Current Endovascular Approach in Adult Patients with Pulmonary Vein Stenosis: A State-of-the-Art Approach

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Abstract: Regarding to more invasive treatment of atrial arrhythmia (atrial fibrillation ablation) and pulmonary vein isolation (PVI), the rate of acquired pulmonary vein stenosis (PVS) is increasing and at present, PV ablation for AF has become the principal cause of PVS in adult patients. On the other way, by improvement in procedural techniques, equipment, and the experience of the operators, the incidence of PVS has been decreased. There is some controversy about the manner of follow-up of these patients and in most centers, just symptomatic patients are considered for imaging and treatment. Almost always, those with PV stenosis more than 70% or multiple PV involvement become symptomatic and if give them up without treatment, pulmonary symptoms and finally irreversible pulmonary hypertension will occurred. So,

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intensive pursue after the procedure is highly recommended. Whereas in pediatric patients with congenital or acquired PVS, the best treatment approach is surgery, in adult patients, the preferred type of treatment is the transcatheter intervention with high acute success rate. In this present review, we have scrutinized about the diagnostic modalities, the indications for intervention, the diverse treatment strategies, and principally clarify an accurate stepwise approach during transcatheter procedure. (Curr Probl Cardiol 2022;47:100850.)

Introduction

Pulmonary vein stenosis (PVS) can be congenital and comprises 0.4% of all cases of congenital heart disease. Patients with PVS most often become symptomatic in the first few months to years of life and frequently have 1 or more additional cardiac anomalies; nonetheless, they have no active inflammation in or around the involved segments of the vein. The estimates of the incidence of associated cardiac defects range from 30% to 80%.[1] The most commonly associated congenital heart defects are septal defects, but PVS has been seen in conjunction with all major types of congenital cardiac malformations. The timing and severity of symptoms in pediatric patients with PVS appear to depend largely on the number of pulmonary veins involved and the obstruction severity of individual pulmonary veins. The progression of the disease is in tandem with increasingly prominent signs of pulmonary hypertension. In acquired instances, which occur after the surgical repair of a total or partial anomalous pulmonary venous connection (10%), myxoma resection, lung transplantation, atrial fibrillation (AF) radiofrequency ablation (the most common cause [60%] of PVS), sarcoidosis, neoplasia (lung cancer and lymphoma), and mediastinal masses resulting in extrinsic compression or infiltration and mediastinal fibrosis,[2] patients most often present with dyspnea or orthopnea and sometimes have a radiographic appearance of localized edema. Hemoptysis is not uncommon. In both congenital and acquired forms of PVS, histological findings show variable manifestations of neointimal proliferation, leading to the occlusion of the lumen of 1 or more of the pulmonary veins. It is estimated that approximately 60% of patients have unilateral disease, with the stenosis overwhelmingly involving the left pulmonary veins. Approximately 40% of patients have bilateral pulmonary vein disease.[3] Three patterns of PVS

have been described: bilateral tubular hypoplasia, which extends from the veno-atrial junction for a variable length; discrete hourglass constriction at the veno-atrial junction; and bilateral multiple short pulmonary veins, which are hypoplastic for their entire extrapulmonary course.[4] The prognosis is often poor. The disease tends to be progressive and is associated with high mortality (30%−50%) in the first 2 years after diagnosis. At present, PV ablation for AF has become the principal cause of PVS; the incidence rate derived from recent studies reaches a mean of 2%.[5] These figures represent a significant reduction in comparison with those reported in pioneer series. The main factors contributing to this finding are operator experience and improvements in the procedure, including the changing of the ablation site from the antra to the ostia of the pulmonary veins, reduction of the temperature applied to the tissue, and cryoablation. However, the real occurrence of PVS is probably underestimated as screening is only performed within the first 3 months in some centers (it has been demonstrated that PVS can occur over this period) and asymptomatic patients are not imaged. Therefore, screening with available imaging modalities in patients with PV ablation who develop respiratory symptoms is warranted.

