Left atrial standstill with paroxysmal atrial fibrillation and atrial flutter after pericardial effusion: A case report

Xuewen Wang[1], Sheng Cao[1], Shaobo Shi[1], Shujuan Zhang[1], He Huang[2], and Qingyan Zhao[1]

[1]Renmin Hospital of Wuhan University
[2]Wuhan University Renmin Hospital

August 28, 2020

Abstract

Atrial standstill is a rare arrhythmia and several cases of atrial standstill have been described in acute myocarditis, hypertrophic cardiomyopathy or dilated cardiomyopathy. However, left atrial standstill with paroxysmal atrial fibrillation and atrial flutter after pericardial effusion has not been reported. Here, we reported a case with left atrial standstill, paroxysmal atrial fibrillation and atrial flutter who successfully received radiofrequency ablation.

Introduction

Atrial standstill (AS) is a rare clinical condition characterized by absence of the electrical and mechanical activity of the atrial myocardium, and the spatial distribution of atrial lesion can be diffuse or partial.[1] Prior cases of AS have been reported in the adult literature with tachyarrhythmias.[2] Several cases of AS have been described in acute myocarditis, hypertrophic cardiomyopathy or dilated cardiomyopathy.[2,3] AS was characterized by the absence of P-waves in the electrocardiogram (ECG) and lack of atrial contraction in the echocardiogram. However, intracardiac electrophysiological examination in AS has not been reported. In the present study, we report a case of left atrial standstill with paroxysmal atrial fibrillation (AF) and atrial flutter (AFL) who successfully received radiofrequency ablation.

Case report

A 56 year old female patient was admitted to our hospital on July 10, 2020 due to palpitation, chest tightness and cough for more than 10 days. There was no obvious abnormality in the laboratory examination, including blood routine, liver and kidney function, pro-BNP, myocardial markers, C-reactive protein, coagulation function, tumor markers, sex hormone, rheumatism and rheumatoid factor. Chest CT indicated double pneumonia, pericardial effusion, bilateral pleural effusion. Echocardiography showed large left atrium with pericardial effusion. Pathological cytology of pericardial effusion showed more mesothelial cells, neutrophils and lymphocytes. The patient was discharged after the effective treatment of diuresis and anti-infection.

After discharge 11 days, the patient suffered from palpitation and chest tightness again. ECG examination showed AF (Figure 1) and the patient was hospitalized again. No obvious abnormalities were found in routine laboratory tests. Transesophageal echocardiography showed that enlargement of both atrium with mitral regurgitation and tricuspid regurgitation (AF associated) and atrial wall thickening.

On August 5, the patient underwent AF and AFL ablation. LASSO mapping showed that there was no potential activity in the left upper pulmonary vein, the right upper and lower pulmonary veins, the left atrium and left atrial appendage, but a small amount of potential in the left lower pulmonary vein (Figure 2). The atrial activation of AFL around the tricuspid annulus in the counterclockwise direction was mapped and part of the right atrium showed no potential activity. Circumferential left pulmonary veins and isthmus of tricuspid valve ablation were performed. After AFL converted to sinus rhythm, electrophysiological examination showed that the ablation line reached bidirectional block and AFL could not be induced (Figure 3).

We found that no pulsation of the ablation electrode during the circumferential left pulmonary veins ablation. So, an echocardiography expert reviewed the previous images and the parameters. The echocardiography indicated pericardial effusion, large left atrium, peak A can be seen in the blood flow spectrum of the mitral valve orifice during diastole on July 10. These results showed that the left atrium had contraction (Figure 4A, B). However, the results of echocardiography showed significant thickening of the wall of left atrial and left atrial appendage on August 3, there was no peak A in the blood flow spectrum of the mitral valve orifice during diastole. These results indicated that the left atrial had no contraction (Figure 4C,D).

