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Pulmonary Artery Stent Implantation for Fibrosing Mediastinitis: Our Clinical Experience

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Keywords: fibrosing mediastinitis | pulmonary artery | stent implantation | treatment

ABSTRACT

Fibrosing mediastinitis (FM) can block pulmonary vessels and airways, hindering treatment efficacy. Pulmonary artery (PA) stenting might provide a solution in such cases. This study involved 30 patients who had 49 PA stenting procedures for FM. Data on baseline characteristics, CT pulmonary angiography images, stent patency, and hemodynamics were collected. Patients with FM often had a history of chronic obstructive pulmonary disease (15/30), tuberculosis (12/30), and pneumoconiosis (11/30). Patients exhibited typical symptoms such as dyspnea, exercise intolerance, and cough. FM appeared as multiple bilateral shadows with enlarged hilar and mediastinal lymph nodes. Our study found that the PA involvement alone was predominantly in the left and right lower basilar trunk, with the left lower pulmonary arteries (LLPA) involved in 80% of cases and the right lower pulmonary arteries (RLPA) in 100%. Moreover, over 2/3 of patients showed involvement of both PA and pulmonary vein (PV), mainly in the bilateral upper lung lobes, then in the right middle lobe and left lingual lobe. After PA stent implantation, patients showed enhanced tricuspid annular plane systolic excursion (20.6 vs. 18.5, $p < 0.001$) and reduced right atrial diameter (35.5 vs. 37.3, $p = 0.042$), along with significant gains in 6-min walk distance (465.2 vs. 392.7, $p = 0.002$) and improved World Health Organization functional class ($p < 0.001$). Hemodynamic parameters improved after PA stent placement with significant reductions in systolic pulmonary artery pressure (PAP) (51.1 vs. 64.2, $p < 0.001$), mean PAP (28.4 vs. 35.2, $p < 0.001$), pulmonary vascular resistance (4.7 vs. 5.9, $p = 0.004$), and stent gradient (11.2 vs. 33.4, $p < 0.001$), along with increased patency (84.8% vs. 28%, $p < 0.001$), and fractional flow reserve (0.84 vs. 0.44, $p < 0.001$). Over a median follow-up of 331 days (range 45–980), no significant stent stenosis occurred ($p = 0.287$). Mild adverse events like cough and mild hemoptysis were noted during the procedure. Secondary intervention was needed for 5 of 49 stents. PA stents placement, especially the LLPA and RLPA, improved pulmonary vascular patency, hemodynamics, and symptoms.

Abbreviations: 6-MWD, 6-min walk distance; CTPA, computed tomography pulmonary angiography; FFR, pulmonary fractional flow reserve; FM, fibrosing mediastinitis; LLBT/RLBT, left/Right lower basilar trunk; mPAP, mean pulmonary artery pressure; **PA** or **PV**, pulmonary artery or vein; PVR, pulmonary vascular resistance; sPAP, systolic pulmonary artery pressure; SVC, superior vena cava; TAPSE, tricuspid annular plane systolic excursion; WHO-FC, World Health Organization functional class.

Cheng Hong, Daibing Zhou, and Haiming Chen contributed equally to this study.

1 | Introduction

Fibrosing mediastinitis (FM) is a rare, nonmalignant condition in the mediastinum that often progresses and causes compression. It may develop after exposure to infectious agents like *Histoplasma capsulatum*, *Mycobacterium tuberculosis*, and *Aspergillus* spp., or due to noninfectious factors such as IgG4 disease, sarcoidosis, silicosis, autoimmune vasculitis, and previous mediastinal radiation [1–3]. Infiltration of CD20-positive B lymphocytes is also considered to be related to FM [4]. Most FM patients are incidentally diagnosed via chest imaging, which shows a localized, invasive and frequently calcified mediastinal soft tissue mass [4]. This mass disrupts the normal fat plane and compresses or obstructs mediastinal structures like the pulmonary artery (PA), pulmonary vein (PV), superior vena cava (SVC), esophagus, bronchi, and thoracic spine [4–7].

FM-induced PA/PV stenosis has been identified as a potential contributor to the development of pulmonary hypertension, heart failure, and airway obstruction resulting from bronchial compression [5, 8, 9]. Rare complications may include elevated intracranial pressure due to superior vena cava obstruction [7] and pericardial varices [10]. Various interventions such as bronchoscopic airway stenting, balloon pulmonary angioplasty and percutaneous vascular stent implantation have been documented to alleviate respiratory symptoms associated with the FM-induced airway or vascular obstruction [8, 11, 12]. However, a study found that 4 out of 16 patients needed further PV intervention after stent placement, and 1 out of 19 required additional PA intervention [11]. Postoperative complications are severe with a 20% mortality [13]. Restenosis occurred in 12 out of 31 PV interventions, leading to serious cardiovascular events [12]. Thus, there is limited documentation and evidence on the effectiveness of vascular stenting in FM patients.

This study evaluated the effectiveness and safety of PA stenting in patients with FM-induced pulmonary vascular stenosis, examining pulmonary hemodynamics, complications, and short-term prognosis. We also reported on vascular stenting for FM-related pulmonary vascular compression.

