17. Pulmonary Hypertension After Atrial Fibrillation Ablation: A Case Report

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Body

Background: Pulmonary hypertension (PH) is a clinical condition characterized as an increase in mean pulmonary arterial pressure(mPAP) ≥25 mmHg at rest as assessed by right heart catheterization[1]. It includes precapillary, post-capillary and mixed PH. The causes of PH range from single organ diseases to systemic diseases, e.g., chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), yet some are unknown. According to the guideline published by the European Society of Cardiology (ESC) and the European Respiratory Society (ERS) in 2015[2], the clinical classification categorized into five groups: pulmonary arterial hypertension; PH due to left heart disease; PH due to lung disease and/or hypoxia; chronic thromboembolic PH and other pulmonary artery obstructions; and PH with unclear and/or multifactorial mechanisms. Globally, left heart and lung diseases have become the most frequent causes of PH, especially left heart failure and chronic obstructive pulmonary disease[1]. Here we investigated a case of PH caused by left-side heart disease. The patient suffered from multiple pulmonary veins stenosis(PVS) after catheterradiofrequency ablation that belongs to Group 2. The case indicates that some uncommon causes in a common classification should be emphasized in clinical practice.

Case: A 58-year-old woman presented with repeated chest discomfort and shortness of breath over 2 months. Echocardiography in the other hospital indicated severe PH. On admission, physical examination simply revealed a pronounced pulmonary valve second heart sound (P2＞A2), without rales in the lungs and edema. Both Troponin I high-sensitive and NT-proBNP were within normal range. The initial electrocardiogram showed depression of ST segment (Ⅱ, Ⅲ, aVF), and bidirectional or inverted T wave (Ⅲ, aVF). The second echocardiography was similar as the last one, with extremely high estimated pulmonary arterial pressure (PAP) of 79mmHg, moderate tricuspid regurgitation and normal left ventricle ejection fraction (LVEF 58%). Chest computed tomography (CT) showed scattered infectious lesions in the lungs with a small amount of pleural effusion on both sides. No obvious abnormality was found in coronary arteries and pulmonary artery computed tomography angiography (CTA). The patient had no history of hypertension, diabetes mellitus, congenital heart disease, and chronic obstructive pulmonary disease. Additionally, drugs and toxins recognized for inducing PAH had not been taken. Based on these evidences, PH due to congestive heart failure, lung disease and pulmonary artery obstructive disease could be excluded. To investigate further, the history of catheterradiofrequency ablation for atrial fibrillation(AF) before approximately 4 months was found out by asking for her medical history in detail again. Therefore, we reviewed the imaging of pulmonary artery CTA, then PVS was suspected. Subsequently, PVCTA disclosed the right middle PV and both left superior and inferior PVs near the left atrium stenosis, especially the left superior one was almost occluded. Consequently, PVS was considered as the main cause of PH. After 3 months of surgery, the patient's symptoms were relieved and the LVEF was reduced to 45% as indicated by color doppler echocardiography.

According to comprehensive clinical classification of PH (updated from Simonneau et al.), the patient belonged to Group 2. Based on the cause, the management of the underlying condition was recognized as the primary therapy. Namely, pulmonary venoplasty and tricuspid valvuloplasty were performed. Results of PV CTA were confirmed by operation findings. Postoperative symptoms were significantly improved. The patient presented for a control echo, which revealed that the estimated PAP dropped to 45mmHg. PVS post-ablation was identified as the cause of PH.

Discussion: The condition PH contains a series of diseases characterized by an increase in mean pulmonary arterial pressure (mPAP)≥25 mmHg at rest, including lung diseases, pulmonary embolism, left heart disease, drugs or toxic induced and connective tissue disease, et al[3]. Besides, a new standard of mPAP≥20mmHg was proposed at the 6th World Symposium on Pulmonary Hypertension[4], but it is not writen into guidelines. The etiology and incidence may be different in different regions. Generally, around 1% of the global population suffer from PH, and the incidence in those over 65 years old is up to 10%[5]. However, the etiology of PH is particularly complicated. It is acknowledged widely that the leading causes are left heart disease and lung disease. Subsequently pulmonary arterial hypertension, chronic thromboembolic PH, and PH with unclear causes are considered[6]. Hence the recommended diagnostic procedure initiates once PH is thought of suspicion and echocardiography is in accorded with the condition. Subsequently, prior to the recognition of the principal groups of PH [Group 2(due to left heart disease) and Group 3 (due to lung disease and/or hypoxia)], then discriminates Group 4 (chronic thrombo-embolic PH), and eventually differentiates the possible causes in Group 1(pulmonary arterial hypertension) and Group 5 (unclear and/or multifactorial mechanisms)[2]. The algorithm was applied as above in this case, firstly we investigated the left heart failure and relevant lung disease. When the leading causes were excluded, the investigation of other diseases was initiated. It implies that those with unknown causes should positively be investigated and not be simply classified as idiopathic pulmonary arterial hypertension (IPAH). However a special disease in Group 2 was ignored unfortunately, namely acquired PVS. Finally, the patient was clearly diagnosed with PH due to PVS after radiofrequency ablation. The case obviously demonstrates that it is crucial to consider the uncommon causes among the leading groups and the normative procedure should be implemented seriously.

