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Medical Treatment of A Symptomatic Acute Pulmonary Vein Stenosis Following Antral Pulmonary Vein İsolation

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Abstract

Pulmonary vein (PV) stenosis is a rare but serious complication of PV isolation. It usually develops 3-6 months after the procedure, but may rarely develop in the acute phase. We present a case of symptomatic PV stenosis within 48 hours after antral PV isolation. Following the initiation of medical treatment including a glucocorticoid, acute changes in the PV wall regressed and the patient's complaint of dyspnea at rest improved rapidly. In addition, long-term renin-angiotensin-aldosterone system (RAAS) blockers were given. The symptoms did not recur during follow-up and PV stenosis was mild at 6 months.

Introduction

Pulmonary vein (PV) stenosis is a rare but serious complication that can be seen following radiofrequency catheter ablation for atrial fibrillation (AF). Clinical presentation may be asymptomatic or symptomatic with severe dyspnea, cough, chest pain, and hemoptysis[1]. Although PV stenosis usually develops 3-6 months after the procedure, it may rarely develop in the acute phase[2]. We present a case of symptomatic moderate PV stenosis within 48 hours after antral PV isolation. Following the initiation of medical treatment including a glucocorticoid, acute changes in PV regressed and the patient's complaint of dyspnea at rest improved rapidly. In addition, long-term renin-angiotensin-aldosterone system (RAAS) blockers were given. The symptoms did not recur during follow-up and PV stenosis was mild at 6 months.

Case

51 year-old woman was admitted to our center for palpitation attacks resistant to antiarrhythmic therapy. She had undergone electrical cardioversion due to persistent AF in our center one year ago. However, AF recurred 6 months after the cardioversion. She had a history of systemic hypertension. Medications were metoprolol 50 mg once a day, dabigatran 150 mg twice a day and ramipril/hydrochlorothiazide 5/25 mg once a day. The patient was taken to the electrophysiology laboratory for persistent AF ablation after consent was obtained. A contrast-enhanced thorax computed tomography (CT) was performed before the procedure (Figure 1A). The procedure was performed under general anesthesia. Double transseptal punctures were performed and a three-dimensional electroanatomic left atrium map (EnSite Precision™ Cardiac Mapping System, St Jude Medical, St. Paul, Minnesota, MN, USA) was obtained. Point-to-point linear lesions were created with an irrigated tip catheter (The TactiCath™ Quartz ablation catheter, St Jude Medical) in the antrum regions of the right and left PVs (Figure 2). Radiofrequency energy was delivered with a maximum power of 30 W at the anterior wall and 25 W at the posterior wall. Maximum temperature was 42 °C and irrigation flow rate was 17 ml/min.

Sudden onset dyspnea at rest developed 36 hours after the procedure. Physical examination revealed crepitant rales in the basal region of the lungs. Transthoracic echocardiography did not demonstrate pericardial effusion or any left ventricular or valvular dysfunction. Chest X-ray revealed pulmonary edema. The patient was given intravenous (IV) furosemide and IV glyceryl trinitrate for 48 hours. The second CT taken 48 hours after the procedure showed 51.9% stenosis in the left upper PV and swelling of the PV wall (Figure 1B). There was no bronchial wall thickening and near parenchymal ground glass opacities which could be thought as bronchial damage near

Key Words
Atrial fibrillation, Ablation, Same day discharge, Complications.

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to ablation area. 100 mg IV methylprednisolone was administered immediately after the CT and 60 mg daily was continued for additional 4 days. Four days after the procedure, the patient's blood pressure (BP) increased (160/100 mmHg) and serum potassium level decreased (3.4 meq/L). Spironolactone 25 mg once a day was added and furosemide was discontinued. The patient's complaint of dyspnea disappeared 5 days after the procedure and BP returned to normal. The control CT at 7 days after the procedure showed decreasements in the thickness of the PV wall and in the degree of PV stenosis (41.7%) (Figure 1C). The patient was discharged 8 days after the ablation and received 20 mg fluocortolone daily for 5 days at home.

