

Late onset cardiac cirrhosis and portal hypertensive ascites after atrial fibrillation ablation

Tse-Ling Fong¹, Michael Fong², Jerold Shinbane², Vaughn Starnes² and Helga Van Herle²

¹Division of Gastrointestinal and Liver Diseases, Keck School of Medicine, Los Angeles, CA, USA; ²CardioVascular Thoracic Institute, Keck School of Medicine, Los Angeles, CA, USA

Abstract

Pulmonary vein stenosis is a potential complication following catheter ablation of atrial fibrillation (AF). We report the case of a patient with refractory ascites late after multiple catheter ablation procedures for AF. This is the first case report of portal hypertensive ascites due to acquired multiple pulmonary vein stenoses resulting in pulmonary hypertension (PH) and cardiac cirrhosis late after AF ablation. Despite extensive surgical reconstruction of the affected pulmonary veins, the patient has PH and right heart failure with persistent ascites and lower extremity edema.

Keywords

ablation, atrial fibrillation, cardiomyopathy, complications, pulmonary hypertension

Date received: 7 August 2018; accepted: 26 November 2018

Pulmonary Circulation 2018; 9(1) 1–3

DOI: 10.1177/2045894018813559

Case report

A 62-year-old man with myocardial infarction 13 years prior and stenting of the right coronary artery developed chronic atrial fibrillation (AF) requiring transcatheter radiofrequency ablation on three occasions over a 2.5-year period at an outside institution, with AF ultimately becoming permanent. The patient also had placement of a dual-chamber pacemaker for sick sinus syndrome. Eight months after his last ablation, he developed exertional dyspnea, weight gain, lower extremity edema, and abdominal distention. He was previously physically active and bicycled over 70 miles/day, five days/week. He did not drink alcohol. Abdominal ultrasound revealed ascites and a nodular appearing liver. Six months after onset of ascites (14 months after last catheter ablation), he underwent 5.5-L large volume paracentesis (LVP). He had two additional LVPs before presenting with worsening dyspnea and acute kidney injury complicated by hyperkalemia, requiring emergency temporary hemodialysis. His renal function recovered, and he was diagnosed with pneumonia and cryptogenic cirrhosis complicated by portal hypertension.

The patient was referred for work-up of liver disease and management of refractory ascites. Physical examination

revealed jugular venous pressure of 12 cm H₂O, respirophasic II-III/VI holosystolic murmur at the left lower sternal border with a right ventricular heave, a palpable, enlarged, and pulsatile liver and moderate ascites with peripheral edema.

Laboratory studies showed BUN 22 mg/dL, creatinine 1.02 mg/dL, total protein 6.8 g/dL, albumin 4.2 g/dL, alkaline phosphatase 281 U/L, total bilirubin 1.4 mg/dL, ALT 12 U/L, AST 29 U/L. Viral hepatitis and autoimmune serologies were negative. Ascites characterization revealed white blood cells 598 /mm³ (23% polymorphonuclear cells), protein 3.3 g/dL, and albumin 2.1 g/dL (serum-ascites albumin gradient 1.9 g/dL). Liver transient elastography (FibroScan Echosens, Paris, France) revealed liver stiffness 16.8 kPa, consistent with stage 4 fibrosis. Transjugular liver biopsy showed sinusoidal dilatation and focal sinusoidal fibrosis. Free and wedged hepatic vein pressures were 16 and 18 mmHg, respectively.

Corresponding author:

Tse-Ling Fong, 1510 San Pablo Street, 3/F, Los Angeles, CA 90033, USA.

Email: tselingf@usc.edu



Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 License (<http://www.creativecommons.org/licenses/by-nc/4.0/>) which permits non-commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the SAGE and Open Access pages (<https://us.sagepub.com/en-us/nam/open-access-at-sage>).

© The Author(s) 2018.