PVS is one of complications of atrial fibrillation (AF) ablation[7]. It is a consequence of thermal injury at the PV-antral junction or inside the PV ostia with the release of radiofrequency ablation(RFA) energy. In the injury process, intimal proliferation, endovascular contraction and myocardial collagen replacement are involved. Ultimately, irreversible damage occurs in venous tissue leading to the narrowing of PV[8]. Widely utilization of catheter ablation as an intervention therapy option in the treatment of atrial fibrillation results that an increasing of individuals may be at risk of PV stenosis[9]. The PVS incidence is not clear yet on account of the lack of authoritative trials and sufficient data[10]. Schoene et al.[8] performed a single-center observational study that evaluated the radiofrequency-induced PVS patients during a thirteen-year span from 2004-2017, and reported that the total incidence of PVS following AF ablation was 0.78%. A multiple-center post-history study by Samuel et al. showed an incidence of 20.8% (41 of 197) in ADVICE trail[11]. Due to the diversity of symptoms, patients may complain of progressive exercise dyspnea, cough, chest discomfort, and fatigue. Some individuals remain asymptomatic if just single-vessel stenosis, even a total occlusion, or mild to moderate stenosis. In these cases, identification of this complication is potentially postponed and the diagnosis may be misleading. It is the character of unspecific and obscure presentation of PVS that adds to the difficulty of recognition and makes the diagnosis process complex[12, 13]. Consequently, not only cardiologists but respirologists need to pay

attention to the disease. The treatment strategy has no a optimal choice. Some reports suggest that stent implantation should be considered as a first-line therapy in patients with radiofrequency-induced severe PVS[14], other reports indicate that surgical repair should be routinely considered as a therapeutic option in individuals with multiple PVS[15]. Considering the recurrence, surgical repair was selected. The patient we concerned presented with repeated chest discomfort and shortness of breath. After excluding left-side heart failure, lung disease and thrombo-embolic PH, our team reviewed the medical history seriously again, ultimately RFA-induced PVS was clearly diagnosed. What the medical history remains pivotal in practice is no more than a platitude, but we have to underline the details once again.

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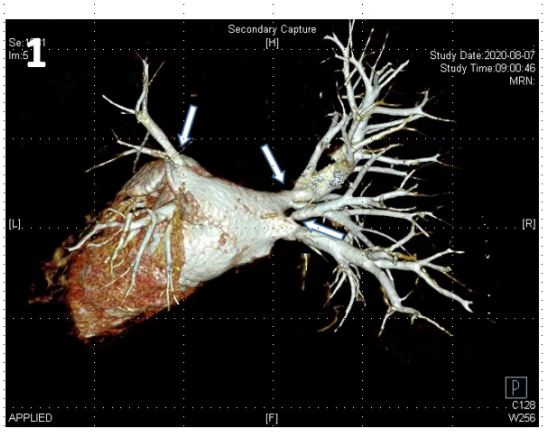

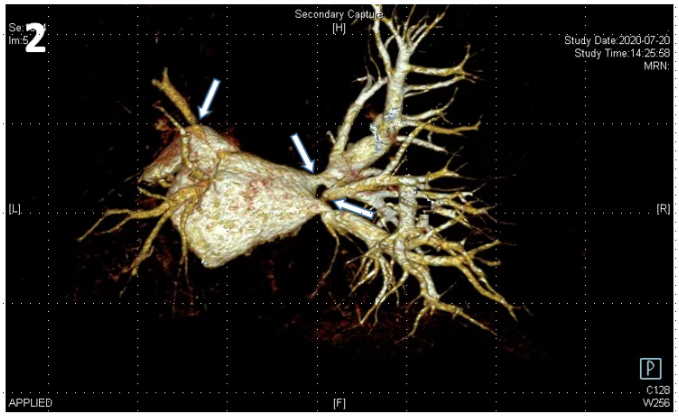

Figure1&2. Pulmonary vien computed tomography angiography: the right middle pulmonary vein and both left superior and inferior pulmonary veins near the left atrium stenosis, especially the left superior pulmonary vein was almost occluded.(the diameter of venous entrance: left superior pulmonary vein 2.5mm, the inferior one 2.2mm; right superior vein 5.1, the middle one 2.5, the inferior one 6.6). The points that arrows refer to is the lesions.