

Case Report

ECG Findings of Acute Myocardial Infarction and Atrioventricular Block During a Transseptal Procedure for Left Atrial Ablation

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We report on a patient with transient atrioventricular block and ST-segment elevation mimicking the ECG of myocardial infarction during transseptal puncture for radiofrequency catheter ablation of atrial fibrillation. Symptoms and ECG findings resolved spontaneously. A neurally-mediated mechanism, activated by the mechanical effects of the transseptal puncture on the interatrial septum and leading to coronary artery spasm, may be considered as a possible explanation of this phenomenon. Coronary artery embolism following the transseptal procedure represents an alternative mechanism. The above mechanisms could also explain the atrioventricular block.

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Radiofrequency ablation (RF) of the pulmonary vein ostia is currently a commonly suggested treatment for patients with symptomatic and drug-refractory atrial fibrillation.¹⁻³ The transseptal (Brockenbrough's) puncture is widely utilised to access the left atrium for this purpose. Known complications of the transseptal procedure include aortic root and atrial perforation, stroke, and transient ischaemic attacks.⁴⁻⁶ The present report describes a case of transient ST-segment elevation with advanced atrioventricular block occurring during the transseptal puncture for RF ablation of atrial flutter/fibrillation. The possible underlying mechanisms are discussed.

Case presentation

A 65-year-old man with a history of drug-refractory paroxysmal atrial fibrillation and typical atrial flutter was referred to our laboratory for RF ablation. Transoesophageal echocardiography was performed prior to

the procedure to rule out the presence of interatrial thrombus. Echocardiographic data regarding left ventricular ejection fraction and left atrial diameter were normal. During atrial flutter, transseptal access was achieved under fluoroscopic guidance, using an 8-French transseptal sheath with a Brockenbrough's needle and applying the standard technique.⁵ Once transseptal access was obtained, 5000 units of heparin were administered intravenously. The transseptal puncture was followed directly by angina pectoris with ST-segment elevation in the inferior leads and lead V₁ (Figure 1). The procedure was immediately interrupted. Two-dimensional echocardiography ruled out acute pericardial effusion. Symptoms and ECG findings resolved spontaneously 30 min later. An interesting finding was that soon after ST-segment elevation, complete atrioventricular block appeared and then the atrial flutter converted spontaneously to sinus rhythm (Figure 2). Coronary angiography excluded significant co-

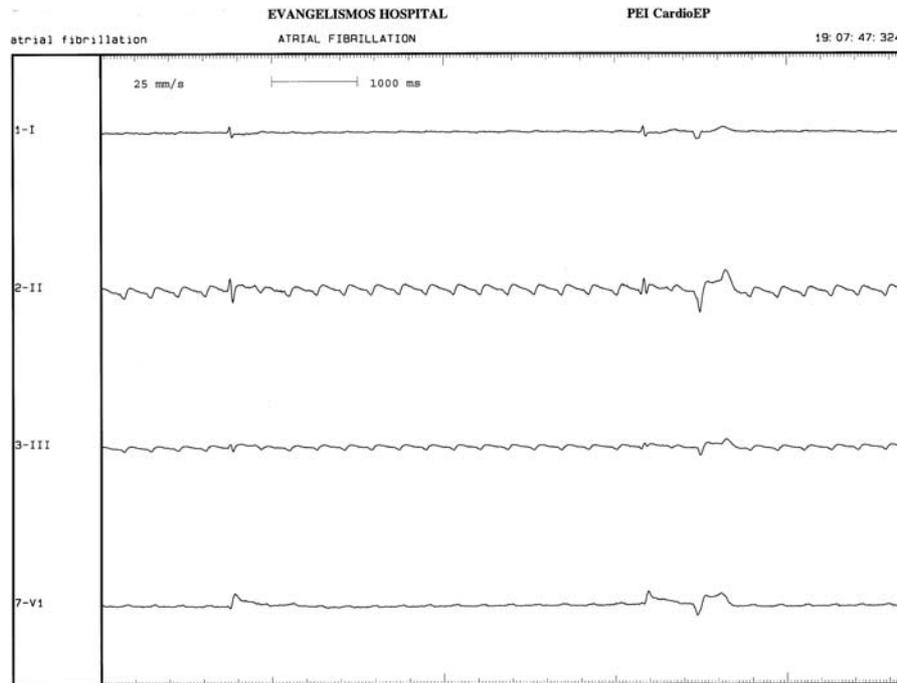


Figure 1. Complete atrioventricular block and ST-segment elevation in leads II, III, and V1 during a transseptal puncture for radiofrequency catheter ablation of atrial fibrillation/atrial flutter.

ronary artery stenosis (right coronary artery dominance). Subsequently, the ablation procedure was continued successfully and the patient was transferred to the intensive care unit for close monitoring. Over a 48-hour period no signs of ST-segment deviations or pericardial effusion were observed.

Discussion

The present report highlights an unexpected and potentially hazardous complication manifested as acute myocardial infarction with complete atrioventricular block during a transseptal procedure for RF ablation of the left atrium.

Recently, a prospective multi-centre trial demonstrated four cases of ST-segment elevation in inferior leads among 43 patients who underwent RF ablation of the left atrium. Neither thrombotic material nor emboli were detected during the elective angiography. An increased autonomic response to the manipulations of the septum and the dorsal and lateral wall of the left atrium leading to coronary vasospasm has been proposed to explain this complication.⁷ In the present case, angina and ECG signs of acute myocardial infarction were observed immediately after the transseptal puncture, before any application of RF energy in the left atrium. Angiography performed with-

out delay excluded coronary artery disease or embolic material, and the ablation procedure was completed successfully.