2 | Materials and Methods

2.1 | Participants

A retrospective study was conducted at our hospital from January 2020 to March 2024 to review patients with FM. The diagnosis of FM was confirmed by computed tomography pulmonary angiography (CTPA) images [4]. Patients with mediastinal malignancy, sarcoidosis, IgG4-related diseases and radiation therapy were excluded. Histopathological examination was not pursued due to the absence of established diagnostic criteria, with exclusion being the primary approach. The study protocol was approved by the Ethics Committee of the First Affiliated Hospital of Guangzhou Medical University (No. 2022-K-12). Written informed consent was obtained from all patients upon hospital admission and before PA stent implantation.

2.2 | Data Extraction

Data were collected from medical records, including encompassing epidemiological information, symptoms, laboratory tests, echocardiography results [14], clinical outcomes, and measurements of the 6-min walk distance [15] (6-MWD), and World Health Organization-functional class (WHO-FC). The findings of CTPA and digital subtraction angiography were evaluated by two experienced radiologists. Procedure notes were examined for details on stent placement, model specifications, adverse drug reactions (ADRs), and right heart catheterization data. Follow-up information was gathered through outpatient clinical visits or telephone consultations, with data available until February 28, 2023.

2.3 | Computed Tomography (CT) Enhancement and Reconstruction

The General Electric Platform performed chest computed tomography enhancement. The specific parameters included a tube voltage of 90–100 kV, utilization of the automatic exposure control mode for tube current. The matrix size was set at 512×512, with a scanning field of view measuring 500 mm. The pitch was configured at 0.9, and the scanning layer thickness was 0.625 mm. Image reconstruction was conducted using a soft tissue algorithm, with a reconstruction layer thickness of 2.00 mm and increments of 0.7 mm. The time required for one complete revolution of the tube was 0.33 s. Enhanced scanning was conducted utilizing a high-pressure syringe to administer an iodixanol contrast agent, dosed at 1.1 times the patient's body weight in kilograms, via the median cubital vein. This was followed by the uniform injection of 30 mL of iodixanol contrast agent at a flow rate of 4.5 mL/s. The patient was instructed to hold breath, and a non-ECG-gated acquisition and smart enhancement tracking-triggered scanning was employed, with the region of interest designated at the bifurcation of the main trunk of the pulmonary arteries. Once the CT value of the main trunk of the pulmonary artery reaching 80 Hounsfield Units, enhanced scanning images of the pulmonary artery were acquired. These images were subsequently transmitted to the reconstruction workstation for post-processing utilizing techniques such as multiplanar reformation, maximum intensity projection, and volumetric rendering.

2.4 | Intervention and Drug Therapy

2.4.1 | Preoperative Assessment

Upon admission, patients diagnosed with FM underwent a thorough evaluation encompassing blood tests, echocardiography, and CTPA. Before the placement of PA stents, a multidisciplinary team consisting of interventional radiologists and pulmonologists assessed patient eligibility. The decision to proceed with stent placement was determined by evaluating the patient's condition, the extent of vascular obstruction, and the potential benefits of obstruction relief. We are before processing the PA without PV stenosis, which can occur if PA relief is effective but PV relief is ineffective. Before the PA stent implantation, the corresponding

PVs showed no delay of contrast agent after the pulmonary angiography with a pulmonary flow grade (PFG) score of 3 [16].

Recommending PA stenting in hemodynamic responses encompassed a pressure gradient exceeding 20 mmHg across the stenotic region, a PA diameter stenosis rate surpassing 50%, or a pulmonary fractional flow reserve (FFR) value below 0.75 (the ratio of mean pressure distal to a PA stenosis [Pd] to proximal mean PA [Pa]) [17, 18]. The symptoms of FM significantly affect daily life. The pressure gradient is the systolic pressure variance between the proximal and distal stenosis. A skilled radiologist evaluated the diameter of the narrowest in-stent site (A) and the unaffected distal pulmonary artery diameter of the stent (B) using angiographic images. Stent patency, or luminal patency, is calculated by dividing A by B [9].

2.4.2 | Catheterization Procedure

Venous access was established via the femoral veins to access the target PA using a vascular sheath, guided catheter, and guidewire. Figure 1 presented the procedure of PA stent implantation, included (a) assessment of stenosis location, the extent of vascular stenosis, FFR, pressure gradient, airway compression, and associated PV reflux; (b) measurement of vessel diameter at the distal end of the targeted vessel; (c) predilation of the stenosis was consistently carried out by selecting a balloon diameter of 2.0 mm up to the desired diameter of the balloon. (d) stent lengths were chosen to encompass the length of the stenosis, and the diameter for stent deployment matched that of the distal, unaffected vessels. All stents were bare metal, pre-mounted balloon expandable stents (Boston Scientific Express TM LD Vascular). Inflation pressure was determined based on the guidelines for pre-mounted balloon-expandable stents to achieve optimal symptom relief.