At 6 months after the procedure, the patient had no clinical complaints. The patient was taking ramipril/hydrochlorothiazide and spironolactone at 6 months after the procedure. Follow- up CT showed that the PV wall, the PV wall thickness was normal and there was less stenosis in the PV (38.5%) than the baseline (Figure 1D).

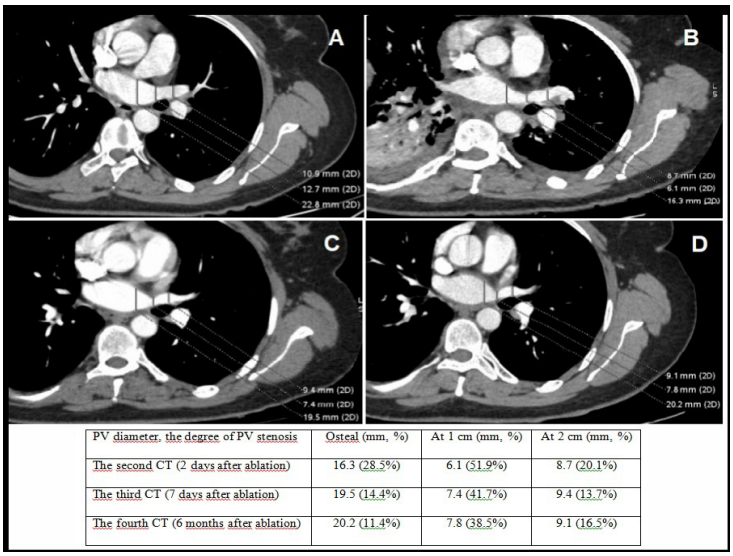
Discussion:

Here, we describe a patient who developed symptomatic moderate PV stenosis in the acute phase after the procedure despite ablation away from the PVs. After the initiation of corticosteroid therapy in the early period, acute changes in the PV wall regressed, and pulmonary congestion recovered rapidly. At 6 months after the procedure, PV stenosis was mild and the patient had no clinical findings.

The PV stenosis is classified as mild (30% to 50%), moderate (50% to 70%), and severe (>70% diameter reduction)[1]. While it was reported up to 42% in early procedures, the incidence of severe PV stenosis after AF ablation has decreased below 1% in the current era[1,3]. However, the actual incidence is unknown because some patients are asymptomatic or symptoms are nonspecific. Symptoms may vary depending on the number of PVs affected, the severity of PV stenosis, the response of the pulmonary vascularity to the lesion, the presence of collaterals, clinical setting, and rate of development of PV stenosis[4]. Promising strategies to reduce PV stenosis include ablation away from PVs, use of an open irrigated-tip ablation catheter, and the use of three-dimensional mapping systems[3,5].

Immediately after ablation, vessel narrowing and PV wall edema were proposed as mechanisms of PV stenosis[5]. Shrinkage due to heat-induced contraction, especially the contraction of the internal elastic lamina, has been proposed as the mechanism of PV diameter reduction[5,6]. Chronic inflammation, intimal proliferation and myocardial fibrosis cause late onset PV stenosis after ablation[3,6].

Corticosteroids have antiinflammatory effects by increasing the transcription of anti-inflammatory cytokines and by reducing the transcription of inflammatory cytokines[7]. The RAAS system is responsible for structural and electrical remodeling in the heart, including the development of fibrosis. The RAAS blockers have been shown to reduce cardiac fibrosis and decrease the expression of inflammatory markers in human and animal models[8]. In a piglet model with PV stenosis, Zhu et al showed that losartan reduced intimal hyperplasia by maintaining vascular endothelial cadherin levels[9].



