Electrophysiological characteristics of a left atrial anomalous muscular band in a case with paroxysmal atrial fibrillation

Shunsuke Uetake, MD, Yasushi Miyauchi, MD, FHRs, Meiso Hayashi, MD, Wataru Shimizu, MD

From the Department of Cardiovascular Medicine, Nippon Medical School, Tokyo, Japan.

Introduction

Anomalous fibromuscular bands located in the left ventricle or the right atrium have been demonstrated by echocardiography and are reported to have a relationship with specific types of tachycardias. Although a left atrial (LA) anomalous band is found in approximately 2% of the cases by necropsy, it is rare to be diagnosed clinically. Therefore, its clinical significance, particularly in the development of atrial fibrillation (AF), is unclear. Furthermore, the electrophysiological characteristics have not been revealed. We experienced a case with a prominent anomalous band in the LA in a patient with paroxysmal AF in whom catheter ablation was performed. During the ablation session, we assessed its electrophysiological characteristics.

Case report

A 72-year-old man with symptomatic paroxysmal AF and atrial flutter, which were resistant to antiarrhythmic drugs including flecainide and cibenzoline, was referred to our hospital for catheter ablation. Preoperative transesophageal echocardiography demonstrated an anomalous band in the LA (Figure 1). Three-dimensional cardiac computed tomography revealed that the anomalous band had a diameter of 3 mm and an overall length of 39 mm and was connected between the LA septum and the posterior LA wall near the right pulmonary vein (PV) (Figure 1).

An electrophysiological study was performed under deep sedation. A transseptal puncture was performed guided by intracardiac echocardiography (Ultra ICE, Boston Scientific, Inc, Natick, MA) at the oval foramen just anterior to the site where the anomalous band attached, and 3 long sheaths were advanced into the LA. Electroanatomical mapping (CARTO, Biosense Webster, Inc, Diamond Bar, CA) and subsequent radiofrequency ablation were performed using an open-irrigated catheter (ThermoCool EZ Steer, Biosense Webster, Inc). The activation map of the anomalous band was merged with the computed tomographic images. The contact of the mapping catheter with the anomalous band was confirmed by intracardiac echocardiography advanced in the LA (Figure 1). An activation map during sinus rhythm revealed that electrical activation propagated from both ends of the anomalous band and collided at the middle (Figure 1). For further evaluation, a decapolar electrode catheter with an interelectrode distance of 2.5 mm (Biosense Webster, Inc) was positioned along the anomalous band (Supplemental Figure S). During sinus rhythm, electrical activation propagated from both ends of the anomalous band and the 2 wave fronts collided in the middle (Figure 2). During pacing from the lateral right atrium or from the interatrial septum, activation propagated from the septal to the posterior end, and during pacing from the roof of the LA, it propagated from the posterior to the septal end (Figure 2). During incremental burst pacing from the roof of the LA at rates starting from 100 beats/min, 1:1 conduction from the LA to the anomalous band was observed until the pacing rate increased to 250 beats/min. The conduction time in the anomalous band, measured from the distal pair to the proximal pair of the decapolar catheter, did not show any obvious decremental properties. The estimated conduction velocity of the anomalous band was 0.88–1.0 m/s, which was consistent with normal working atrial muscle (Figure 3).

During AF, the activation frequency of the anomalous band was lower than that of any other locations including the PVs, the coronary sinus, and the right atrium, suggesting that the anomalous band was excited passively during AF. No macroreentrant tachycardias involving the anomalous band as the reentrant circuit were induced. After evaluating the electrophysiology of the anomalous band, circumferential antral PV isolation and LA roof linear ablation were performed. We paid attention not to mechanically damage the anomalous band with the ablation catheter or to entrap it with the ring catheters. The activation sequence of the anomalous band after that ablation exhibited septal to posterior conduction, suggesting that the entrance from the posterior end was blocked by the linear lesion of the LA roof (Figure 2). Again, no macroreentrant atrial tachycardias...
involving the anomalous band as a reentrant circuit were induced by programmed stimulus. At the end of the study, we tried to induce non-PV ectopic beats using isoproterenol and adenosine triphosphate. However, no ectopic activity from the anomalous band was induced.

**Discussion**

LA anomalous bands were initially described in the literature in 1896 as a cord attached to the septal wall beside the fossa ovalis and left auricular wall below the appendage in 2 necropsy cases. More recently, Yamashita et al. reported a series of 1100 cases with necropsy, and an LA anomalous band was found in 22 cases (2%). In 19 cases (1.7%), the anomalous band connected to 2 areas in the LA, one of which was the LA side of the fossa ovalis and the other was the anterior (6 cases), superior (5 cases), posterior (6 cases), and inferior (2 cases) endocardium. The sizes ranged from 1.5 to 4 mm in width, from 0.5 to 2 mm in thickness, and from 4 to 55 mm in length. Histopathological studies showed that the anomalous bands were composed of fibrous and muscular tissue with no Purkinje cells, which is consistent with normal working atrial muscle and had no arrhythmogenic activities.