Following stent placement, hemodynamic measurements and angiography were repeated to evaluate pressure gradients and anatomic obstruction. Successful PA stenting was defined by (a) the systolic PAP gradient dropped over 10 mmHg, or the mean PAP decreased, or the FFR $\geq$ 0.75 [17], (b) Improvement in WHO-FC or 6-MWD. An increase of at least 30 m in 6-MWD

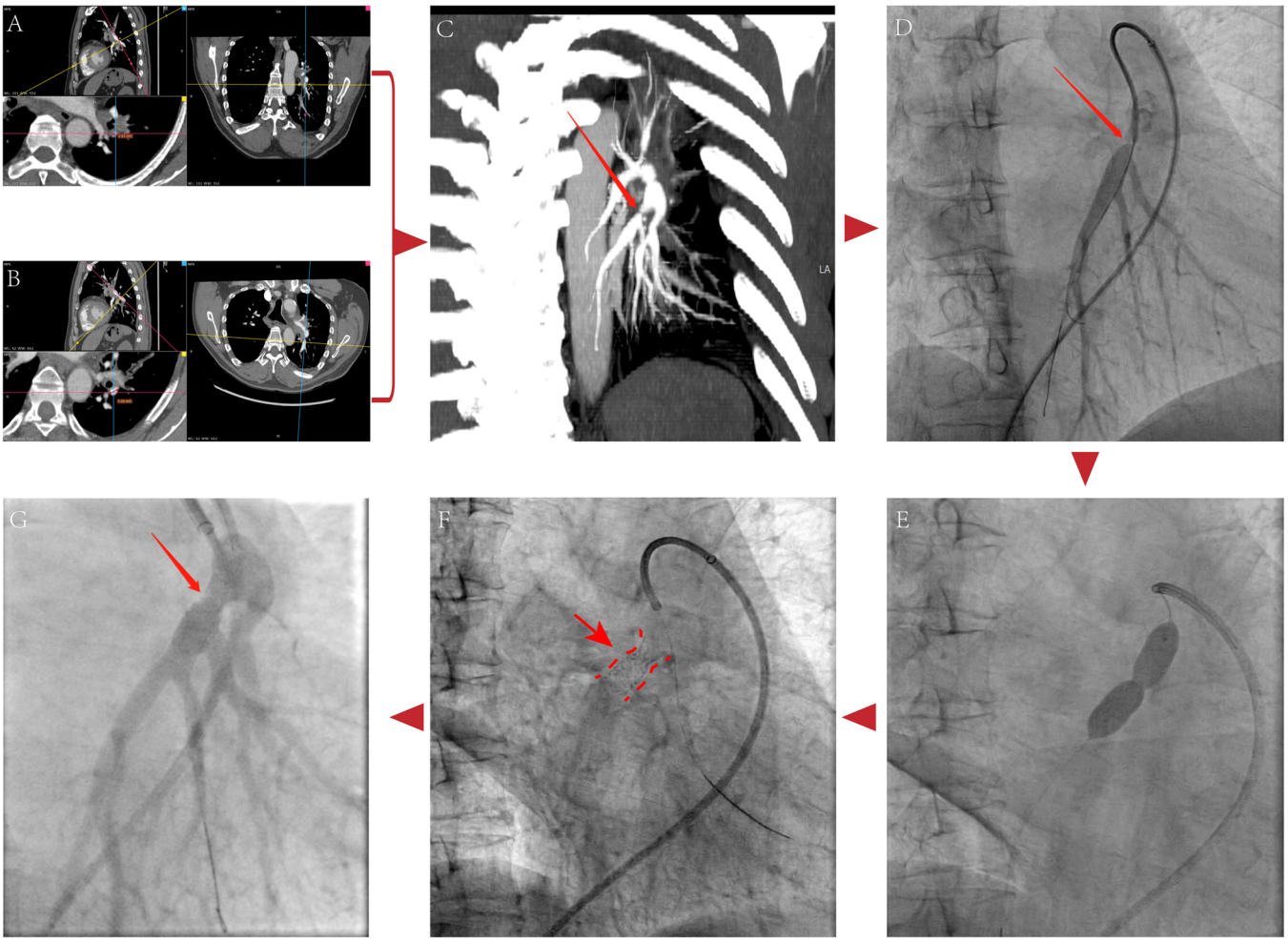


FIGURE 1 | The procedure of pulmonary artery stent implantation. (A) and (B) Three-axis CT images with anatomical slices in the sagittal, dorsal, and transverse planes. The narrowest PA in-stent location. The distal, uninfluenced PA diameter of the stent. (C) Preoperative three-diameter CT reconstruction image (red arrow). (D) Pulmonary angiography to evaluate the location of the lesion during the procedure (red arrow). (E) Before stent implantation, the balloon dilates the targeted PA. (F) After stent implantation, pulmonary angiography was again used to evaluate stent patency (red arrow). (G) Evaluation of stent patency by pulmonary angiography at follow-up (red arrow).

after PA stent implantation was deemed an improvement [15, 19]. Dual antiplatelet therapy (aspirin and clopidogrel) was started for 3 months post-PA stent placement, followed by single antiplatelet therapy for at least 6 months. The second intervention is to be considered if the stent stenosis and WHO-FC has worsened.

2.5 | Statistical Analysis

The statistical analyses were conducted using SPSS Statistics version 27.0 (IBM, NY, USA). Descriptive statistics were used to present continuous variables as mean ± standard deviation or median (range) and categorical variables as counts (frequency). Paired sample *t*-tests were employed to assess differences in continuous variables before and after treatment, while non-parametric tests were utilized for analyzing categorical variable data. Statistical significance was defined as a p-value < 0.05 by two-tail.