Article reuse guidelines:
sagepub.com/journals-permissions
journals.sagepub.com/home/pul



Chest X-ray demonstrated a right pleural effusion. Transthoracic echocardiogram revealed left ventricular ejection fraction of 65% with interventricular septal flattening during diastole. The right ventricle was moderately dilated with severely reduced systolic function. Peak velocity of 1.2 m/s was noted in the right superior pulmonary vein (RSPV) as it entered the left atrium, with turbulent flow evident at the inflow of the left upper pulmonary vein. The left atrium was normal in size with a volume index of 20 mL/m² by biplane method. There was moderate tricuspid regurgitation. Peak pulmonary artery systolic pressure (PASP) was estimated at 55 mmHg. Grade 1 diastolic function was noted. Cardiac computed tomography angiography (CTA) revealed an enlarged main pulmonary artery with severely narrowed ostia of the RSPV (1–2 mm), left superior pulmonary vein (LSPV) (4 mm), and left inferior pulmonary vein (LIPV) (2 mm) (Fig. 1a). The right inferior pulmonary vein was widely patent. Heart catheterization findings were as follows: right atrial pressure = 24 mmHg; right ventricular pressure = 72/24 mmHg; pulmonary artery pressure (PAP) = 63/27 mmHg; pulmonary wedge pressure = 35 mmHg; cardiac index = 1.48 L/min/m²; pulmonary vascular resistance = 4.94 Woods units; systemic vascular resistance = 14.81 Woods units; left ventricular end diastolic pressure (LVEDP) = 11 mmHg; and severe tricuspid regurgitation.

The patient was evaluated by both interventional cardiology and cardiothoracic surgery services and it was felt that, given the concomitant degree of tricuspid regurgitation, the patient would have a better outcome with surgical repair. The patient underwent surgical repair of the PVS where the RSPV, LSPV, and LIPV were confirmed to be stenotic with calcium deposited around the orifices. RSPV was incised and reconstructed with harvested pericardium. Reconstruction of LSPV and LIPV was performed with the

veins opened and sewn together posteriorly with the left atrial appendage opened and sewn over the left upper and lower pulmonary veins as a hood. The tricuspid valve was repaired with a 30-mm annuloplasty band.

Postoperative transthoracic echocardiogram showed a decrease in right ventricular size with improved systolic function, peak PASP of 37 mmHg, and resolution of tricuspid regurgitation. There was transient improvement in his fluid retention; however, six months after surgical reconstruction, he redeveloped ascites that required LVP every two months. Repeat right heart catheterization showed persistent pulmonary hypertension: PAP = 60/31 mmHg. Ascites is currently controlled with diuretics—bumetanide and spironolactone—but he has azotemia and hyponatremia.

Discussion

Our patient, who had no risk factors for chronic liver disease, developed cardiac ascites characterized by high SAAG and protein concentration. High SAAG was indicative of a portal hypertensive etiology. Low wedged-free hepatic vein gradient (<5 mmHg) was compatible with post-hepatic portal hypertension.¹ We propose that this patient developed severe pulmonary hypertension (PH) because of PVS after catheter ablation resulting in elevated right-sided heart pressures, congestive hepatopathy, and ascites.² These events occurred insidiously without the patient experiencing respiratory symptoms which are the more common manifestation of serious complications associated with PVS following ablation for AF.^{3–5} This patient progressed to advanced liver fibrosis which has been shown to correlate with severity of tricuspid valve regurgitation, renal dysfunction, and abnormal (obstructive pattern) liver tests, but not necessarily the duration of heart failure.⁶

Changes seen in congenital vein stenosis involving “upstream” pulmonary veins and progressive PH have been replicated in piglets subjected to bilateral pulmonary vein banding. Reduction of pulmonary vein obstruction in the banded animals, by stent placement in the right middle pulmonary vein, resulted in some reversal of local pulmonary venous hypertension and endothelial-mesenchymal transition and return of endothelial markers.⁷