Caution should be exercised to avoid any mechanical damage to the AB while manipulating the catheters as well as entrapment of ring catheters.

**KEY TEACHING POINTS**

- Left atrial anomalous band (AB) is a rare anomaly that is found in approximately 2% of the cases by necropsy.
- Histopathological studies showed that the AB was composed of fibrous and muscular tissue with no Purkinje cells.
- The present case showed that the AB had electrophysiological properties consistent with normal working atrial muscle and had no arrhythmogenic activities.
- Caution should be exercised to avoid any mechanical damage to the AB while manipulating the catheters as well as entrapment of ring catheters.

Figure 1  A: Transesophageal echocardiogram. The anomalous band, indicated by the arrows, was attached to the posterior rim of the fossa ovalis (arrowheads). B: Intracardiac echocardiogram from the left atrium (LA), showing a cross-section of the anomalous band (AB). Note that the map catheter (Map) was in contact with the AB. C: Left anterior oblique (LAO) cranial view (left panel) and left lateral (LL) endoscopic view (middle panel) of the activation map of the AB during sinus rhythm merged with the 3-dimensional computed tomographic image, showing that activation propagates from both ends of the AB and collides in the middle. The right panel shows the ablation lesion sets. Red and pink tags represent the ablation sites for the circumferential PV isolation and LA roof line, respectively. Cra = cranial; LSPV = left superior pulmonary vein; RA = right atrium; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein; Sept = septal.
Figure 2  Activation sequence of the decapolar catheter located along the anomalous band (AB) in the left atrium (LA) during sinus rhythm before ablation. A: Activation during sinus rhythm before ablation, showing activation propagating from both ends and colliding in the middle. B: Activation during septal pacing, showing activation propagating from the septal end of the AB. C: Activation during pacing from the roof of the LA, showing activation propagating from the posterior LA side of the AB. D: Activation during sinus rhythm after the antral pulmonary vein isolation and roof ablation, showing entrance block from the posterior LA side of the AB. CS = coronary sinus; d = distal; p = proximal; PLA = posterior wall of the left atrium; RA = right atrium.

Figure 3  A: Local electrogram of the decapolar catheter located along the anomalous band during pacing from the roof of the left atrium at a rate of 100 beats/min. The conduction time from the distal pair to the proximal pair was measured. B: Conduction time of the anomalous band at various pacing rates. C: Conduction velocity calculated from the conduction time between the 2 pairs of electrodes. CS = coronary sinus; p = proximal; RA = right atrium; Stim = pacing stimulus artifact.
with working cardiac muscle. The embryological origin of the LA anomalous band is unclear but is thought to be the result of an elongation of abnormal adhesions between the outer wall of the atrium and the part of the atrial septum formed by the endocardial cushions, or a remnant of a part of the right valve of the sinus venosus, which enters into the left atrium through the foramen ovale.

Right atrial anomalous bands, which are the so-called Chiari’s network, are reported to cause supraventricular arrhythmias. The left ventricular anomalous band or false tendon has been reported to cause specific types of ventricular arrhythmias. However, the clinical significance of the LA anomalous band is less clear, probably owing to its rare prevalence and difficulty in finding it by echocardiography. At present, there is only 1 case report of a patient with a diagnosis of an LA anomalous band by echocardiography, where the anomalous band between the septum and the anterior mitral leaflet was the cause of mitral regurgitation. Yamashita et al compared the incidence of supraventricular arrhythmias documented by the 12-lead electrocardiogram recorded within 1 year before the postmortem examination and found that 22 patients with an LA anomalous band had a significantly greater incidence of atrial premature contractions than did those without (41% vs 11%), but the incidence of AF was similar (14% vs 13%). They speculated that stretch of the anomalous band and/or vibration of the band with the blood flow lead to the occurrence of premature atrial complexes through contraction-excitation feedback. In the present case, arrhythmogenesis was found in the PVs, and no ectopic activity was observed or induced in the anomalous band by pharmacological stimulation with isoproterenol or adenosine triphosphate. Furthermore, no macroreentrant tachycardia was induced by programmed stimulation. The anomalous band had electrophysiological properties consistent with intact atrial cardiac muscle, with an estimated conduction velocity between 0.88 and 1.0 m/s. These electrophysiological characteristics suggested that an LA anomalous band may not have caused AF by itself and may have been a bystander of AF. However, caution should be exercised to avoid any mechanical damage to the anomalous band while manipulating the catheters as well as entrapment of ring catheters.

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Appendix
Supplementary data
Supplementary material cited in this article is available online at http://dx.doi.org/10.1016/j.hrcr.2015.01.017.

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