3 | Results

3.1 | Demographics and Baseline Characteristics

In this study, 30 FM patients, 9 women and 21 men received 49 stent implantations, as detailed in Table S1. Table 1 summarizes patient demographics, showing an average age of 58.7 years, a BMI of 23 kg/m^2, and the median time from symptom onset to stent placement of 58.7 months (range 2–144). Most FM patients had a history of chronic obstructive pulmonary disease (COPD) (15/30), pneumoconiosis (11/30), and tuberculosis (12/30), with a majority also being smokers. Common symptoms included dyspnea, exercise intolerance, cough, and chest tightness or pain, while less common symptoms included hemoptysis and weight loss. Many patients had a history of respiratory diseases such as COPD, pneumoconiosis, old tuberculosis, and asthma. But none had histoplasmosis or coronary artery disease.

The average pre-stenting white blood cell count was 7.88×10^9/L, and hemoglobin levels were 134.4 g/L, as detailed in Table 1. No significant differences were found in NT-pro BNP (184.6 vs. 268.2, $p = 0.205$, pg/mL), serum uric acid (363.6 vs. 371.7, $p = 0.736$, μmol/L), and D-dimer levels (598.1 vs. 520.6, $p = 0.24$, ng/mL FEU) before and after stenting. Predicted values for FEV1, FEV1/FVC, and DLCO were 69%, 87.4%, and 70.9%, respectively. 30 patients with 49 PA stents were monitored for a median of 331 days (range 45–980).

During the 126-day follow-up period, patients demonstrated improvement following PA stent implantation in tricuspid annular plane systolic excursion (TAPSE, mm) (20.6 vs. 18.5, $p < 0.001$) and right atrial diameter (RA d, mm) (35.5 vs. 37.3, $p = 0.042$). Additionally, there were significant increases in the 6-MWD (m) with values of 465.2 compared to 392.7 ($p = 0.002$) and an improvement in WHO-FC ($p < 0.001$) at a median time of 3 months (range 0.5–5) in the follow-up. However, no differences were observed in parameters such as main PA diameter (MPA d, mm), right ventricular diameter (RV d, mm), and ejection fraction (EF, %).

3.2 | The Feature of CTPA Images in Fibrosing Mediastinitis

Table 2 summarizes CTPA findings in FM patients. Initially, all 30 patients had bilateral lung lobar involvement, with the right lung having three lobes and the left two lobes. All patients showed PA and PV compression and bronchial involvement. A small number (10%) had mild SVC compression without treatment, and all had mediastinal lymphadenopathy. CTPA revealed lung abnormalities in all patients: nodular (100%), patchy shadows (93.3%), pulmonary atelectasis (73.3%), consolidation (66.7%), pleural effusion (46.7%), and pleural thickening (50%). No pulmonary cavitation was observed.

Figure 2 illustrates the segmental compression of the pulmonary artery (PA) or pulmonary vein (PV). The table at the bottom of the figure provides detailed information about the affected PA and PV within the left lobes (Figure 2A) and the right lobes (Figure 2B). The radar map on the left depicts the involvement of eight segments and the left lower basilar trunk (LLBT) structure. In the left lung lobe, segments L1 + 2 ($n = 23$) and L3 ($n = 22$) exhibited compression in both the PA and PV. Segment L4 demonstrated PA compression alone in 9 patients and compression of both the PA and PV in 15 patients. The lingual lobe L5 was affected in 15 patients with PA involvement alone, in 13 patients with both PA and PV involvement, and in no patients with exclusive PV involvement. PA involvement was noted in 24 patients within the left lower pulmonary arteries (LLPA) alone. The L7 + 8 segment was associated with a higher incidence of PA compression alone, whereas 17 patients exhibited no impairment of either the PA or PV in the L9 segment. In the L10 segments, 11 patients showed PA involvement alone, while 14 patients exhibited no involvement of either the PA or PV.

Regarding the right lung lobes, the CTPA imaging results for the left lung demonstrated similarities to those of the right lung. The concurrent compression of both pulmonary artery (PA) and pulmonary vein (PV) was most frequently observed in regions R1 ($n = 17$), R2 ($n = 20$), R3 ($n = 18$), R4 ($n = 21$), and R5 ($n = 19$). PA involvement alone was noted in 30 patients within the right lower pulmonary arteries (RLPA). Compression of the PA was predominantly observed in the lobe of R7 ($n = 20$) and R8 ($n = 16$). The sequence of unaffected PA and PV was RA10 ($n = 20$), RA9 ($n = 18$), and RA8 ($n = 10$).

3.3 | Hemodynamics Improvement After Stent Implantation

49 stenting procedures were conducted on 30 patients, with the resulting hemodynamic changes detailed in Table 3. The systolic PA pressure (sPAP, mmHg) exhibited a statistically significant decrease from 62.4 to 51.1 ($p < 0.001$), and mean PA pressure (mPAP, mmHg) decreased by an average of 7 mmHg (35.2 vs. 28.4, $p < 0.001$) following stent placement. Post-stenting, the mean pulmonary vascular resistance (PVR, WU) also decreased (5.9 vs. 4.7, $p = 0.001$). However, there were no significant changes observed in diastolic PA pressure (dPAP, mmHg) (18.8 vs. 17.2, $p = 0.061$), cardiac output (CO, L/min)

TABLE 1 | Baseline characteristics.

