Pulmonary vein stenosis: Etiology, diagnosis and management

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Pulmonary vein stenosis (PVS) is a rare condition characterized by a challenging diagnosis and unfavorable prognosis at advanced stages. At present, injury from radiofrequency ablation for atrial fibrillation has become the main cause of the disease. PVS is characterized by a progressive lumen size reduction of one or more pulmonary veins that, when hemodynamically significant, may raise lobar capillary pressure leading to signs and symptoms such as shortness of breath, cough, and hemoptysis. Image techniques (transesophageal echocardiography, computed tomography, magnetic resonance and perfusion imaging) are essential to reach a final diagnosis and decide an appropriate therapy. In this regard, series from referral centers have shown that surgical and transcatheter interventions may improve prognosis. The purpose of this article is to review the etiology, assessment, and management of PVS.

Key words: Pulmonary vein stenosis; Pulmonary vein stenosis etiology; Pulmonary vein stenosis causes; Pulmonary vein stenosis diagnosis; Pulmonary vein stenosis management; Pulmonary vein stenosis treatment

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the causes, diagnosis or treatment of pulmonary vein stenosis. However this is simple yet complete and updated review of all these matters that may guide physician’s decision making when facing a suspected or confirmed case of this unusual disease.

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INTRODUCTION

Despite pulmonary vein stenosis (PVS) is an uncommon entity (estimated incidence about 2-3 cases per year in large centers)¹ its morbidity and mortality rates are high at advance stages². The condition, linked in the past to congenital heart diseases in childhood and mediastinal processes (i.e., tumors) in adults, is nowadays firstly associated to injury from radiofrequency ablation (PVA) for atrial fibrillation (AF). It is essential to consider the possibility of the disease in patients at-risk to guarantee early detection (image techniques play a key role in this regard) and treatment. The aim of this article is to review the etiology, assessment and management of PVS.

ETIOLOGY

Congenital PVS

Congenital PVS is an exceptional abnormality (0.4% of congenital heart diseases) consequence of a failed incorporation of the common right and/or left PV into the left atrium (LA) during the embryologic development of the vessel that leads to partial or complete obliteration of the PVs on one or both sides³. From a histological point of view its main feature is an overgrowth of connective tissue with medial hypertrophy and intimal fibrosis which results in obstruction. Even though diagnosis is usually made within the first 3 years of life, it may be delayed till adulthood in some cases³. Congenital PVS is frequently associated (50%) with other cardiac defects⁴,⁵, hence imaging examination protocols applied to patients with congenital heart diseases should include a systematic evaluation of the PVs (Table 1).

Acquired PVS

PVA for AF

At the present time PVA for AF has become the principal cause of PVS. Incidence derived from recent studies reaches a mean and median of 2% and 3.1%, respectively. These figures represent a significant reduction in comparison with those reported in pioneer series (mean: 6.3% and median: 5.4%, estimated from papers published between 1999 and 2004)⁶. Main factors contributing to this finding are operator experience and improvements in the procedure [changing of ablation site from the PVs antra to ostia, reduction of temperature applied to tissue, cryoablation and intracardiac echocardiography (ICE) guidance]⁷. However real occurrence of PVS is probably underestimated as screening is only performed within the first 3 mo in some centers (it has been demonstrated that PVS can occur over this time period)⁸ and asymptomatic patients are not always imaged.

Mediastinal processes

Extrinsic compression by lymphadenopathies or granulomatous involvement may cause PVS in sarcoidosis⁹. Fibrosing mediastinitis, a rare complication of tuberculosis and Histoplasma capsulatum infection, characterized by uncontrolled fibrosis around the affected mediastinal lymph nodes, may lead to invasion and obstruction of the surrounding PVs⁹.

Neoplasm adjacent to the PVs may cause stenosis due to compression or infiltration⁴,¹⁰,¹¹.

Cardiovascular surgery

Clinically significant PVS in pediatric population is most frequently seen after total anomalous pulmonary venous return repair (estimated incidence approximately equal 10%)¹²,¹³. Obliteration may be localized either at the level of the anastomosis of the PV into the LA or further into the center of the vessel. Isolated cases of PV injury leading to obstruction after myxoma resection¹⁴, suture repair of a PV cannulation site¹⁵ and lung transplantation¹⁶ can be found in literature.