Discussion

It has been reported that hypertrophic cardiomyopathy or dilated cardiomyopathy can cause AS.[2,3] Some scholars have proposed the AS is defined by: Atrial electrical activity could not be recorded by routine ECG; The jugular pulse and wave disappeared; The atrial contraction disappeared under X-ray fluoroscopy; QRS wave is the supraventricular pattern; Higher intensity atrial stimulation does not produce atrial excitement.[4] Studies have found that atrial muscle degeneration, necrosis, fibrosis, fat cell accumulation and other pathological changes occurred in patients with AS and the changes were mostly irreversible.[5] Chhabra L et al. reported that interatrial block will affect atrial electrical and mechanical activity, especially the patients with left atrial enlargement. The atrial injury leads to interatrial block, and finally leads to atrial standstill.[6] However, intracardiac electrophysiological examination in AS has not been reported.

In this case, a small amount of potential was observed in the left inferior pulmonary vein, and no electrical activity was measured in other parts of the left atrium, which could indicate AS not caused by interatrial block. AS caused by acute myocarditis has been reported.[7] Atrial nonpotential and AS after pericardial effusion have been rarely reported. In this case, rheumatism, tumor, tuberculosis and myocarditis were excluded according to the examination. Pericardial effusion test suggested that inflammatory cells increased, and pericardial effusion decreased after anti-inflammatory treatment. However, the specific mechanism of AS was unknown. The risk of thrombosis and stroke was significantly increased in patients with AS and stroke has been reported in patients with AS,[8] so anticoagulation therapy should be actively used in patients with AS. In this case, anticoagulation therapy was performed and there may be other types of arrhythmia in the right atrium during the follow-up.

Acknowledgment

This study was funded by the National Natural Science Foundation of China (No.81670303 and No.81970277).

Conflict of interests

The authors declare that there are no conflict of interests.

Reference

1. Fuenmayor A AJ, Rodríguez S YA. Atrial standstill. Int J Cardiol. 2013; 165(3): e47-48.
2. Chawla D, Jahangir A, Cooley R, Sra J, Tajik AJ. Isolated Left Atrial Standstill in Patients with Hypertrophic Cardiomyopathy and Atrial Fibrillation after Restoration of Sinus Rhythm.Am Soc Echocardiogr. 2019;32(10):1369-1372.
3. Duncan E, Schilling RJ, Earley M. Isolated left atrial standstill identified during catheter ablation. Pacing Clin Electrophysiol. 2013;36(4):e120-e124.

4. Gal DB, Wojciak J, Perera J, Tanel RE, Patel AR. Atrial standstill in a pediatric patient with associated caveolin-3 mutation. HeartRhythm Case Rep. 2017;3(11): 513-516.
5. Rosen KM, Rahimtoola SH, Gunnar RM, Lev M. Transient and persistent atrial standstill with His bundle lesions. Electrophysiologic and pathologic correlations. Circulation.1971;44(2):220-236.
6. Chhabra L, Baranchuk A, Bayés de Luna A. Left Atrial Standstill and Interatrial Block. Am Soc Echocardiogr.2020;33(7):910.
7. Prabhu MA, Srinivas Prasad BV, Thajudeen A, Namboodiri N. Persistent Atrial Standstill in Acute Myocarditis. Indian Pediatr. 2016;53(2):162-164.
8. Lee GH, Kim DK, Song YJ, et al. Stroke in a Young Individual with Left Ventricular Noncompaction and Left Atrium Standstill. Korean Circ J. 2015;45(5):432-438.

Figure legends:

Figure 1: The initial electrocardiographic (ECG) shows atrial fibrillation.

Figure 2: LASSO mapping showed there was no potential activity in the left upper pulmonary vein, the left atrium and left atrial appendage (A,B and C). A small amount of potential activity was recorded in the left lower pulmonary vein (D).

Figure 3: Ablation electrode show part of right atrium without potential. Atrial flutter stopped and converted to sinus rhythm during the process of tricuspid isthmus linear ablation.

Figure 4: A,B: Pericardial effusion, peak A can be seen in the blood flow spectrum of the mitral valve orifice during diastole, showing left atrial had contraction on July 10th. C,D: Significant thickening of the wall of left atrial on August 3th, no Peak A was found in the blood flow spectrum of the mitral valve orifice during diastole, indicating left atrial had no contraction.