Characteristics	Pre-stenting	Post-stenting	*p*
Sex (female/male)	9/21		
Age (years)	58.7 ± 11 (39–81)		
BMI (kg/m^2)	23 ± 2.5 (18.4–26.8)		
From symptoms to stents (months)	58.7 ± 44.7 (2–144)		
Smoking (%)	16/30 (53.3%)		
History			
COPD	15/30 (50%)		
TB infection	12/30 (40%)		
Pneumoconiosis	11/30 (36.7%)		
Asthma	2/30 (6.7%)		
Histoplasmosis	0		
Coronary artery disease	0		
Symptoms (%)			
Dyspnea	30/30 (100%)		
Exercise intolerance	30/30 (100%)		
Cough	25/30 (83.3%)		
Chest tightness/pain	12/30 (40%)		
Hemoptysis	4/30 (13.3%)		
Weight loss	6/30 (20%)		
Blood testing			
WBCs (×10^9/L)	7.88 ± 2.56 (3.9–13.7)	—	
Hb (g/L)	134.4 ± 21.1 (79–162)	—	
NT-pro BNP (pg/mL)	268.2 ± 383.3 (10.6–1514)	184.6 ± 312.0 (13.5–1709)	0.205
Uric Acid (μmol/L)	371.7 ± 113 (181.6–614.2)	363.6 ± 126.4 (149–634.9)	0.736
D-dimer (ng/mL FEU)	520.6 ± 445.8 (141–2047)	598.1 ± 278.8 (227–1234)	0.24
Pulmonary function			
FEV1% pre	69 ± 14.2 (41.7–100.8)	—	
FEV1/FVC	87.4 ± 67.5 (25.5–87.4)	—	
DLCO% pre	70.9 ± 13 (38.3–93.6)	—	
Echocardiography			
Follow-up time (days)	—	126.0 ± 67.2 (30–270)	
TAPSE ($n = 21$) (mm)	18.5 ± 3.9 (11.2–27.3)	20.6 ± 4.3 (13–32)	**< 0.001**
MPA d (mm)	25.3 ± 4.1 (17–34)	25.3 ± 3.9 (21–34)	0.951
RAd (mm)	37.3 ± 6.2 (27–52)	35.5 ± 5.3 (24–46)	**0.042**
RVd (mm)	37.5 ± 8.7 (26–54)	35.3 ± 6.7 (24–50)	0.056
EF (%)	68.3 ± 5.1 (56–78)	69.1 ± 6.2 (52–84)	0.571
6-MWD[a] (m)	392.7 ± 90 (176–548)	465.2 ± 77.2 (327–600)	**0.002**
WHO-FC			
Follow-up time[b] (months)	—	2.8 ± 1.2 (0.5–5)	
I/II/III/IV	0/9/14/4	1/23/3/0	**< 0.001**

Abbreviations: 6-MWD, 6-min walk distance; COPD, chronic obstructive pulmonary disease; DLCO, diffusing capacity for carbon monoxide; EF, ejection fraction; FEV1%, the percentage of forced expiratory volume in exhaled in 1 s; FVC, forced vital capacity; MPA d, diameter of main pulmonary artery; RAs and RVd, refer to the diameter of right atrial and ventricular transverse diameter, respectively; TAPSE, tricuspid annular plane systolic excursion; TB, tuberculous bacillus.

[a] $n = 24$.

[b] $n = 27$.

TABLE 2 | Details of structural compression in the CTPA images.

CTPA images	$n = 30$ (%)
Location of involved lobes	
Bilateral	30/30 (100%)
Unilateral	0/30 (0)
Right upper lobe	30/30 (100%)
Right middle lobe	30/30 (100%)
Right lower lobe	30/30 (100%)
Left upper lobe	30/30 (100%)
Left lower lobe	30/30 (100%)
Involved structures	
Pulmonary arteries	30/30 (100%)
Pulmonary veins	30/30 (100%)
Bronchi	30/30 (100%)
SVC compression	3/30 (10%)
Mediastinal lymphadenopathy	30/30 (100%)
Nodular shadows	30/30 (100%)
Patchy shadows	28/30 (93.3%)
Pulmonary atelectasis	22/30 (73.3%)
Consolidation	20/30 (66.7%)
Pleural effusion	14/30 (46.7%)
Pleural thickening	15/30 (50%)
Cavity	0/30 (0)

(5.0 vs. 5.1, $p = 0.264$), and cardiac index (CI, L/min/m^2) (3.0 vs. 3.2, $p = 0.060$) before and after stent implantation.

The study demonstrated a significant increase in mean stent patency immediately postprocedure (84.9% vs. 28%, $n = 49$, $p < 0.001$) (Figure 3A), accompanied by a notable reduction in mean gradient following stenting (33.4 vs. 11.2, $n = 39$, $p < 0.001$) (Figure 3B) and a significant enhancement in FFR (0.84 vs. 0.44, $n = 39$, $p < 0.001$) (Figure 3C).

3.4 | Low Incidence of ADRs Following Stent Implantation

During the surgical procedure, it was observed that the ADRs such as cough ($n = 7$) and hemoptysis ($n = 3$) were infrequent and of low severity (Table S1). Ceasing the operation and applying balloon tamponade to the affected artery for an average of 10 min alleviated ADRs without needing further medication or rescue interventions.