Coronary artery spasm secondary to an autonomic imbalance appears to be the most likely underlying mechanism of this phenomenon. Manipulations of the long sheath during or shortly after the transseptal puncture may irritate the interatrial septum vagal branches, leading to a hypervagotonic state and thence to coronary artery spasm. A Bezold-Jarisch-like reflex that has been previously described during transseptal puncture favours the above hypothesis.⁸ The atrioventricular block that occurred in the present report may be related to a specific neurally-mediated response during the transseptal procedure. The spontaneous conversion of the atrial flutter to sinus rhythm could be explained as a mechanical modulation of the atrial re-entry. Atrial stretch through the modulation of the electrophysiological properties (i.e. refractory period and conduction velocity) may not only favour the onset of arrhythmias but may also modulate the rate of atrial arrhythmias.⁹⁻¹³ The atrial pressure increases after each ventricular contraction, and then decreases in a cyclic fashion. Thus, during atrial flutter a mechano-electric feedback mechanism operates. Cyclic variations in atrial volume and pressure following ventric-

ular contractions modulate the atrial flutter cycle length on a beat-to-beat basis and account for the spontaneous variability of the arrhythmia.^{14,15} Prolongation of the atrial flutter cycle coincides with an increase in atrial pressure and volume when the variation of atrial flutter cycle length after the ventricular contraction is compared with the variation in atrial pressure on the same time scale. Since stretch is known to slow down the conduction velocity,¹⁶ the prolongation of atrial flutter interval after the ventricular contraction (Figure 2) could be explained by an increase in the conduction time, which increases the revolution time of an anatomical re-entry and facilitates the conversion of atrial flutter to sinus rhythm.

Previous data have shown that the flutter interval fluctuations are independent of autonomic tone.¹⁷ Air or thrombus coronary artery embolism is a different potential cause of this phenomenon. Coronary air embolism occurring as a complication of transseptal RF ablation of left free wall accessory pathways has been described in previous reports.^{18,19} Recently, Bourke et al reported two cases of ECG changes in inferior leads immediately after transseptal sheath manipulation in a series of 100 patients who underwent pulmonary vein RF ablation. Air embolism to

the right coronary artery was suggested as the underlying mechanism.⁴ The risk of thromboembolic complications following pulmonary vein RF ablation has been emphasised before.²⁰ Ren et al, using an intracardiac diagnostic ultrasound catheter, revealed thrombus formation on the sheath in the left atrium in two patients who underwent RF pulmonary vein isolation.²¹ However, meticulous care to prevent air entry into the left atrial sheath, along with continuous irrigation of the sheath with heparinised saline, minimises the risk of air or thrombus embolism. Additionally, left atrial thrombi usually embolise the cerebral circulation and less commonly the coronary arteries.

In conclusion, ST-segment elevation during transseptal puncture for RF ablation of the left atrium appears to be a transient and completely reversible phenomenon, as described by our study and the cases reported in the literature. A neurally-mediated pathway, activated by the mechanical effects of the transseptal puncture on the interatrial vagal network and leading to coronary artery spasm, appears to be a possible explanation of this unexpected complication. However, coronary artery embolism should always be considered in these cases.

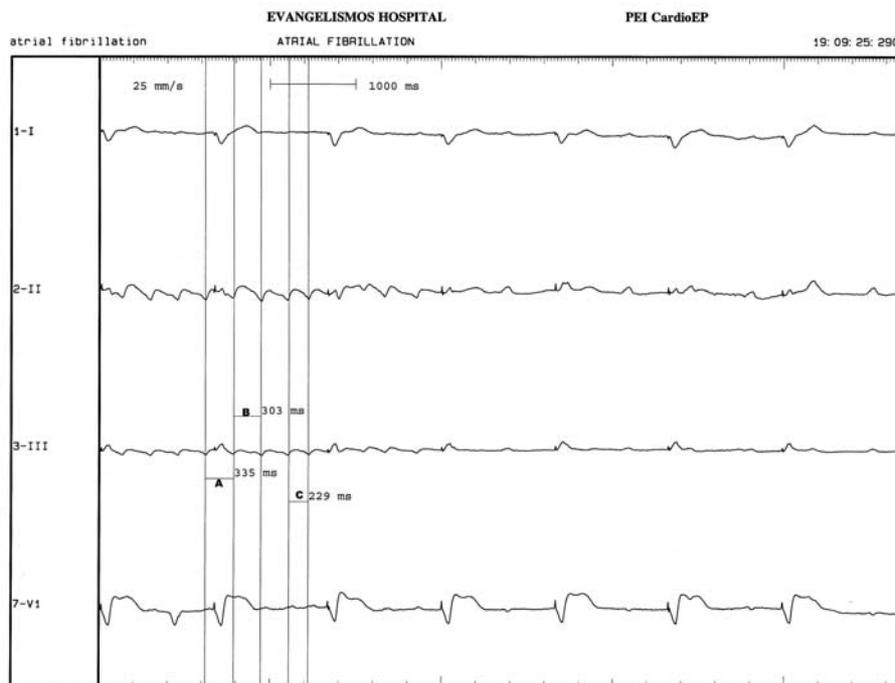


Figure 2. Spontaneous conversion of atrial flutter to sinus rhythm. Prolongation of atrial flutter interval after the ventricular contraction (A and B versus C).

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