Clinically, almost all patients with mild ($< 50\%$) or moderate stenosis (50%−70%) of the pulmonary veins do not have symptoms. (For symptoms to occur, at least 60% vessel diameter narrowing is usually necessary.) The occurrence of respiratory symptoms including coughing, hemoptysis, and dyspnea, which usually appear 3 to 6 months after the procedure, is associated with the severe stenosis of a single pulmonary vein ($> 70\%$), lung perfusion reduction by more than 20% to 25%, or the involvement of multiple pulmonary veins.[6] Nevertheless, it is also known that patients with severe stenosis or the occlusion of a single pulmonary vein can remain asymptomatic. In this context, albeit still controversial, the strategy of systematically screening for PVS with computed tomography (CT) scan or cardiac magnetic resonance imaging (CMR) for all patients undergoing pulmonary vein isolation, regardless of symptom manifestation, can be considered.[7] In asymptomatic patients, the failure or delay in recognizing PVS and its resultant interruption of postprocedural anticoagulation can result in the thrombotic occlusion of the pulmonary vein, with adverse effects on pulmonary perfusion and even long-term pulmonary hypertension and irreversible pulmonary damage.

Diagnostic Workup

In the clinical classification of pulmonary artery hypertension, PVS is arranged in Class II (post-capillary pulmonary hypertension). The past

decade has witnessed an increase in AF ablation (pulmonary vein isolation), which has been allied to a rise in the incidence of PVS. It appears that AF ablation is the most iatrogenic underlying cause of PVS in adult patients. Most patients present within 3 to 6 months of ablation. Still, as their symptoms are nonspecific and mimic pulmonary and cardiac disorders such as pneumonia, bronchitis, pulmonary emboli, and even malignancy, the rate of underestimation and mismanagement is high, which calls for a high index of suspicion.[8] In the absence of a consensus based on comprehensive randomized trials on the screening of these patients, the risk of disease progression to total occlusion, pulmonary edema, infarction, and lung parenchymal damage is likely to increase. Indeed, it seems that each center currently has its own strategy for screening, the time of screening, and the type of diagnostic imaging tools.

Chest X-ray may illustrate signs of waxing and waning localized or diffuse congestion (opacification). Echocardiography, either transthoracic (TTE) or transesophageal (TEE), is a noninvasive modality to demonstrate the stenotic severity (an increased maximum pulmonary vein Doppler flow velocity (> 1.1 m/s), combined with color Doppler turbulence)[9] and number of the involved pulmonary veins, as well as other anomalies and pulmonary artery pressure. Based on recent studies, dedicated contrast CT scans timed for pulmonary vein enhancement and 3D reconstruction constitute the optimal noninvasive imaging modality. CT scanning is used to measure the pulmonary venous diameter, with the narrowest diameter set as the minimum diameter and the widest diameter in the upstream portion used as the reference diameter. If a bifurcation of the peripheral pulmonary vein is noted at this point, the reference diameter is considered the diameter of the portion just before the bifurcation. Sometimes in patients with totally occluded pulmonary veins in another imaging tool, CT scans can visualize microchannels, but the overestimation of stenosis severity in near-total pulmonary vein occlusion has also been reported in such scans. CMR is a sensitive and accurate alternative tool to recognize PVS by measuring the maximal diameter, perimeter, and cross-sectional area at a location in the sagittal plane at which the pulmonary veins separate from the left atrium and each other. CMR can also assess the functional measurements of flow in the pulmonary veins and the quantification of flow redistribution. Ventilation-perfusion (VQ) scanning is another functional test to determine the severity of PVS, but it is not specific. PVS leads to a longer blood-contrast transit time in the lung, resulting in decreased perfusion to the lung or non-opacification of the lobar, segmental, and subsegmental veins in the lung. CT scanning and CMR confer an evaluation of the severity of stenosis, the number of stenotic pulmonary veins, the diameter of the pre-and post-stenotic pulmonary veins, the length between the pulmonary vein origin

