis, is a rare yet severe disease characterized by abnormal proliferation of fibrous tissue within the mediastinum[1,2]. The diagnosis of FM is challenging due to its insidious onset and nonspecific symptoms, which may include fatigue, dyspnea, wheezing, chest pain, cough, or hemoptysis, depending on the anatomic structures involved[2,3]. Thus, definitive diagnosis is typically delayed, and misdiagnosis is frequent. As the disease progresses, compression from fibrous tissue can lead to pulmonary vein stenosis (PVS) or occlusion, eventually resulting in recurrent episodes of pleural effusion, pulmonary

hypertension (PH), right heart failure, or even death[3,4]. Therefore, early diagnosis is of paramount importance for patients with FM-associated PVS[5].

To date, computed tomography pulmonary angiography (CTPA) is the primary noninvasive diagnostic approach for PVS and can be utilized to evaluate pulmonary vein (PV) anatomy, lesion characteristics, and comorbidities[6]. With the aid of CTPA, the number of involved pulmonary veins (PVs) can be characterized, the etiology of PVS can be determined, the location of the stenosis or occlusion can be identified, and the residual diameter of the most stenotic segment can be measured[5]. Furthermore, three-dimensional volume-rendered (3-D VR) CTPA reconstructions can separate the pure vascular component of anatomical structures from adjacent osseous and lung abnormalities[6], thereby enhancing visualization quality. Based on the involvement of pulmonary vessels, the trachea, and bronchi in the mediastinum, FM can be classified into three subcategories: arterial, venous, and mixed[7]. These are also referred to as type I, type II, and type III[4]. Type I (arterial) FM indicates stenosis of the pulmonary arteries (PAs) but not the PVs and is typically accompanied by stenosis of anatomically adjacent bronchi. Type II (venous) FM indicates stenosis of the PVs without involvement of the PAs, and bronchial involvement is uncommon. Type III (mixed) FM involves stenosis of the PAs, PVs, and bronchi.

Currently, drug therapy for FM-associated PVS is palliative and has limited efficacy, while surgical treatment is associated with a significant risk of mortality[8]. As an emerging technique, catheter-based intervention serves as a viable alternative for patients suffering from PVS; timely angioplasty effectively alleviates obstruction, thereby normalizing venous flow into the left atrium and yielding significant improvements in outcomes based on studies with small sample sizes[3,9]. However, formulating treatment strategies for FM-associated PVS is challenging and requires consideration of the holistic functioning of both PAs and PVs[7], as well as the involvement of adjacent structures such as the lung parenchyma, bronchi, esophagus, and pericardium. Additionally, comorbidities, the patient's clinical history, and their current condition must be taken into account. Generally, for mixed FM, it is recommended to prioritize PV intervention[7]. Once the stenosed PV has been successfully reopened, PA intervention can be considered promptly. However, if the occlusion of the PV cannot be resolved, PA intervention should be avoided. Similarly, if it is anticipated that the occlusion of the PA cannot be alleviated, PV intervention should not be pursued. Based on previous treatment experiences[9,10,11], the endovascular approach is recommended for PVS caused by FM (PVS-FM).

However, owing to the complexity of treatment strategies and the absence of standardization for interventional procedures, the clinical application of this technique is limited. This study elucidates a stepwise approach for the transcatheter procedure in a patient with FM-associated PVS. The patient was a 77-year-old man who presented with exertional dyspnea and recurrent episodes of unilateral pleural effusion for 3 years. His past medical history was unremarkable, with no evidence of cardiovascular disease, infectious disease, immune system disorders, or a history of radiotherapy exposure or chest surgery. CTPA was performed at the time of admission, and the patient was diagnosed with mixed FM (type III). He had been undergoing drug therapy, but it had shown limited efficacy. Owing to the high mortality risk associated with surgery and the ineffectiveness of drug therapy, as well as the patient's preference for minimally invasive procedures, we planned

to perform transcatheter pulmonary vessel angioplasty to alleviate symptoms. Considering the involvement of PVs, PAs, bronchi, and recurrent episodes of pleural effusion, as well as the possibility of recanalization of the stenotic PA, a therapeutic strategy for PV intervention was first developed by the intervention team.