PV diameter, the degree of PV stenosis	Osteal (mm, %)	At 1 cm (mm, %)	At 2 cm (mm, %)
The second CT (2 days after ablation)	16.3 (28.5%)	6.1 (51.9%)	8.7 (20.1%)
The third CT (7 days after ablation)	19.5 (14.4%)	7.4 (41.7%)	9.4 (13.7%)
The fourth CT (6 months after ablation)	20.2 (11.4%)	7.8 (38.5%)	9.1 (16.5%)

Figure 1: **The CT image of the left superior PV in the axial plane A) Before B) 48 hours after C) 7 days after D) 6 months after the procedure. The PV diameter were measured at the ostial level, at 1 and 2 cm more distally. At each level, PV diameter was measured with a manual caliper as the distance between 2 points perpendicular to the PV axis. Post-ablation PV diameter was compared with the pre-ablation and the degree of PV stenosis was calculated at each level. The table below shows PV diameter, the degree of PV stenosis at the measured levels, and the date the CT was taken. CT: Computed tomography, PV: Pulmonary vein.**

In our case, although the ablation site was far from the PV ostia, PV diameter decreased in the acute period after the procedure. The possible mechanism in our case was acute tissue edema due to ablation. Edema may extend well beyond the ablation site[6]. Although PV stenosis was moderate, pulmonary edema symptoms developed. This may be due to the rapid development of changes in the PV wall. An acute reduction in atrial natriuretic peptide level may have contributed to the pulmonary congestion in our case. There are other methods other than CT to diagnose PV stenosis such as perfusion scans or pulmonary venography[3]. In our case, we only performed CT, and in this way, we diagnosed PV stenosis and were able to eliminate other possible causes. She was in pulmonary edema and we did not perform a more invasive imaging method in view of clinical benefit/ risk ratio.

Glucocorticoid treatment was initiated within the first 48 hours after the procedure. After steroid treatment, acute changes in the PV wall decreased dramatically and the pulmonary congestion resolved. Glucocorticoids may have caused regression of the acute changes in the PV wall with anti-inflammatory effects[7]. In addition, we started spironolactone due to hypertension and hypokalemia. The RAAS blockers may have contributed to the prevention of the development of PV stenosis in the long term with their antifibrotic effects.

Some of the patients undergoing PV isolation may have some degree of PV stenosis immediately after the procedure. Jin et al demonstrated that some degree of PV stenosis was seen immediately after PV isolation in 14 (16%) of 87 veins in 12 (46%) of 26 patients[10]. Berkowitsch et al. showed that a 25% reduction in PV diameter was observed immediately after ablation in 36 of 357 PVs[11]. In our case, the degree of stenosis at the 6th month was

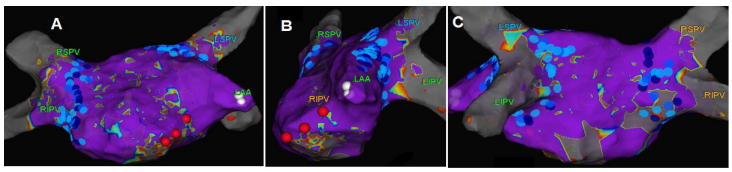


Figure 2: **Three-dimensional electroanatomic mapping views of the patient from A) Anteroposterior projection B) Left anterior oblique projection C) Posteroanterior projection. LAA: Left atrial appendage, LIPV: Left inferior pulmonary vein, LSPV: Left superior pulmonary vein, RIPV: Right inferior pulmonary vein, RSPV: Right superior pulmonary vein.**

mild (38.5%). Acute changes in the PV diameter may lead to the development of PV stenosis in the chronic period[5]. For this reason, it may be important to monitor patients with mild / moderate PV stenosis after the procedure for the development of more severe PV stenosis. We planned to follow our patient with an annual CT to monitor the degree of PV stenosis.

We left the circular mapping catheter in the PV ostium during ablation to demonstrate the PV ostium. However, it should be kept in mind that the three-dimensional mapping system may not show the PV ostium completely during ablation and there may be a shift in the map. Stenting is an important treatment option in individuals with symptomatic significant PV stenosis after AF ablation. Stenting is superior to balloon angioplasty in terms of reducing restenosis[1]. However, restenosis may occur in 24% of cases after stenting in such cases[12].

In summary, acute PV stenosis may rarely develop after antral PV isolation. It is very important to keep in mind the possibility of PV stenosis in patients who develop consistent clinical findings after such a procedure. Acute changes in the PV wall can be reversed by intensive corticosteroid therapy in the early period. Again, the RAAS blockers may contribute to the prevention of the development of higher grade PV stenosis in the future in such patients. However, further studies are needed to clarify this issue.

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