3.5 | Low Restenosis in the Follow-Up

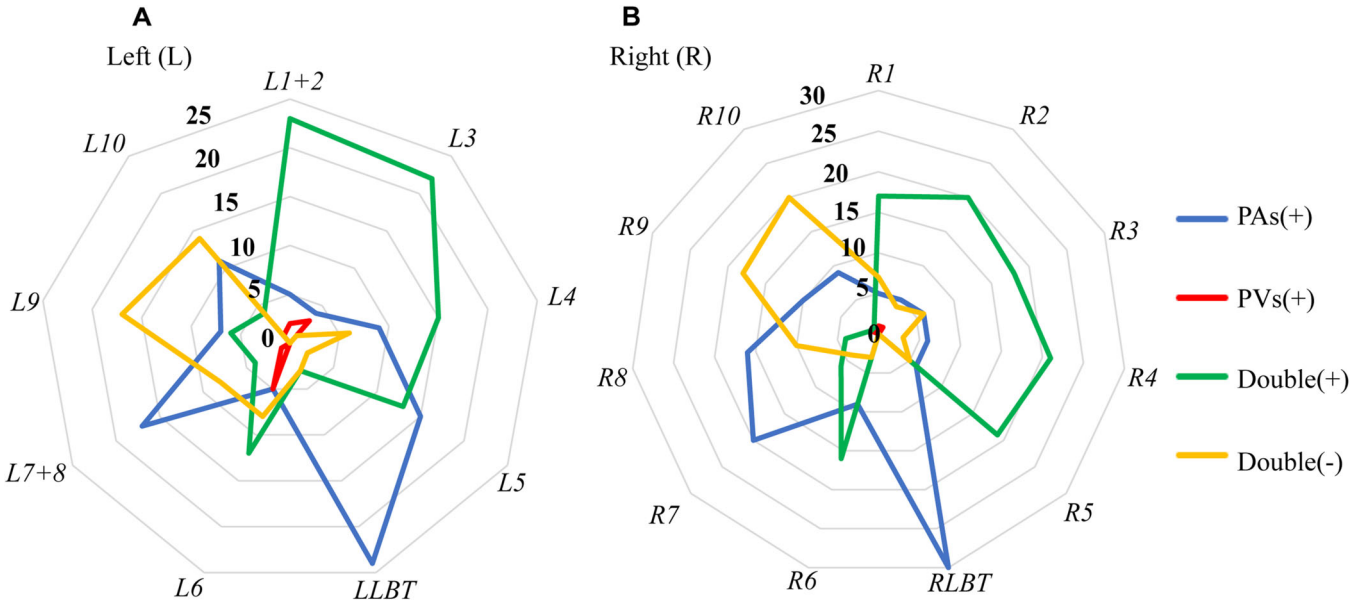
To assess short-term stent restenosis, CTPA images were used to analyze stent patency before and during follow-up, with immediate postimplantation patency determined via pulmonary angiography. The median follow-up period for 49 stents in patients was 331 days (range 45–980). There was no significant difference in patency rates before and after stenting (84.9% vs. 83.7%, $p = 0.287$) (Figure 3D). In the follow-up, five patients diagnosed with FM required a second balloon dilatation procedure. One patient failed to adhere to medical drugs and inconsistently utilized antiplatelet medications, leading to stent thrombosis. Among the other four patients, inadequate dilation or circular stenosis posed challenges immediately after post-stent implantation, necessitating secondary intervention.

4 | Discussion

Our study examined the institutional experience of FM-induced pulmonary vascular stenosis, revealing that patients exhibit clinical characteristics of segmental PA and PV compression in the upper lobes and segmental PA involvement primarily in the lower lobes. Specifically, PA involvement is commonly observed in the LLBT and RLBT. Consequently, PA stent implantation in both the LLBT and RLBT may be necessary to enhance hemodynamics, alleviate respiratory symptoms, and minimize the risk of ADR. These findings suggest that PA stenting would be a safe and promising strategy for FM.

FM is a mechanical compression disease characterized by the involvement of adjacent mediastinal structures. Evaluation of the characteristic imaging abnormalities and the structures involved may aid in diagnosing FM [20–22]. Chest radiographs may suggest mediastinal involvement. Additional radiographic and laboratory studies may help to differentiate FM from other conditions that mimic these clinical manifestations (e.g., sarcoidosis and malignancy). Radiographic lung abnormalities were often subtle and nonspecific, including multiple nodules, patchy shadows, atelectasis, consolidation, effusion, and pleural thickening, as reported in previous studies, where patients with PV compression were more likely to have pulmonary atelectasis and pleural effusion [5, 22, 23]. In our study, all cases (100%) showed bilateral lobe involvement, unlike a previous study's 53.4% (31/58) [11]. Only three cases had SVC involvement, with a common diffuse distribution across both lungs aligning with conditions like COPD, pneumoconiosis, and tuberculosis, which rarely compress the SVC [24, 25]. We found that FM patients were generally older (58.7 ± 11, years), predominantly male, and had a history of tobacco exposure (53.3%) contrasting with another study showing younger (36.7 ± 10.5), female FM patients [11]. Importantly, none of the 30 FM patients exhibited cavitary imaging abnormalities, aiding differentiation from *Mycobacterium tuberculosis* and *Aspergillus* infections [26, 27].