Figure 1

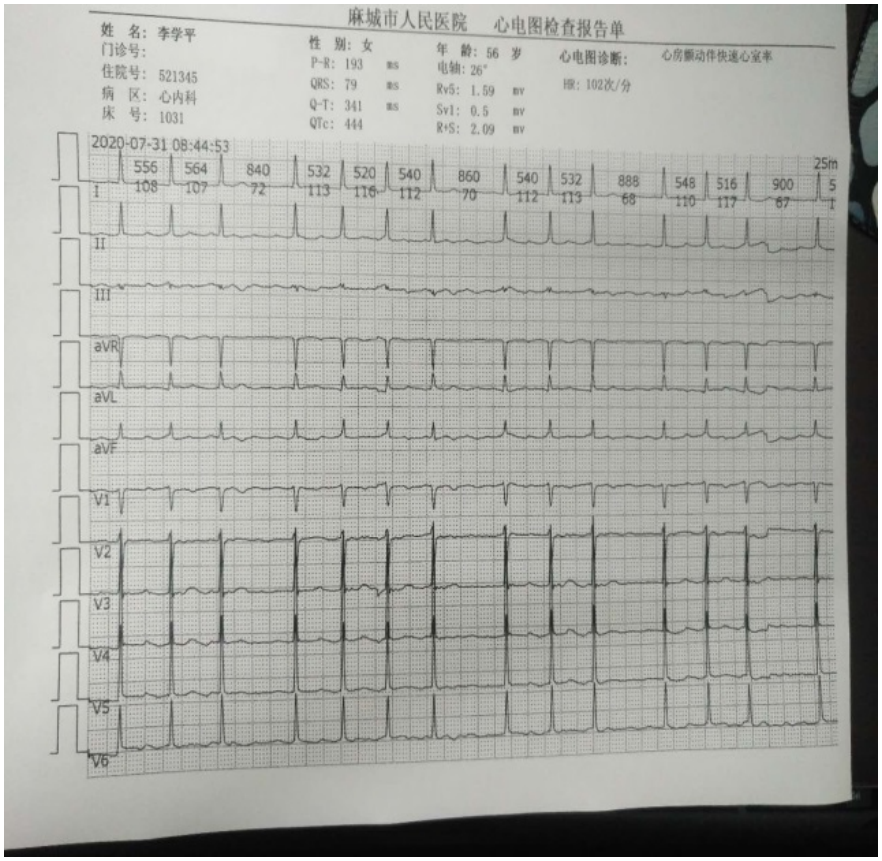
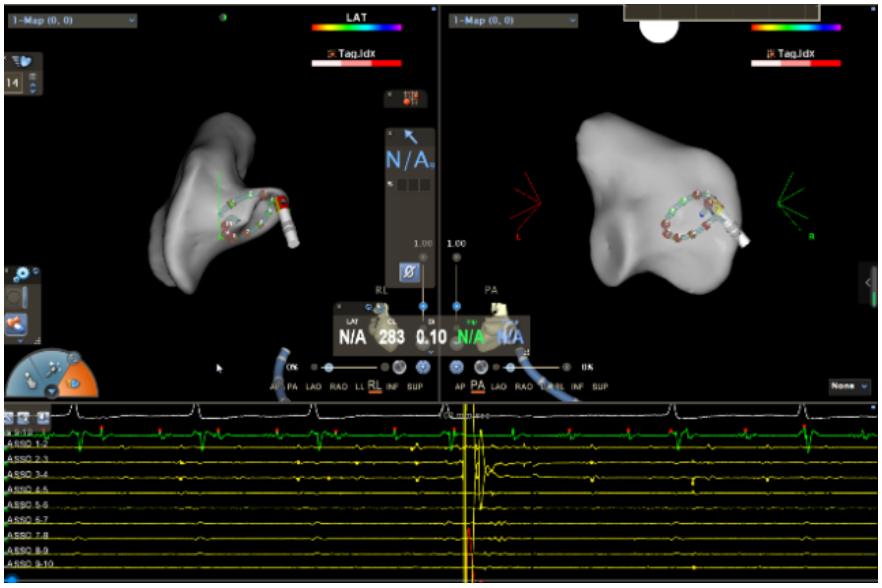