Protocol

The procedures involving human subjects were approved by the ethics committee of Sichuan Provincial People's Hospital. Patient consent was obtained to include anonymized data for the protocol and video publication. The inclusion criterion was patients who were diagnosed with PVS-FM through CTPA, met the indications for interventional therapy, and were scheduled to undergo PV intervention. The exclusion criteria were as follows: patients with end-stage disease, active bleeding, expected intolerance to long-term antithrombotic therapy, or allergies to contrast agents. Details of the reagents and equipment used in this study are listed in the **Table of Materials**.

1. CTPA evaluation

NOTE: CTPA assessment is crucial for establishing an accurate diagnosis and formulating an effective intervention strategy for patients.

1. Evaluate the lung parenchyma, trachea, bronchi, pulmonary vessels, and heart using CTPA prior to the interventional procedure.
2. Examine the CTPA images (**Figure 1A**) to identify proliferative fibrous tissues in the pulmonary hila, compressing the pulmonary veins, pulmonary arteries, and bronchi, resulting in stenoses of the pulmonary vessels, which indicate the mixed FM.
3. Observe the 3-D VR image (**Figure 1B**) to assess severe stenoses in the ~~proximal right~~ superior pulmonary vein (~~R~~SPV) and the ~~proximal left~~ superior pulmonary vein (~~L~~SPV).

2. Monitoring vital signs and preparing sterile equipment

1. Assist the patient in lying down on his back, connect the monitoring equipment, and record the ECG, heart rate, peripheral oxygen saturation, and blood pressure.
2. Establish a peripheral venous access line and connect it to 500 mL of normal saline.
3. Prepare rescue medications, including adrenaline, norepinephrine, atropine, dopamine, lidocaine, nitroglycerin, and vasopressin, as well as rescue equipment such as the tracheal intubation kit, pericardiocentesis kit, and defibrillator device. Ensure cardiovascular ultrasound is available for procedural assistance if needed.
4. Prepare sterile equipment with the materials required by the catheter assistant or nurse.

   NOTE: The main equipment needed includes the Judkins Right/Left (JR/JL) Guiding Catheter, Transseptal Guiding Introducer, Transseptal Needle, Catheter Sheath Introducer, Balloon Dilatation Catheter, Vascular Premounted Stent System, Guide Wire, and Everest Inflation Device.
5. Flush all the catheters with heparinized saline to prevent thromboembolic complications.

3. Establishing femoral vein access and performing selective right inferior pulmonary artery (RIPA) angiography

NOTE: Risk may increase due to left atrial atrophy in patients with PVS, and routine RIPA angiography is performed to facilitate the localization of adjacent structures and reduce complications.

1. Sterilize the right groin using a povidone-iodine solution. Administer 3-5 mL of lidocaine subcutaneously above the puncture site in the groin. Access the right femoral vein using palpation or ultrasound guidance.
2. Insert an 8 French catheter sheath into the right femoral vein.
3. Insert a 0.035-inch guide-wire and deliver the guiding catheter to the ~~RIPA~~ through femoral vein access *via* the guide-wire.
4. Withdraw the guide-wire and perform selective ~~RIPA~~ angiography to establish the location of the left atrium in the 45° right anterior oblique (RAO) view. Observe the initial site of contrast agent arrival at the ~~RIPA~~, followed by the right ~~inferior~~ pulmonary vein (~~RIPV~~), left atrium, left ventricle, and ascending aorta.
5. Pull out the JR guiding catheter.

4. Transseptal procedure

1. Insert a 0.032-inch "J" tip guide-wire into the superior vena cava (SVC) and remove the 8 French catheter sheath.
2. Advance the transseptal guiding sheath/dilator assembly into the SVC over the "J" tip guide-wire under anterior-posterior (AP) fluoroscopy.
3. Remove the guide-wire from the dilator.
4. Position the transseptal needle and stylet assembly until the stylet is positioned just proximal to the dilator tip inside the sheath.
5. Remove the stylet.
6. Adjust the pointer flange so that the needle is positioned perpendicular to the fossa ovalis in the anteroposterior (AP) view, typically between 3:00 and 5:00 o'clock, as seen from the foot end of the patient.
7. Verify that the needle tip is within the dilator using fluoroscopy. Gradually retract the entire sheath/dilator/needle assembly. Monitor the dilator tip for any sudden medial (or rightward) movement during retraction, which would indicate engagement with the fossa ovalis.
   NOTE: Ensure that the assembly parts remain stationary relative to each other. Maintain the initial orientation of the pointer flange while retracting the assembly.
8. Visualize and identify anatomic landmarks in the 45° RAO view.
9. Extend the needle to full engagement within the sheath/dilator assembly and advance it across the interatrial septum. Ensure the blood is arterial blood.
   NOTE: If resistance is encountered during needle advancement, retract the needle and reassess the anatomical landmarks. In the event of pericardial or aortic entry, do not advance the dilator over the needle. If the needle has punctured the pericardium or aorta, withdraw it immediately. Continuously monitor vital signs.
10. Confirm left atrial access *via* fluoroscopy with contrast injections.
    NOTE: Under pressure monitoring, confirm left atrial entry when the pressure tracing shows a left atrial pressure waveform.

