Pulmonary vein stenosis: still the Achilles heel of ablation for atrial fibrillation?

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This editorial refers to 'Pulmonary haemodynamics at rest and during exercise in patients with significant pulmonary vein stenosis after radiofrequency catheter ablation for drug resistant atrial fibrillation'† by T. Arentz et al., on page 1410

Atrial fibrillation (AF) can be eliminated by (i) targeted delivery to focal ‘sites’ within the pulmonary vein (PV), by (ii) PV isolation through circumferential or segmental ablation at the venoatrial junction, or by (iii) electrical isolation of the left atrium (LA) outside the PV ostia. PV stenosis develops in 1–10% of patients undergoing ablation. So far, the clinical presentation, investigation, management, and outcome of this disease have been incompletely reported.

Understanding PV anatomy is crucial both for PV ablation and for prevention of PV stenosis. Using magnetic resonance (MR) scanning, Cato et al.¹ have reported that 38% of patients exhibit a variant anatomy (short or long common left trunk, right middle or right upper PV). In addition, very close proximity between the ostia of the right and the left PVs was observed. The diameters of the four regular PVs did not differ significantly, but PV and LA seemed larger in AF patients than in controls and left PVs showed a longer neck. PV ostia were usually oblong with the anteroposterior dimension lesser than the superoinferior dimension.

Taylor et al.² studied the pathological effects of extensive radiofrequency energy applications in the PVs in nine mongrel dogs. Histological examination revealed intimal proliferation with organizing thrombus, necrotic myocardium in various stages of collagen replacement, endovascular contraction, and a proliferation of elastic lamina.

Procedural parameters which may affect the development of PV stenosis are the targeted sites for energy delivery and energy requirements. By applying either focal or segmental ablation, reported rates for PV stenosis vary between 42 and <1%, depending on several factors including imaging technology, PV ablation methodology, and years of experience.³ ⁴ The prevalence for linear ablation is less well documented but is estimated to account for 2–7% of ablations. Analysing the perimetric distribution of radiofrequency energy in 37 consecutive patients undergoing ostial PV isolation in our institution, we have shown that, despite a trend, delivered energy showed no significant correlation with the degree of stenosis when serial computed tomography (CT) scans were performed at pre-defined time points before and after ablation.⁵

In the largest series reported so far dealing with the presentation, investigation, and management of symptomatic PV stenosis, Packer et al.⁶ reported 23 patients with severe stenosis of 34 PVs. Of the total number of patients, 52% had undergone a second and 22% a third procedure before symptom development, which occurred 1–3 months (103 ± 100 days) after the last ablation. The most frequent symptom reported was dyspnoea on exertion (83%), followed by dyspnoea at rest (30%), recurrent cough (39%), chest pain (26%), flu-like symptoms (13%), and haemoptysis (13%).

CT and/or MR scanning, transesophageal echocardiography (TEE), and lung scanning are widely used non-invasive imaging techniques used for detecting and visualizing PV stenosis.

We have used serial CT scanning prospectively the day before the procedure, before discharge, and at 3- and 6-month follow-up to detect, quantify, and predict the occurrence of PV stenosis, irrespective of symptoms.⁵ In our series of 37 patients undergoing ostial PV isolation by irrigated tip ablation, 6% of patients developed minor (<50% luminal narrowing) and 1.7% developed significant (>90% luminal narrowing) PV stenosis after a mean follow-up of 275 ± 100 days. The left inferior PV was at higher risk. Minor stenosed PVs showed no further progression after 3 months. In the Mayo clinic experience,⁶ the diameter at the site of maximal PV stenosis measured by CT scanning decreased from 14 ± 3 mm (baseline) to 3 ± 2 mm before PV dilatation/stenting, with the tightest region localized 12 ± 6 mm from the orifice and a stenosis length of 7–35 mm (mean 19 ± 6 mm) and the lesions appearing typi-