Patients with FM may benefit from endovascular repair with stents and balloon angioplasty because these procedures can immediately relieve obstruction and increase blood flow [28, 29]. We have previously noted that balloon angioplasty would only be available for those with endovascular stenosis in small vessels without optional stents and nonmechanical compression. One study has described an alternative surgical strategy – intraoperative PA stenting through the median sternotomy, with 44% of patients undergoing re-intervention of the stented PA for a median time of 2.6 years due to the progression of FM. The procedural complication rate of intraoperative PA was 4.4% [30]. One patient with FM benefited from the improved hemodynamics after PV stenting [29]; however, a small



	PAs(+)	PVs(+)	Double(+)	Double(-)
L1+2	5	2	23	0
L3	4	3	22	1
L4	9	0	15	6
L5	15	0	13	2
LLBT	24	0	3	3
L6	5	5	12	8
L7+8	17	1	4	8
L9	7	0	6	17
L10	11	1	4	14

	PAs(+)	PVs(+)	Double(+)	Double(-)
R1	5	1	17	7
R2	5	1	20	4
R3	6	0	18	6
R4	6	0	21	3
R5	6	0	19	5
RLBT	30	0	0	0
R6	9	2	16	3
R7	20	0	6	4
R8	16	0	4	10
R9	10	1	1	18
R10	9	0	1	20

FIGURE 2 | Involved segments of PA and PV in FM. The radar map above showed the vascular involvement of PA segments in the left lung lobe (A) and the right lung lobe (B). The table below shows the detailed vascular involvement in the left (A) and right (B) lung lobes. 30 cases were included. The items "PA (+)", "PV (+)", and "double (+)" refer to the compression of "PA alone", "PV alone", and "both PA and PV", respectively. "LLBT" and "RLBT" refer to the left and right lower basilar trunk, respectively.

TABLE 3 | Hemodynamics in the RHC.

Characteristics	Pre-stenting	Post-stenting	*p*-value[a]
sPAP (mmHg)	64.2 ± 21.2 (33–111)	51.1 ± 13.5 (26–76)	**< 0.001**
dPAP (mmHg)	18.8 ± 6.7 (7–32)	17.2 ± 6.0 (8–32)	0.061
mPAP (mmHg)	35.2 ± 10.7 (17–52)	28.4 ± 7.4 (15–46)	**< 0.001**
PAWP (mmHg)	7.9 ± 1.8 (3–12)	—	
PVR (WU)	5.9 ± 3.1 (1.1–12.2)	4.7 ± 2.4 (1.5–10.5)[b]	**0.001**
CO (L/min)	5.0 ± 1.0 (2.7–7.2)	5.1 ± 1.1 (2.4–7.9)	0.264
CI (L/min/m2)	3.0 ± 0.6 (1.7–4.2)	3.2 ± 0.8 (1.7–5.1)	0.063

Abbreviations: CI, cardiac index; CO, cardiac output; dPAP, diastolic PAP; mPAP, mean PAP; PAP, pulmonary arterial pressure; PAWP, pulmonary artery wedge pressure; PVR, pulmonary vascular resistance; sPAP, systolic PAP.
[a]Paired-samples *t*-test.
[b]PAWP refers to the PAWP before the stenting.

sample has described that four out of eight patients would experience at least one episode of re-stenosis, and four cases died within 4 weeks after their first PV intervention [31]. Furthermore, seven out of 31 patients with FM-associated PV stenosis died after PV stenting or balloon angioplasty, and the complications of PV stent implantation mainly included hemoptysis, phrenic nerve injury, acute coronary events, hemothorax, and even death [12]. PA stenting appears superior to PV stenting, with no patient dying after implantation [9]. However, more evidence is needed to evaluate the efficacy and safety of PA stenting.

Preoperative evaluation of lesion location, extent of involvement, and relationship to adjacent anatomical structures is