and the first bifurcation, and any associated cardiac or pulmonary disease. Consequently, via these noninvasive modalities, before the endovascular procedure, the operator can designate the treatment options (eg, observation, balloon pulmonary angioplasty, and primary stenting) and the required equipment in the catheterization laboratory (eg, the type, diameter, and length of the balloon, drug-eluting balloons, cutting balloons, balloon-expandable or self-expandable stents, and covered stents). Ultimately, the gold-standard method for the confirmation of PVS (anatomical and hemodynamic data) is invasive venography, pulmonary artery wedge angiography, and the measurement of the actual pressure gradient between the pulmonary veins and the left atrium by withdrawing the catheter. Occasionally, in cases reported in CT scanning or other modalities to have completely occluded pulmonary veins, venography may visualize some microchannels, which enable the successful recanalization of the pulmonary veins with specific coronary wires similar to chronic total occlusion wires.

Indications for Treatment of Pulmonary Vein Stenosis in Adulthood

There are no dedicated guidelines or consensus about the kinds of management in patients with PVS; nonetheless, according to most authorities on this issue, patients with symptomatic severe stenosis ($>$ 70%) of 1 or multiple pulmonary veins should be treated. For asymptomatic patients with severe stenosis in a single pulmonary vein, there is no consensus concerning the type of treatment, with centers deciding individually. Imaging monitoring every 3 to 6 months is recommended in asymptomatic patients with 50% to 85% stenosis.[10]

Treatment

In adult patients, the most frequent causes of PVS are neoplastic infiltration and/or extrinsic compressions, non-neoplastic infiltration and/or extrinsic compressions such as fibrosing mediastinitis, and iatrogenic interventions.[8] Nowadays, pulmonary vein isolation for AF ablation is the most common cause. Whereas in pediatric patients with congenital or acquired PVS, the best treatment approach is surgery, in adult patients, the preferred type of treatment is the transcatheter intervention by expert interventionalists with high acute success rates and minimal complications.

Step-by-Step Approach

1. The cornerstone of treatment is a meticulous assessment of noninvasive imaging (CT scanning or magnetic resonance venography) by the expert radiologist and decision-making by the PVS team, comprised of a general cardiologist, an interventional cardiologist, a cardiac surgeon, an adult congenital cardiologist, a radiologist, and a cardiac anesthesiologist. Before catheterization, with the aid of imaging tools, the operator needs to characterize the indication for the intervention, the normal variants of the origin of the pulmonary veins (Fig 1), the severity of PVS (as a normal pulmonary vein has an average diameter of 10 to 15 mm, a diameter of approximately 4 to 6 mm is usually necessary for symptoms to occur), the number of the involved pulmonary veins (left upper, left lower, right upper, right middle, and right lower pulmonary veins), the etiology of PVS (iatrogenic interventions or extrinsic compressions by mediastinal or pulmonary masses) (Fig 2), the location of the stenosis or occlusion (discrete veno-atrial junction, progressive PVS that extends into the intraparenchymal or the so-called "upstream veins", the residual diameter of the most stenotic segment, and the widest diameter in the upstream portion considered to be the reference diameter. If a bifurcation of the peripheral pulmonary vein is noted at this point, the reference diameter is appraised as the diameter of the portion just before the bifurcation (Fig 3A-C). Notably, the salient predictor of restenosis whether after ballooning or stenting is the reference size of the vessel: the chances of restenosis are remarkable when the reference size is less than 10 mm in comparison with when the reference size exceeds 10 mm (70% vs 10%).[11] Hence, in our center, the recommended strategy is based on the reference vessel diameter. In other words, primary stenting with larger stents is performed when the reference diameter in imaging is greater than 8 mm, whereas sequential balloon dilatation is carried out when the distal diameter in imaging is less than 7 or 8 mm. Stenting is done in another session in the following instances: if the residual stenosis is less than 20%, if the pressure gradient is reduced to below 2 mm Hg, if there is no acute recoil (under TEE and fluoroscopic guidance), and if the reference diameter exhibits an increase in follow-up imaging. The length of the stent depends on the distance from the stenotic site to the first distal branching point (Fig 3C). In patients with totally occluded pulmonary veins, microchannels should be sought meticulously.