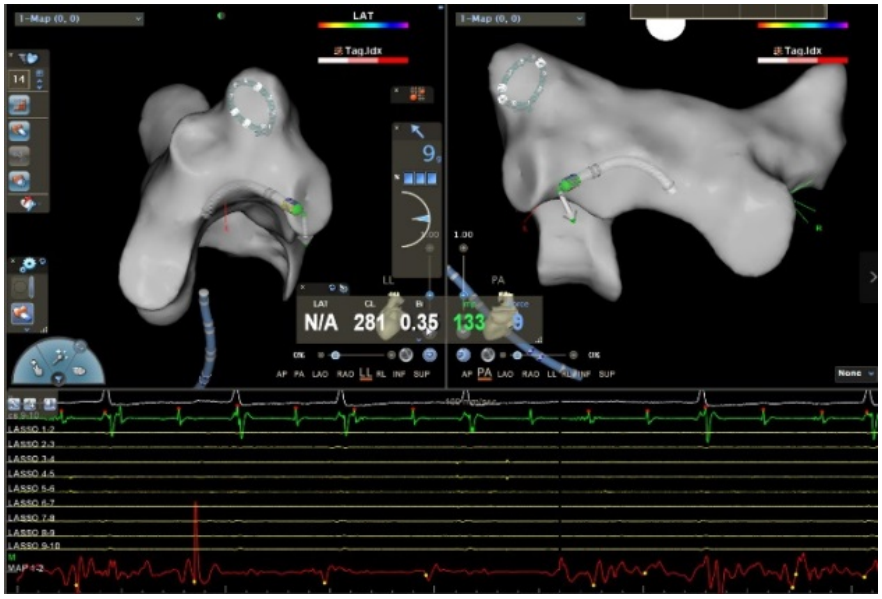
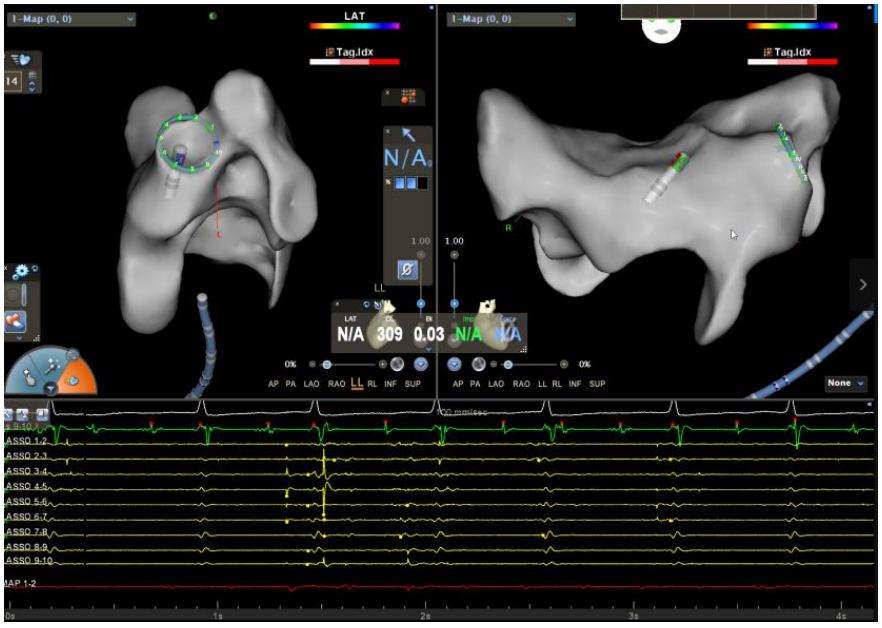