11. Maintain a fixed needle position within the left atrium and advance the sheath/dilator assembly fully over the needle into the left atrial cavity.
12. Remove the transseptal needle from the dilator.
    NOTE: Air infiltration may occur when removing objects from the hemostasis valve of the sheath. Withdraw objects gradually to avoid vacuum formation in the sheath and monitor the sheath fluoroscopically for the presence of air during device insertion.
13. Insert the 0.035-inch guide-wire and advance it into the LSPV.
14. Advance the sheath fully into the left atrial cavity over the dilator, and then withdraw it.
15. Insert the guiding catheter and advance it into the left atrium over the guide-wire. Withdraw the guide-wire.
16. Measure the left atrial pressure *via* the guiding catheter.

5. Selective pulmonary venography

NOTE: Maintain the activated clotting time within an appropriate range of 250-350 s to avoid thromboembolic complications.

1. Advance a 0.014-inch Run-through guide-wire into the distal LSPV.
2. Gently pass the guiding catheter over the wire, positioning its tip at the distal part of the stenosis.
3. Monitor changes in pressure. An increase in pressure compared to the left atrial pressure indicates that the guiding catheter is either at or has passed through the narrow lesion.
4. Perform selective pulmonary venography to accurately evaluate the lesion's location, the severity of stenosis, and the grade of pulmonary vein flow.
5. Record pressure measurements of the distal part of the stenosis.
6. Retract the JR guiding catheter to the proximal part to measure the proximal pressure and calculate the pressure gradient (PG) ~~between the two sides of~~ the lesion.

6. Balloon angioplasty and stent implantation for pulmonary veins

NOTE: Ensure the ~~guide wire~~ is in the correct position *via* venography to avoid vessel dissection, perforation, or ~~mistakenly entering~~ the pericardium. Measuring the pressure gradient (PG) across the lesion is crucial for assessing hemodynamic changes before and after balloon or stent angioplasty. Additionally, peripheral oxygen levels, cough response, and hemoptysis ~~must~~ be monitored during the procedure for the timely ~~discovery~~ of complications.

1. Choose an appropriately sized balloon based on the CTPA and pulmonary venography data, and ~~perform sequential dilation at~~ the most stenotic part of the dedicated vein.
2. Remove the balloon delivery catheter.
3. Repeat pulmonary venography and pressure measurement. If there are no significant improvements in PG, hemodynamics, or the degree of stenosis, indicating limited effects or acute recoil, ~~perform~~ further angioplasty ~~with~~ a stent.
4. Select an appropriately sized stent based on the characteristics assessed ~~by~~ imaging tools.
5. ~~Exchange the guide wire with a 0.035-inch angled guide wire.~~
6. ~~Withdraw~~ the guiding catheter.

7. Advance the stent over the guide-wire into the dedicated vein and perform the stenting procedure. Establish the stent location *via* anatomic landmarks.

8. Reinsert the guiding catheter and perform pulmonary venography and pressure measurements repeatedly. If images reveal inadequate stent expansion and the PG is greater than 5 mmHg, perform post-dilation of the stent.

9. Deliver a suitably sized balloon and perform in-stent dilation.

10. At the end of the procedure, perform the final venography to rule out any oozing or perforation and re-evaluate the PG. If the PG is less than 5 mmHg, the procedure is successful.

11. Insert the guiding catheter and advance it into the proximal right superior pulmonary vein (RSPV), then perform the interventional procedures following the aforementioned steps.