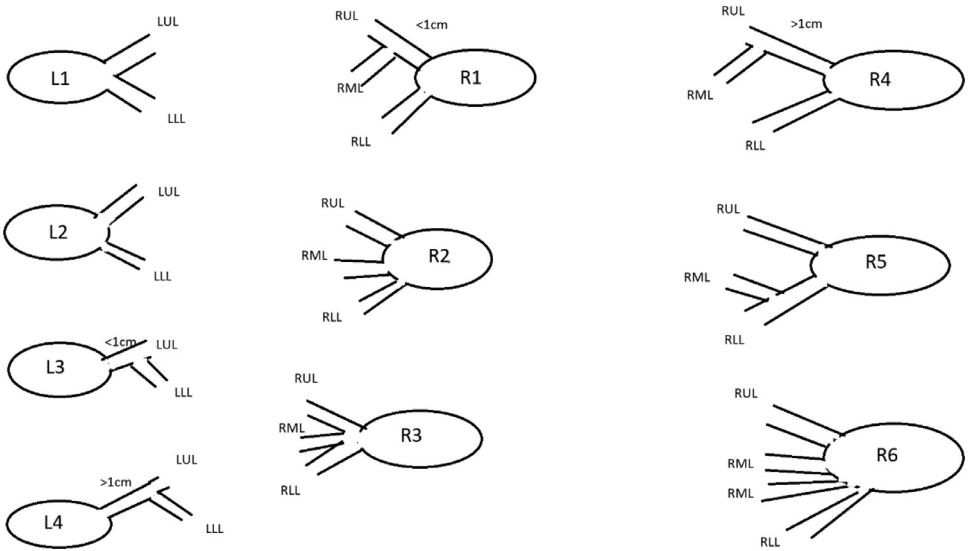

FIG 1. The diagram demonstrates the normal anatomic variants of the pulmonary veins L1, two separate ostia for the left pulmonary veins that are not separated by the atrial wall; L2, two separate ostia for the left pulmonary veins that are separated by the atrial wall; L3, the left lower pulmonary vein drains into the left upper pulmonary vein with a short (<1 cm) common segment; L4, the left lower pulmonary vein drains into the left upper pulmonary vein with a long (>1 cm) common segment; R1, two separate ostia for the right pulmonary veins, with the vein draining the right middle lobe joining the right upper pulmonary vein and forming a short (<1 cm) common segment; R2, three separate ostia for the right-sided pulmonary veins; R3, the common ostium for the right-sided pulmonary veins; R4, two separate ostia for the right pulmonary veins, with the vein draining the right middle lobe joining the right upper pulmonary vein and forming a long (>1 cm) common segment; R5, two separate ostia for the right pulmonary veins, with the vein draining the right middle lobe joining the right lower pulmonary vein; R6, four separate ostia for the right-sided pulmonary veins, with 2 veins draining the right middle lobe. The most common anatomic pattern is L1/R1, with a total of 4 pulmonary veins draining into the left atrium. LUL, left upper lobe; LLL, left lower lobe; RUL, right upper lobe; RML, right middle lobe; RLL, right lower lobe; RTPV, right top pulmonary vein; PV, pulmonary vein. (Color version of figure is available online.)