Percutaneous catheter interventions and surgical approaches have been used to treat PVS after AF ablation. The high rate of PV restenosis after balloon angioplasty (30–87%) has decreased with stenting (14–57%).⁸ Surgical approaches to PVS include endarterectomy, pericardial patch venoplasty, and sutureless pericardial marsupialization with similar results. Surgical repair factors that are associated with poorer prognosis include a greater number of pulmonary veins involved and smaller upstream indexed total pulmonary vein cross-sectional area.⁹

This is the first case report demonstrating cardiac cirrhosis and portal hypertensive ascites due to acquired multiple PVS late after AF ablation. The location and severity of the stenoses were demonstrated by CTA. Despite extensive

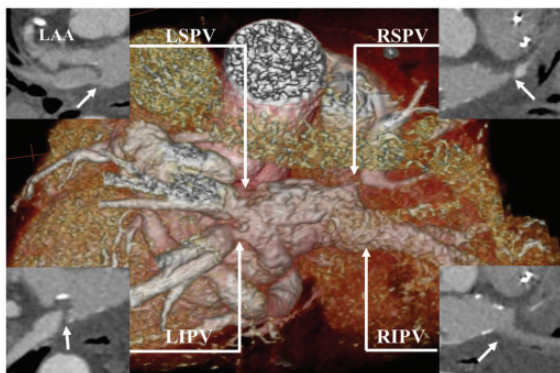


Fig. 1. Preoperative CTA three-dimensional (center) and two-dimensional double oblique views (corners) views demonstrating severe stenoses of the LSPV, LIPV, and RSPV. LAA, left atrial appendage; LSPV, left superior pulmonary vein; LIPV, left inferior pulmonary vein; RSPV, right superior pulmonary vein; RIPV, right inferior pulmonary vein.

surgical reconstruction of the affected pulmonary veins, patient has persistent PH and cardiac ascites.

Conflict of interest

The author(s) declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

References

1. Runyon BA. Cardiac ascites: a characterization. *J Clin Gastroenterol* 1988; 10: 410–412.
2. Verma I, Tripathi H, Sikachi RR, et al. Pulmonary hypertension due to radiofrequency catheter ablation for atrial fibrillation: The lungs, the atrium or the ventricle? *Heart Lung Circ* 2016; 25: 1177–1183.
3. Holmes DR Jr, Monahan KH and Packer D. Pulmonary vein stenosis complicating ablation for atrial fibrillation: clinical spectrum and interventional considerations. *JACC Cardiovasc Interv* 2009; 2: 267–276.
4. Cappato R, Calkins H, Chen SA, et al. Updated worldwide survey on the methods, efficacy, and safety of catheter ablation for human atrial fibrillation. *Circ Arrhythm Electrophysiol* 2010; 3: 32–38.
5. Edriss H, Denega T, Test V, et al. Pulmonary vein stenosis complicating radiofrequency catheter ablation for atrial fibrillation: A literature review. *Respir Med* 2016; 117: 215–222.
6. Gelow JM, Desai AS, Hochberg CP, et al. Clinical predictors of hepatic fibrosis in chronic advanced heart failure. *Circ Heart Fail* 2010; 3: 59–64.
7. Kato H, Fu YY, Zhu J, et al. Pulmonary vein stenosis and the pathophysiology of “upstream” pulmonary veins. *J Thorac Cardiovasc Surg* 2014; 148: 245–253.
8. Rostamian A, Narayan SM, Thomson L, et al. The incidence, diagnosis, and management of pulmonary vein stenosis as a complication of atrial fibrillation ablation. *J Interv Card Electrophysiol* 2014; 40: 63–74.
9. Lo Rito M, Gazzaz T, Wilder TJ, et al. Pulmonary vein stenosis: Severity and location predict survival after surgical repair. *J Thorac Cardiovasc Surg* 2016; 151: 657–666.