12. Remove the guide wire and transseptal sheath.

13. Press the puncture site manually and apply a tight pressure bandage to prevent local bleeding.

7. Postintervention care

1. Continuously monitor vital signs, including blood oxygen saturation, to detect post-procedural complications in a timely manner.

2. Recommend novel oral anticoagulants or vitamin K antagonists for 6-12 months, and aspirin (100 mg/d) or clopidogrel (75 mg/d) for at least 3-6 months for antithrombotic therapy.

3. Perform regular follow-ups and monitor for possible intrastent restenosis (ISR) using CTPA.

Representative Results

In this study, catheter-based endovascular angioplasty was successfully performed on a patient with FM-associated PVS. Improvements were observed in the two stenotic vessels after the endovascular angioplasty, including the achievement of patency of the dedicated veins with a flow grade of 3 and a decrease in the pressure gradient (PG) across the stenoses. Hemodynamic parameters before and after endovascular angioplasty are presented in **Table 1**. Symptoms of dyspnea were relieved upon discharge, accompanied by an improvement in exercise capacity. Long-term follow-up is necessary to assess for intrastent restenosis.

In patients with FM-associated PVS, short-term efficacy was observed after interventional treatment, with improvements in hemodynamics, WHO functional class, 6-min walking distance, mean pulmonary arterial pressure, refractory pleural effusion, and clinical symptoms[8,9,12]. Since 1980, Massumi et al.[12] successfully performed the first catheter-based treatment for a 32-year-old woman diagnosed with severe PH caused by significant stenosis of PVs, and an increasing number of catheter labs have started performing interventional procedures to relieve clinical symptoms in patients with PVS.

Periprocedural complications may occur with varying incidence rates across centers. Common complications, such as puncture site bleeding, hemoptysis, phrenic nerve injury, and transient ST segment elevation, generally do not require additional treatment[8]. The incidence of hemoptysis ranges from 16.1% to 20%, with the majority of cases being mild[8,9,13]. However, severe hemoptysis necessitates interventions[3]. Pericardial tamponade, PV

tear, PV perforation, severe pulmonary hemorrhage, hemothorax, stroke, and stent dislodgement are additional procedure-related risks that require immediate clinical management[3,8,13].

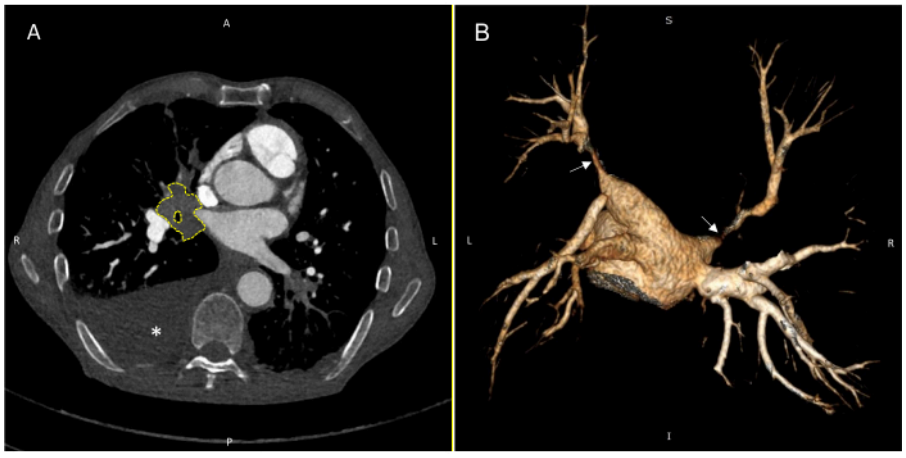

Figure 1: CTPA ~~of the mediastinal window~~. The CTPA image of the mediastinal window ~~on admission~~ shows hyperplastic soft tissue (dotted line in **A**) compressing the pulmonary vessels, resulting in pulmonary vein stenosis (PVS) (arrow in **B**). The asterisk in A indicates pleural effusion.CTPA: computed tomography pulmonary angiography; PVS: pulmonary vein stenosis. Please click here to view a larger version of this figure.