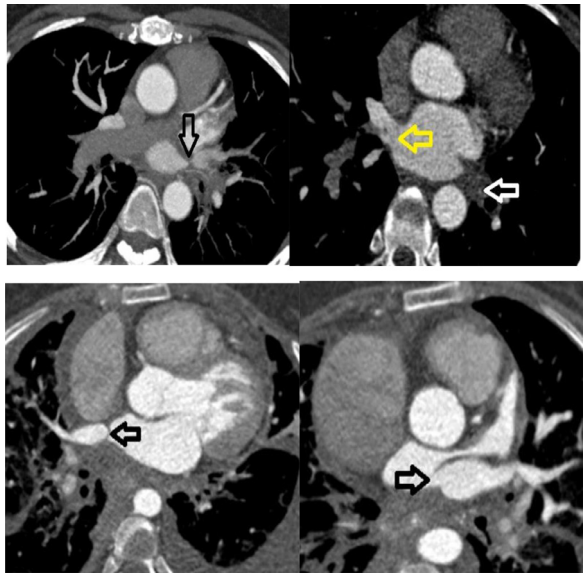

FIG 2. **(A-D)** The CT Angiography images illustrated acquired pulmonary vein stenosis in adults secondary to percutaneous radiofrequency catheter ablation for atrial fibrillation and fibrosing mediastinitis. Significant stenosis of LUPV (black arrow) followed by AF ablation is visualized (A); and in the same patient, RUPV (yellow arrow) is open whereas the LLPV is totally occluded (white arrow) (B). In figure C and D, significant stenosis of RUPV and LUPV (black arrow) is demonstrated in a patient with advanced idiopathic mediastinal fibrosing. (Color version of figure is available online.)

2. The confirmation of PVS in symptomatic patients is followed by diagnostic catheterization and therapeutic intervention. Under general anesthesia, the right common femoral artery and vein are cannulated with short sheaths (6 F), and hemodynamic data, including left ventricular end-diastolic pressure, right ventricular pressure, right atrial pressure, and mean pulmonary artery pressure, are measured. Thereafter, pulmonary artery wedge angiography can be performed on the dedicated vein, with the catheter wedged in the related pulmonary artery. Levo-phase angiography can reveal the delayed filling of the related pulmonary vein and its stenosis degree.
3. Under TEE guidance and through the venous access, septostomy (the best site of septostomy for PVS interventions is the posterior and superior rims of the fossa ovalis) with the Brockenbrough needle is done and diluted contrast is injected to confirm its placement in the left atrium (Fig 4A).
4. The Spiral wire (coiled tip, 0.025″) is maintained in the left atrium for further support and LA border illustration (Fig 4B).

FIG 3. (A-C) The images illustrate the planning of a stenting procedure for pulmonary vein stenosis treatment by using contrast-enhanced computed tomography multiplanar reconstruction. Measurement of the minimum diameter at the stenosis and measurement of the reference diameter (ie, the maximum diameter of the pulmonary vein distal to the stenosis) (A). measurement of the distance between the stenosis site to the first distal branching point (B). The minimum diameter at the stenotic segment and the distance between that and the reference distal segment (C). (Color version of figure is available online.)

5. Intravenous heparin is completed to achieve an activated clotting time of greater than 250 seconds.
6. With a 125 cm Multipurpose diagnostic catheter (6 F), which is telescoped through a steerable trans-septal sheath (Agilis sheath), a 0.035″ straight hydrophilic wire is advanced to cross the target lesion within the pulmonary vein under TEE and fluoroscopic guidance as distally as possible. Care should be taken about the position of the wire since one of the most frequent intraprocedural complications is pulmonary vein perforation by the wire. Afterward, the multipurpose catheter is passed gently over the wire, with its tip at the most distal part. In patients with the subtotal or total occlusion of the pulmonary vein, the dedicated pulmonary vein should sometimes be crossed and recanalized with coronary (0.014″) wires in the same manner as the wiring of totally occluded coronary arteries (chronic total occlusion wires).
7. The hydrophilic wire is exchanged with the Amplatzer Super Stiff Guidewire.
8. The catheter is retracted so that the mean pressure gradient (MPG) across the lesion can be measured.
9. Contrast-enhanced venography is carried out to confirm the stenosis and to define both the characteristics of the lesion (as noted in Part 1) and the degree of the distal opacification of the pulmonary vein precisely (Fig 5A, B).
10. After the withdrawal of the Agilis sheath, a long delivery sheath (Cook 12 F) is advanced through the septum. The end tip of the sheath is passed over the guidewire in order that it can cross the ostium of the pulmonary vein.