ASSESSMENT

PVS may be symptomatic when vein caliber is reduced significantly (> 50% stenosis), as a consequence of a raise in lobar wedge pressure, or lung perfusion is decreased by > 20%-25%¹⁷-¹⁹. Clinical manifestations, which in case of PVA normally appear 3-6 mo after the procedure, are clearly related to the number of PVs affected and include progressive exertional dyspnea, cough, chest pain (frequently following a

| Table 1 Causes of pulmonary vein stenosis |
|------------------------------------------|
| **Congenital**                            |
| Cardiac defects associated:              |
| Total anomalous pulmonary venous return  |
| Septal defects                           |
| Transposition of the great vessels       |
| **Acquired**                             |
| Pulmonary vein ablation                  |
| Sarcoïdosis                              |
| Neoplasm                                 |
| Fibrosing mediastinitis                  |
| Post cardiac vascular surgery            |

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pleuritic profile) and hemoptysis. Chest X ray may demonstrate signs of congestion (peribronchovascular and septal thickening, Kerlley B lines, alveolar edema) either diffuse or localized (mimicking other processes such as pneumonia), depending on the PVs involve.

Other findings can be found depending on the cause of the stenosis (i.e., lung size reduction in congenital PV atresia, a thoracic mass in the case of a tumor, mediastinal calcifications in fibrosing mediastinitis or calcified mediastinal lymph nodes in sarcoidosis). As the clinical picture is nonspecific, collateral flow development may mitigate symptoms, and occasionally physicians do not bear in mind the possibility of the disease, the diagnosis is commonly missed or delayed. Therefore screening with available imaging modalities in patients at risk (especially those with history of PVA) who develop respiratory symptoms is warranted.

Echocardiography

Transesophageal echocardiography (TEE) is a useful tool for PV investigation. Studies have shown high diagnosis accuracy for detection of PVS after PVA (sensitivity: 82%-100%, specificity: 95%-100%) compared to other techniques [computed tomography (CT), magnetic resonance imaging (MRI) and angiography]. Advantages of TEE are its wide availability, avoidance of radiation exposure, low cost, and applicability to patients with ferromagnetic implanted devices (i.e., pacemakers, defibrillators). There is no standard definition of PVS, nevertheless it seems that an increased maximum PV Doppler flow velocity (> 1.1 m/s) combined with color Doppler turbulence may be a reliable index (Figure 1).

ICE has been successfully used to guide PVA and evaluate PV ostial narrowing. The invasive nature of the technique restricts however its use to patients undergoing a redo PVA. Although diagnostic accuracy of ICE has not been investigated, an increased peak Doppler flow velocity over 1.6 m/s is consistent with PVS according to initial experiences.

CT

CT allows assessment of the extension of mediastinal neoplastic and non tumoral diseases infiltrating or compressing the PVs and enables the diagnosis of PVS after PVA by directly depicting vessel diameter (significant stenosis > 50%) (Figure 2). Although the choice of CT protocol depends on daily practice in every center ECG gated scanning improves quality and allows postprocessing with 3D reconstruction software which
Perfusion imaging
Perfusion of a pulmonary lobe draining to a PV with a significant stenosis may be decreased and detected using radionuclide quantitative pulmonary flow imaging (TC99m macroaggregated albumin) (Figure 4). This test however is not valuable for an etiological diagnosis of a PVS, may be altered in other pathologies that decreased lobar perfusion (i.e., pulmonary thromboembolism), is not suitable for detection of < 50% stenosis and may be inaccurate if significant compensating ipsilateral PV flow is present. Moreover, even small, it implicates radiation exposure (Table 2).

MRI
MRI is diagnostic in most cases by analyzing PV anatomy (MR angiography) and flow dynamics (MR phase contrast imaging; velocity and gradients across the vessel) (Figure 3). This modality can be also used to evaluate congenital cardiopathies and processes in the vicinity of the heart associated with a PVS (i.e., neoplasm). The main advantage of MRI over CT is that it does not expose the patient to radiation. Nevertheless drawbacks are considerable: Spatial resolution is lower than CT, it is contraindicated in patients with implanted non-compatible metal devices, and it may not be possible to perform in individuals with claustrophobia, unable to cooperate, large body habitus or severe renal impairment when gadolinium contrast is needed. Additionally, scanning time is considerably long.