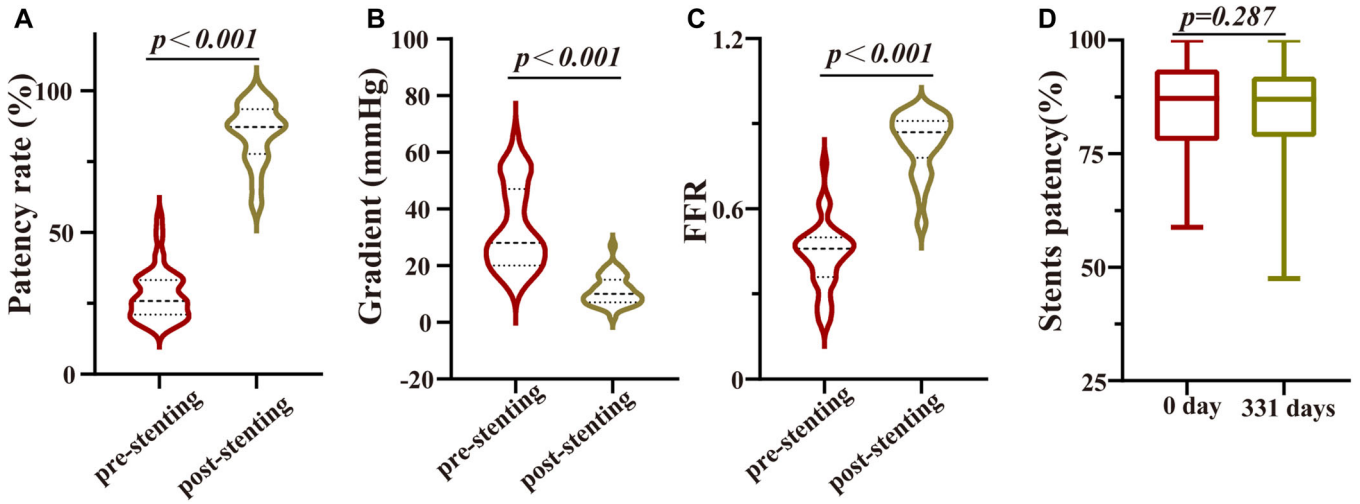


FIGURE 3 | Outcome and follow-up before and after stenting. (A–C) The targeted PA's patency, gradient, and FFR before and after stenting, respectively. (D) show the stent patency in the follow-up in a median duration of 331 days, compared with immediate stenting in the postprocedure. $p < 0.05$ was reported as significant.

essential for planning PA stent implantation. Our CTPA study revealed that all 30 cases had bilateral lobar invasion of the PA, PV, and bronchi, with no unilateral lobar compression observed. PA involvement was mainly located proximal to the RLPA and LLPA. The involvement of the superior both PV and PA were more common than that of the inferior. The PA and/or PV (L9 and R9/10) seem to be free from compression by FM, which may be related to the mediastinal lesions away from the PA/PV in the lower lobes. In addition, better compliance and higher pulmonary perfusion are carried out in the lower lobes of both lungs. Therefore, the target was the pulmonary artery stents in the lower lobes.

Stenting is typically performed to improve clinically significant central or large airway branching PA stenoses when the internal diameter of the pulmonary vessels is large enough without significantly affecting the adjacent PV, airways, contralateral vessels, or stents. Stent implantation presents new challenges to clinicians at PA bifurcations, PV, and involved FM sites. Treating just pulmonary artery stenosis without addressing the corresponding pulmonary vein stenosis raises the risk of pulmonary edema post-stenting, and thus, it is of importance to evaluate the perfusion of PFG score before the PA stents [16]. Catheter-based interventions have limited clinical utility primarily due to the significant risk of restenosis. The clinical management of PA and PV restenosis has been challenging. Given the sample size and short follow-up, our study demonstrated that a targeted PA stent can adequately dilate. To assess restenosis following stent implantation, two patients who underwent the procedure were examined using optical coherence tomography. The findings revealed intimal thickening of the peri-stent area, particularly at the most severe stenotic site. We speculate that restenosis may have been caused by the static interaction between the stent, mediastinal mass, and the shear force induced by blood flow. Furthermore, the neointimal proliferation on the inner stent surface postimplantation provides additional support for these hypotheses.

Bare-metal stents have significantly improved the prognosis of patients with FM but may increase the risk of in-stent restenosis and in-stent thrombosis, especially in patients with poor compliance and irregular use of anticoagulation or antiplatelet therapy. Cutting balloon angioplasty and drug-eluting stents have recently been introduced in Takayasu arteritis and coronary artery disease [32, 33]. These techniques have effectively reduced the incidence of restenosis and improved safety. However, the heterogeneity of FM with structural compression makes it different from stenosis caused by vasculitis and coronary atherosclerosis. More data are needed to demonstrate the efficacy and safety of cutting balloon angioplasty and drug-eluting stents in FM patients.

5 | Study Limitations

This study assessed the efficacy of PA stents in patients with FM and conducted a short-term follow-up. Several limitations should be acknowledged: (1) this is a single-center retrospective analysis in China, and the sample size of patients with PA stents was limited. (2) the duration of follow-up varied significantly due to poor adherence to medical recommendations, leading to a lack of long-term follow-up data. (3) assessment of primary stent patency immediately post-surgery was based on pulmonary angiography, which may differ from measurements obtained from CTPA images. (4) the diameter of stenotic lesions may not accurately represent valid values due to their elliptical and irregular shapes and the confounding effect of respiratory motion on the measurement. (5) The PFG score mainly evaluates perfusion in segmental or subsegmental pulmonary vessels, but not in the main PA or left/right pulmonary trunks. Using larger-sized stents in the PA compared to coronary stents might lead to a more reliable assessment of stent patency by CTPA [34]. We should develop more optimized PFG scoring methods, and collaborate with more medical centers to increase sample size and extend follow-up for detailed analysis of the benefits of PA stents in FM patients.

6 | Conclusion

We conclude that PA stenting, especially located in the LLPA and RLPA, improves pulmonary vascular patency and

hemodynamics and has symptomatic improvement for FM-induced vascular obstruction, and it shows low restenosis in the short term and ADRs in operation for patients with FM.

Author Contributions

Cheng Hong, Daibing Zhou, and Haiming Chen designed the study and drafted and revised the manuscript. Xiaofeng Wu, Wenliang Guo and Jiangyu Cui organized and analyzed the figures and performed the literature. Weijie Guan and Nanshan Zhong: revised the manuscript for important points. Jielong Lin designed the study and edited and revised the manuscript. All authors declare that they approved the final version of the manuscript.

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Ethics Statement

The Ethics Committee of the First Affiliated Hospital of Guangzhou Medical University approved this study protocol (NO. 2022-K-12). Written informed consent was obtained from all enrolled patients after hospital admission and before the implantation of the pulmonary artery stent.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data generated during and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.