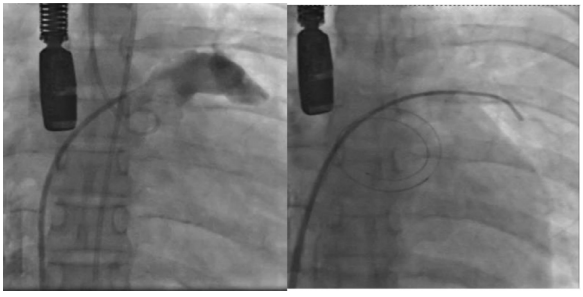

FIG 4. (A, B) After septostomy, diluted contrast is injected to confirm the placement of the wire in the left atrium (A). We recommend that the Spiral wire (coiled tip, 0.025″) be maintained in the left atrium for further support and LA border illustration (B). (Color version of figure is available online.)

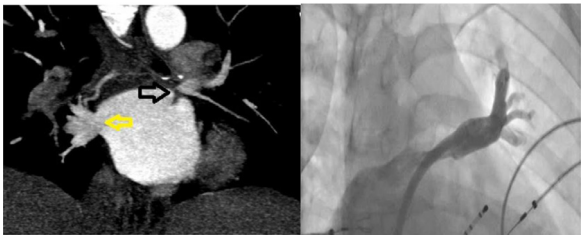

FIG 5. (A, B) In CTA, significant stenosis of LUPV origin (black arrow) and patent RUPV origin (which balloon dilatation was performed for that 3 months ago) after AF ablation is illustrated (A). Contrast-enhanced venography is carried out to confirm the stenosis of the left upper pulmonary veins and to define the characteristics of the lesion and the degree of the distal opacification of the pulmonary vein precisely (B). (Color version of figure is available online.)

11. According to the CT scan or magnetic resonance venography data, an appropriately sized peripheral balloon (over-the-wire [OTW]) is chosen and the sequential balloon dilatation of the most stenotic part of the pulmonary vein is performed under TEE and fluoroscopic guidance (Fig 6A, B). After each dilatation, the MPG is measured via TEE. A successful procedure is defined as MPG of less than 2 mm Hg and residual stenosis of less than 20%. The diameter of the selected balloons should not exceed the distal vessel by a factor of 1.1 (Fig 6A, B) as recent studies have shown that selecting a balloon or a stent significantly larger than the reference size is associated with more neointima hyperplasia and restenosis. Some lesions, especially right PVS, render the passage of the catheter through the lesion difficult. In this situation, via the balloon swallowing technique, a microcatheter such as the Navi-Cross can be passed through the lesion for distal vessel venography.

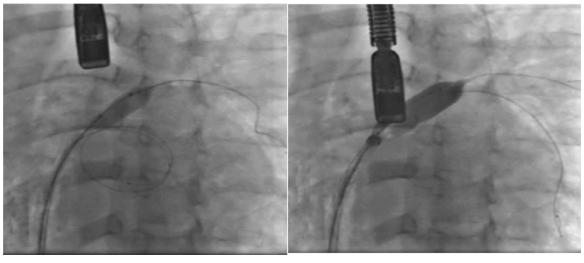

FIG 6. (A, B) The diameter of the selected balloons should not exceed the distal vessel by a factor of 1.1/1(A). In some patients with resistant and fibrotic lesions, BIB balloons could be used (B). (Color version of figure is available online.)

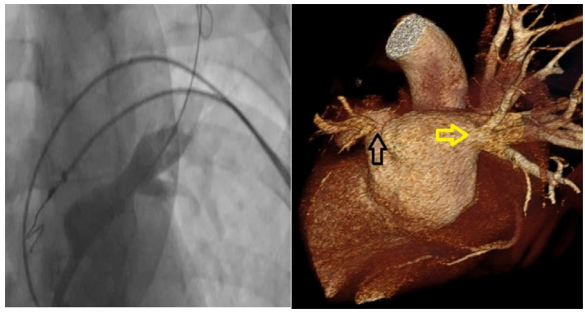

FIG 7. (A, B) Final venography after balloon dilatation illustrates less than 20% residual stenosis (A). Follow-up CTA, demonstrated patent LUPV (yellow arrow) with significant stenosis of RUPV (black arrow) 6 months later (B). The patient scheduled for balloon dilatation and stenting of RUPV for another session. (Color version of figure is available online.)