MANAGEMENT
PVS in pediatric population
Mild and asymptomatic PVS may not need intervention;
clinical and image surveillance is advised as the disease can evolve over time. Surgery is the preferred approach in most congenital or acquired significant symptomatic PVS. The conventional interventions...
LA endocardium); and (2) pericardial patch venoplasty (resection of the stenotic tissue and patch anastomosis (Figure 5) include: (1) endarterectomy (excision of the stenotic ring and direct anastomosis of the PV to the LA ostium); B: Endarterectomy; the stenotic tissue has been excised and the PVs directly anastomosed to the LA; C: Pericardial patch venoplasty; the stenotic tissue has been resected and a pericardial patch anastomosis has been used to enlarge the tightened ostia of the vessels; D: Sutureless marsupialization; the veins ostia have been incised longitudinally, excess fibrotic tissue has been excised and in situ pericardial flaps have been sewn directly to the left atrium so direct stitches over the cut edges of the pulmonary veins are avoid. PV: Pulmonary vein; LA: Left atrium.

(Figure 5) include: (1) endarterectomy (excision of the stenotic ring and direct anastomosis of the PV to the LA endocardium); and (2) pericardial patch venoplasty (resection of the stenotic tissue and patch anastomosis

Figure 6  Stent implantation in a pulmonary vein stenosis. A: Angiography showing a critical stenosis in the ostium of the left lower pulmonary vein; B: Bare metal stent release; C and D: Final result. The stenosis was resolved. Normal flow can be seen in the main superior (C) and inferior (D) branches of the vein.
to enlarge the tightened segment). The newer sutureless marsupialization technique (the pericardium surrounding the affected PV is directly attached to the LA so direct stiches over the cut edges of the vessel are avoided) can help to prevent deformity of the suture line and reduce tissue growth stimulus decreasing therefore restenosis risk[27]. Overall, published surgical outcomes are modest; only half of cases are free from reintervention or death at 5 years[27,28]. Pneumectomy may be mandatory in cases of severe or uncontrolled hemoptysis and lung transplantation has been performed in patients with relentless PVS progression and severe pulmonary hypertension[29]. There is limited experience with percutaneous interventions in childhood; angioplasty is technically challenging (high pressures are needed to released stenosis and in case of stent implantation prosthesis should allow future expansion to adult dimension (> 12 mm) and results are suboptimal (repeated dilatations are frequently needed as instent restenosis rate is high)[30].

PVS in adult population

Transcatheter therapy is the most common chosen approach (Figure 6). While evidence of treatment of PVS due to extrinsic compression, infiltration or cardiac surgery is restricted to cases reports in literature[16] several small studies have evaluated the efficacy of percutaneous interventions for PVS after PVA. There are discrepancies among EP labs about management of asymptomatic PVS. Despite most authors recommend clinical and imaging monitoring every 3-6 mo in patients with 50%-85% stenosis, some promote angioplasty if a single stenosis > 75%[17] and others in cases of a cumulative stenosis index (average stenosis of the PVS of one site) > 75%[18]. Main arguments for early intervention are: Inadequate recovery of lung perfusion at advance stages caused by fixed venoconstriction leading to permanent pulmonary hypertension; and fast progression to PV occlusion in some cases which may be difficult to amend. Regarding the technique itself stenting appears better than isolated balloon venoplasty in terms of vessel restenosis (60% vs 36% for PV over 8 mm)[5]. Mid to long term patency is directly related to vessel size with higher rates of restenosis observed in PV < 1 cm. Drug eluting stents may have a better restenosis profile than conventional bare metal stent however studies regarding their use this scenario are scarce[31].

Limited data about antithrombotic regimes are available: (1) anticoagulation with warfarin, with an international normalized ratio target of 2-3, is generally recommended for at least 12 mo in the case of stents > 1 cm and indefinitely for those smaller[19], (2) dual antiplatelet therapy, added to anticoagulation, with aspirin plus clopidogrel is usually prescribed for a minimum of 3 mo, however optimal duration is not known; and (3) new oral anticoagulants (dabigatran, rivaroxaban, apixaban) or antiaggregants (prasugrel, ticagrelor) have not been tested.

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