Interventional Process	LSPV		RSPV	
	the pressure of the distal part (mmHg)	the pressure of the proximal part (mmHg)	the pressure of the distal part (mmHg)	the pressure of the proximal part (mmHg)
pre-intervention	42	11	36	16
post-balloon dilation	43	13	35	16
post-stenting	23	13	14	13
post-dilation of the stent	15	14	/	/

Table 1: Hemodynamic parameters pre- and post-endovascular angioplasty. ~~LSPV: left superior pulmonary vein; RSPV: right superior pulmonary vein.~~

Discussion

The critical steps of the interventional procedure include ensuring safe transeptal access without complications, administering heparin to maintain an activated clotting time of approximately 250-350 s to prevent embolic events, utilizing hemodynamic monitoring to guide treatment strategies, and confirming proper placement of the guide-wire through venography to avoid vessel and adjacent structural injuries. Moreover, it is crucial to adequately flush all catheters to avoid air embolization and select appropriate balloons or stents to ensure proper expansion. For the management of complications, the use of a small balloon was initially suggested, with an upgrade to the most suitable size stage by stage. Once tamponade, hemothorax, or massive hemoptysis occurs, emergency aid and treatment must be administered immediately. If first aid measures do not work, endotracheal intubation is necessary to maintain airway patency, and surgical intervention may be required.

Symptomatic patients with PVS can be treated with either stenting or balloon angioplasty (BA), both of which are primarily limited by restenosis[14]. Data to date suggest that stenting is associated with more durable results. Compared with BA, stenting is associated with a lower risk of restenosis. The incidence of restenosis is 54% for BA and 22.3% for stenting[14]. The primary factor contributing to restenosis after BA is the phenomenon of elastic recoil, while neointimal hyperplasia appears to be the predominant cause in cases involving stenting[5]. Additionally, stents with a larger diameter demonstrated a lower occurrence of restenosis compared to smaller ones[14,15]. PVS caused by pulmonary vein isolation (PVI) is always located at the ostia of PVs with a larger caliber. On the other hand, PVS caused by FM is due to compression from proliferative fibrous tissue, and the lesion always occurs at the proximal first tributary of PVs with a smaller caliber. Literature reports that the restenosis rate in PVS caused by PVI ranges from 19% to 39% after a median follow-up period of 6.0 to 55.2 months[16,17,18]. However, the rate of in-stent restenosis (ISR) could be higher in PVS-FM.

A recent study[19] has shown that the rate of ISR in PVS-FM is 6.3%, 21.4%, and 39.2% at the 3-, 6-, and 12-month follow-ups, respectively, and the ISR is independently associated with the reference vessel diameter and stenosis of the corresponding PA. Despite the lack of randomized studies, current research[8,14,19] indicates that stenting is a preferred choice for PVS. For individuals diagnosed with ISR, the preferred treatment is high-pressure BA, and in those with BA failure, a stent-in-stent approach is considered as a final option[5]. One study reported a successful percutaneous approach using a drug-coated balloon (DCB) to treat PV restenosis, suggesting that DCB angioplasty could be a good strategy when restenosis occurs to avoid metallic multilayering[20].

Other treatments for FM-associated PVS include drug therapy and surgical intervention. To date, drug therapy is still in the exploratory stage, and its efficacy remains uncertain. Medical treatments such as antifungal, anti-inflammatory, and antifibrotic therapies have not achieved the desired results in addressing the burned-out proliferative tissues of FM[4]. Based on the hypothesis that B lymphocytes in tissue play a pathogenic role in progressive FM, Varghese et al.[21] treated 22 patients with metabolically active, progressive FM with off-label rituximab therapy. The results showed no disease progression in any patient after a median clinical follow-up of 42 months and imaging follow-up of 21 months. Histoplasmosis-associated FM accounted for 82% of the cases. After receiving rituximab treatment, there was a significant reduction of approximately 49.6%

in lesion volume within 24 months compared to ~~before treatment~~. The estimated successful response rate to this treatment was around 47.9%. These findings suggest that rituximab holds promise as an alternative for symptomatic and progressive FM. Surgical procedures, such as resection of the fibrous mass, pneumonectomy, pulmonary vein angioplasty, bronchoplasty, and lung transplantation, present challenges for both congenital and acquired PVS. The mortality rate can ~~be more than~~ 20%[7], and the long-term outcomes ~~are~~ uncertain, making its clinical application controversial.

Disclosures

The authors declare that they have no competing interests.

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