12. Hemodynamic improvement should immediately follow successful ballooning. In some patients with resistant and fibrotic lesions, BIB balloons (Fig 6B) and scoring balloons could be used for long durations (30−60 seconds).
13. At the end of the procedure, if the final MPG is below 2 mm Hg and the residual stenosis is less than 20%, final venography is performed to assess the degree of the distal opacification and to rule out any oozing or perforation (Fig 7A, B).
14. If the distal diameter of the dedicated pulmonary vein exceeds 8 mm after balloon angioplasty (BA), stenting the lesion should be preferably performed in the same session. However, in patients with smaller diameters and successful balloon dilatation, follow-up imaging (1−6 months later) findings and patients' symptoms guide the next step. Stenting is performed in cases of failed BA (Fig 8A, B), acute recoil after BA, and dissected pulmonary veins. Balloon-expandable stents are recommended for stenting since the mid-to-long-term patency of the vessel is directly related to the vessel size. Higher rates of restenosis are observed in pulmonary veins less than 10 mm in size. The diameter of the selected stent should be at least 8 mm, and the length of the stent (which is characterized before the procedure via imaging tools) should be chosen based on the distance between the stenotic site and the first distal branching point (Fig 3). Considering that the diameter of coronary drug-eluting stents does not exceed 5 mm, peripheral vessel stents such as Cheatham platinum (CP) and Valeo stents are recommended. During stenting, while the tip of the delivery sheath is beyond the stenosis, the selected stent is advanced through the sheath; and while the sheath is slowly withdrawn via

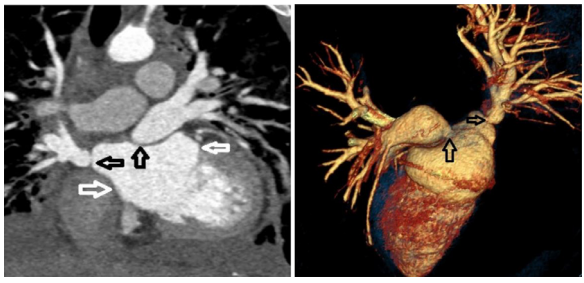

FIG 8. (A, B) This patient was a case of Idiopathic mediastinal fibrosis who in CTA had significant stenosis of LUPV and RUPV (black arrow) and also totally occluded LLPV and RLPV (white arrow-A). In the first session, balloon dilatation of both LUPV and RUPV was done, but 6 months later, she was symptomatic and in CTA had significant restenosis of both PVs (B). So, she scheduled for stenting of PVs. (Color version of figure is available online.)

venography in multiple projections, the precise location of the stent deployment is established (Fig 9 A-C). The essence of stent deployment is to cover the lesion totally, to avoid side-branch compromise, and to prevent the stent from overhanging into the left atrium too much. Whether or not to post-dilate the stent is at the discretion of the interventionalists.

15. If stenting is done and the final MPG is below 2 mm Hg, final venography is performed to confirm the appropriate position and expansion of the stent, to corroborate the patency of the first branching, to determine the degree of the distal opacification, and to rule out any oozing or perforation.

The Current Approach in the World

Due to lack of any definite randomized trials and consensus about the screening and treatment options in patients with post AF ablation pulmonary vein stenosis (the most common cause of acquired PVS), every center has its own manner for screening, imaging modalities and the interval of surveillance. Based on the pervious studies, dedicated contrast CT scans timed for pulmonary vein enhancement and 3D reconstruction is the best modality to characterize the narrowest segment, the widest diameter in the upstream portion which is considered to be the reference diameter. Some believe that the initial treatment approach is BA and the diameter of the selected balloons should not exceed the distal vessel by a factor of 1.5. Otherwise, as the rate of PV restenosis (either immediately due to acute recoil and dissection or after several months due to intimal hyperplasia) is high following BA, some others, prefer primary balloon-

FIG 9. (A-C) During stenting, while the tip of the delivery sheath is beyond the stenosis, the selected stent is advanced through the sheath; and while the sheath is slowly withdrawn via venography in multiple projections, the precise location of the stent deployment is established (A). Notably, the essence of stent deployment is to cover the lesion totally, to avoid side-branch compromise, and to prevent the stent from overhanging into the left atrium too much (B). In the same session, also, stenting of the RUPV was performed (C). (Color version of figure is available online.)