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conjunction with a decrease in the degree of stenosis from 80 to 9%. In addition, relative perfusion in lung scanning increased from 4 to 9%. However, there were procedural complications in four patients including transient ST-elevation, peripheral guidewire perforation with bleeding, dissection of the left superior PV, and a stent embolization to an iliac artery. Moreover, symptoms recurred 3.2 ± 2.8 months after the initial intervention and 14/23 patients (61%) exhibited in-stent or in-segment restenosis without a significant difference between PVS which were originally either dilated or stented. After multiple interventions, 15/23 patients (65%) were rendered asymptomatic after a follow-up of 18 ± 12 months. In our experience, the approach dealing with significant PV stenosis has been somewhat different. After performing serial CT scanning of the PVs before and after ablation irrespective of any symptoms, we decided to treat PV stenosis in a case of significant luminal narrowing (>70%) in conjunction with a functional abnormality in lung scanning with or without symptoms. Our rationale was to prevent possible undesired sequelae later on, such as PV occlusion, recurrent pneumonia, and pulmonary hypertension. We have performed a total of seven uneventful PV interventions in 6/92 (6.5%) consecutively screened patients. Despite restenosis in two dilated PVs of two patients, all six patients (with single vessel disease in five/six patients) remain asymptomatic after a post-interventional follow-up of 7 ± 2 months.

Arentz et al. report on pulmonary haemodynamics at rest and during exercise in patients with significant PV stenosis or PV occlusion after AF ablation. Their series consists of 111 patients (9.4%) who underwent initial MR scanning 12–24 months after the ablation procedure. All patients were either asymptomatic or only mildly symptomatic at this point in time, hence none of these was considered for PV angioplasty.

Patients were re-examined using MR scanning and additional Swan Ganz right heart catheterization at rest and during exercise after a total follow-up of 50 ± 15 months following the last procedure. When compared with prior MR imaging, no significant changes were noted. At rest, no patient had pulmonary hypertension. However, at 100 W, seven patients had elevated PA pressures, three of them probably caused in part by left ventricular dysfunction. All three patients with stenosis/occlusion of two PVs were affected.

This is an interesting study because of various reasons. First, it sheds light on the natural course of asymptomatic significant PV stenosis over a long-term follow-up if left untreated. Referring to a previous paper by the same authors only 3/13 patients (23%) were symptomatic 2 years after the index ablation. Their finding is consistent with the experience of other working groups, including our own, that particularly a single PV vessel disease is often only mildly symptomatic or even asymptomatic. In addition, symptoms occurring within the first months after ablation may subside because of collateral vessel formation or even spontaneous resolution or PV stenosis. Secondly, PV luminal diameters stabilize after the initial 12–24 months, as demonstrated by MR imaging. Finally, the risk for developing pulmonary hypertension either at rest or during exercise seems low for single PV stenosis. On the contrary, multivessel veno-occlusive disease appears to be at a higher risk. However, as the authors correctly point out, their sample size is too small to be able to draw any firm conclusions. Results will have to be confirmed in larger studies. Additionally, there is a lack of a control group of AF patients undergoing right heart catheterization without PV stenosis or after successful PV angioplasty.

The best strategy for PV stenosis is of course to prevent it. High volume ablation centres claim that the prevalence of PV stenosis has steadily decreased in their centres over the last years. The most important determining factors for this drop are the abandonment of in-vein focal ablation, ablation at/outside the PV orifice, reduction in ablation temperature and amount of energy, the use of intracardiac echocardiography or various 3D-mapping systems and, last but not least, increased operator experience.

There is still a lot to learn. The following are some of the unresolved issues. Which is the optimal PV ablation approach to prevent PV stenosis (energy setting, energy source, ablation technique)? Which is the optimal technique for PV angioplasty (dilatation and/or stenting, drug-eluting stents, antithrombotic drug regimen to prevent restenosis)? How does one treat asymptomatic single PV stenosis? What is the long-term effect of exercise-induced pulmonary hypertension in this population? On the basis of the findings of Arentz et al., a randomized prospective trial dealing with single PV vessel disease in asymptomatic or only mildly symptomatic patients and comparing a conservative with an interventional approach may be warranted. In addition, an international multicenter PV stenosis registry should be encouraged.

Will PV stenosis remain the Achilles heel of AF ablation? The answer is: probably not. However, with the currently widespread application of AF ablation, it is here to stay for the years to come.

References
