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CASE REPORT

Inflammatory Response and Congestive Heart Failure Following Extensive Left Atrial Ablation

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KEY WORDS: *atrial fibrillation; pulmonary vein isolation; left atrial ablation; heart failure; radiofrequency catheter ablation; systemic inflammation*

Abbreviations
AF = atrial fibrillation
CFAEs = complex fractionated atrial electrograms
Hs-CRP = high sensitivity C-reactive protein
NT-pro-BNP = N-terminal pro-brain natriuretic peptide
PV = pulmonary vein

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ABSTRACT

A case of a patient with persistent atrial fibrillation and normal left ventricular systolic function who developed acute heart failure symptoms after a procedure of extensive left ablation is presented and possible aetiologies are being discussed.

INTRODUCTION

Catheter ablation is increasingly utilized in patients with atrial fibrillation (AF).[1] For patients with paroxysmal AF, the ablation procedure is limited to pulmonary vein isolation, but for persistent AF, some investigators have proceeded to more extensive strategies of left atrial ablation, which may confer a higher complication rate.[2] Development of heart failure has not been been recognized until recently as a complication of this procedure, particularly in patients undergoing more extensive left atrial ablation.[3] We herein describe a patient who developed such a complication the next day after the procedure.

CASE PRESENTATION

A 67-year-old man with non-obstructive hypertrophic cardiomyopathy and persistent AF underwent left atrial catheter ablation. Following a single transseptal puncture, the three-dimensional geometry of left atrium was reconstructed using the CARTO 3 system (Biosense Webster, Inc., Diamond Bar, Calif., USA). Wide circumferential lesions around both ipsilateral pulmonary veins (PVs) aiming at electrical isolation and ablation of complex fractionated atrial electrograms (CFAEs) were performed using a 3.5-mm-tip ablation catheter (Thermo Cool Navi-Star, Biosense Webster, Inc., Diamond Bar, Calif., USA). Following this initial ablation strategy, AF was not terminated, and sinus rhythm was restored with direct current cardioversion. Roof and mitral isthmus lines aiming at bidirectional block were subsequently performed. Additional lesions were delivered within coronary sinus. Overall, 3.9 liters of fluids

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were administered during the procedure.

The next day after the procedure, the patient developed mild dyspnea, while pulmonary rales were audible at the lower lung fields. Transthoracic echocardiography showed normal left ventricular systolic function with asymmetric septal hypertrophy (20 mm), left ventricular diastolic dysfunction, and left atrial enlargement (45 mm). The presence of pericardial effusion was excluded. Computed tomography scan was compatible with pulmonary congestion and ruled out pulmonary embolism or PV stenosis. Laboratory examinations revealed increased white blood cell count at 12600 mm^3, high-sensitivity C-reactive protein (hs-CRP) at 8.6 mg/dl and N-terminal pro-brain natriuretic peptide (NT-pro-BNP) levels at 839 pg/ml. Renal function was normal. The fourth day after the procedure, serum markers of inflammation including white blood cell count (5160 mm^3) and hs-CRP (3.6 mg/dl) decreased, while NT-pro-BNP levels (3247 pg/ml) increased. Intravenous loop diuretics significantly improved the symptoms and signs of congestive heart failure in this patient within 24 hours.

DISCUSSION

Congestive heart failure is a new but increasingly recognized complication following AF ablation.[3-5] In a recent study, the prevalence of congestive heart failure after extensive ablation was 2.5%.[5] The exact mechanism underlying this phenomenon is unclear. In its initial description, Weber et al presented 4 patients with pulmonary edema following extensive left atrial ablation. These authors suggested that pulmonary edema was the result of a non-infective systemic inflammatory response syndrome.[3] Singh et al suggested that this syndrome may be related to a combination of volume overload, increased circulating cytokine levels, or reduction in natriuretic peptides with extensive ablation of atrial tissue.[4] The appearance of symptoms 3-4 days post-ablation procedure makes direct volume overload an unlikely cause of this syndrome. Tan et al have lately shown that patients with congestive heart failure display a significant increase in white blood cell counts, serum CRP and BNP levels compared to those without heart failure.[5] These data are indicative of an inflammatory response. It is well-known that patients with AF may suffer acute pulmonary edema after electrical cardioversion, and the exact mechanism is unknown.[6] Subjects undergoing extensive left atrial ablation display significant left atrial dysfunction, despite restoration and maintenance of sinus rhythm.[7] Left atrial stunning may be also implicated in the pathophysiology of this phenomenon. However, volume overload cannot be entirely excluded, especially in presence of diastolic dysfunction as in this case with hypertrophic cardiomyopathy, which has been considered as a predictor of early complications after ablation for AF.[2] A rare occurrence of developing a Tako-tsubo cardiomyopathy after an ablation procedure may also be another cause to be looked out for.[8]

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