expandable stenting (which the size of the appropriate stent was selected according to the non-invasive modalities and by selective venography); although the chance of PV stent restenosis and stent thrombosis is not infrequent in stents with diameter less than 8mm. In some centers after BA of PVs, bailed out stenting is done if the patient being symptomatic or in follow-up imaging, PV restenosis more than 70% be detected. So, the kind of the treatment approach is individualized in each center regarding the experience of the PVS team.

Complications

The complication rate during and after the transcatheter treatment of PVS is highly dependent on appropriate decision-making by the PVS team. During the procedure, the most important complications are bleeding, pulmonary vein perforation and tamponade by the wire, stent migration into the left atrium, cerebrovascular accident, acute stent thrombosis, hemoptysis, ST-elevation, pleural effusion, and pulmonary hemorrhage.[8] The mid-to-late outcome of these patients is determined by the final vessel diameter and the size of the stent deployed. Restenosis after BA or stenting is the rule; nevertheless, regarding the technique itself, stenting appears superior to isolated BA in terms of vessel restenosis (25% vs 49% within 3 years' follow-up).[12] Despite the lack of randomized studies, currently available published research indicates that stent implantation should be the first-line therapy in patients with radiofrequency ablation-induced severe PVS. For the minimization of the restenosis rate, stents with diameters of 8 mm or greater should be selected. The most common time for the occurrence of in-stent restenosis is 6 to 12 months after the procedure. The etiology of post-BA restenosis is elastic recoil; and in those with stenting, neointima hyperplasia seems to be the primary cause.[13] The first line of treatment of in-stent restenosis is high-pressure BA (either drug-eluting or not); and in those with BA failure, stent-in-stent is done as the last resort.

Postprocedural Surveillance

After the endovascular treatment of PVS, it is recommended to administer lifelong clopidogrel (75 mg/d) and anticoagulant agents (warfarin or novel oral anticoagulants [NOACs]) in the absence of contraindications.[14] Considering the high incidence of restenosis, it is also advisable that these patients be monitored with the aid of CT angiography before discharge and then 6 months and 12 months afterward (Fig 10 A, B). If no significant restenosis is detected, annual CT angiography or magnetic resonance venography is suggested for 4 years, followed by clinical and anatomical follow-ups every 2 to 3 years.

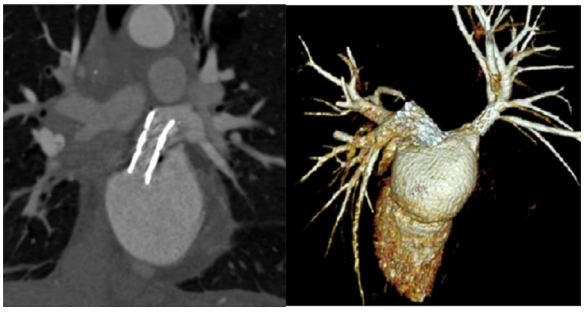

FIG 10. (A, B) Follow-up CTA after 6 months, illustrated patent both stents (A, B). (Color version of figure is available online.)

Conclusion

As PVS is a rare circumstance (either congenital or acquired) and misdiagnosis or management is frequent, early diagnosis is of paramount importance. According to studies with the longest follow-up durations, stenting is associated with a higher patency rate and lower restenosis and reintervention rates than BA. Even in patients with stenting (peripheral balloon-expandable stents or bare-metal stents), the rate of restenosis is remarkable (20% within 1 year). Accordingly, self-expandable drug-eluting stent implantation might be related to lower restenosis rates. Every effort should be made to implant larger stents (> 8 mm)[15] with appropriate lengths (a larger diameter with a shorter stent). The high incidence rate of restenosis necessitates intensive clinical follow-ups. Surgical repair can be considered in selected patients with 3 stenotic pulmonary veins or more severe PVS or complex cases with prior BA failure.[16]

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