Figure 2







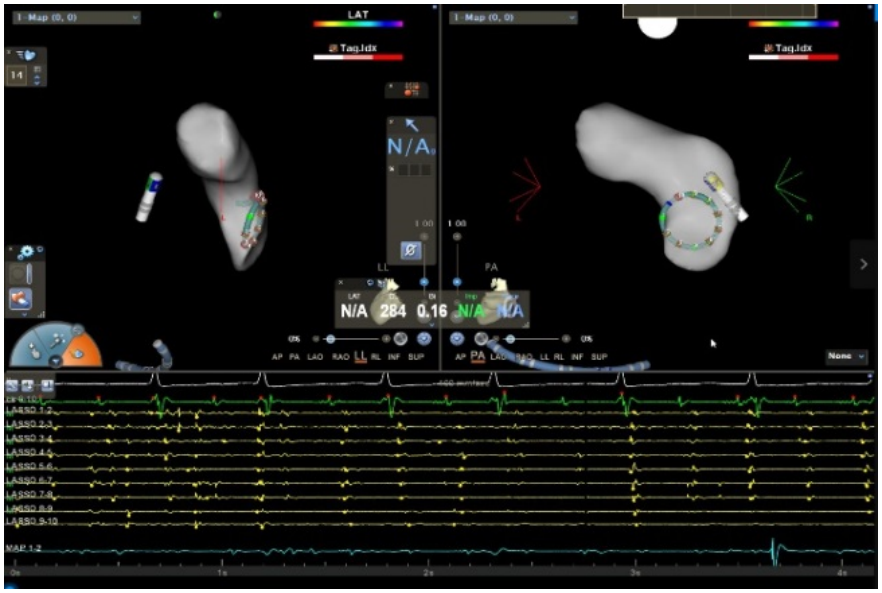
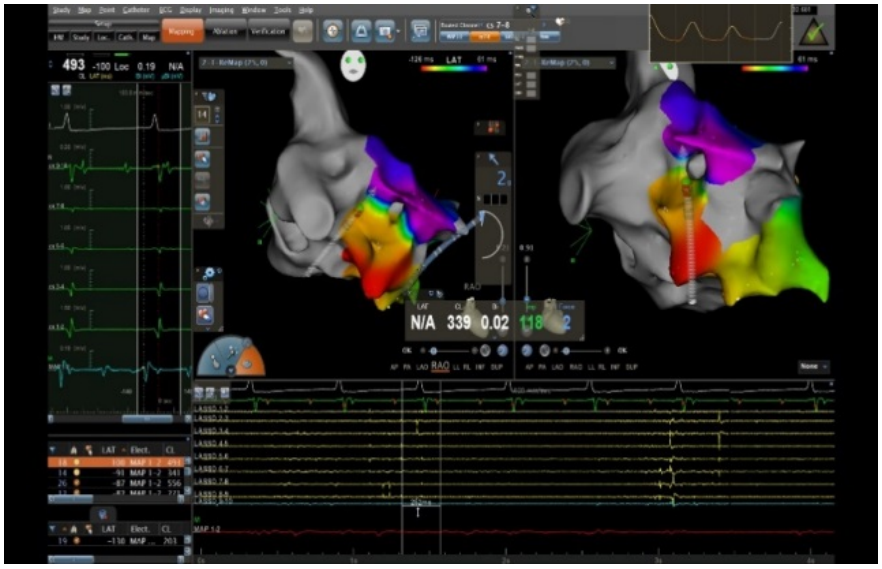
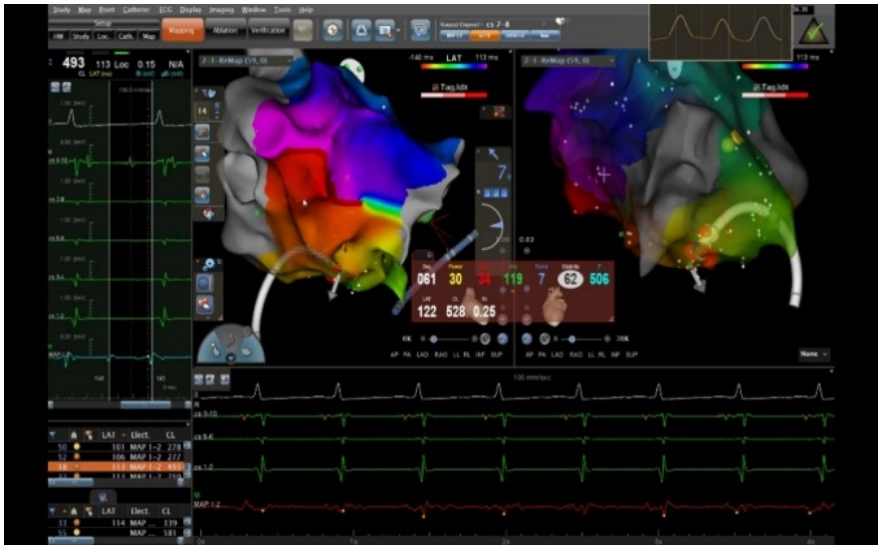


Figure 3





P wave

Figure 4