

Venous spasm during pacemaker implantation

Pacemaker implantasyonu sırasında venöz spazm

A 75-years-old female presented with dyspnea and syncope. Electrocardiogram revealed atrial fibrillation and complete atrioventricular block. She was therefore listed for pacemaker implantation on the next day. Initial attempt at venous access was left axillary vein. A venogram showed good venous anatomy (Fig. 1a). Several attempts to puncture axillary vein were made. However, there was no good flashback, and the attempts to pass guidewire were unsuccessful. The selection of venous access was changed to left subclavian vein. But, the puncture was still unsuccessful. Venogram was taken once again and revealed significant venous spasm (Fig. 1b). Application of nitroglycerin was taken but without significant effect (Fig. 1c). More than half an hour later, the implantation was changed to right side for the spasm of the vessel had not relieved. A single-chamber pacemaker system was implanted (Fig. 1d).

Venous spasm during pacemaker implantation was rarely reported. The knowledge of it has not been elucidated, including incidence rate, risk factor, effective therapy, and so on. It may be an ignored cause of failure of vein puncture during the implantation of pacemaker.

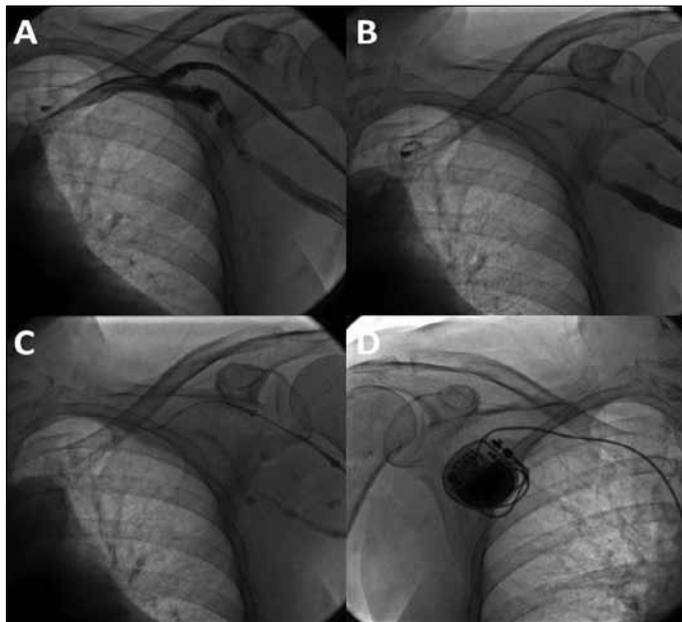


Figure 1. a) Venogram view of good venous anatomy before puncture b) Venogram view of significant venous spasm c) Persistence of vessel spasm despite of nitroglycerin application d) View of implanted pacemaker at right side

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Challenging electrocardiography

Yorumlanması güç bir elektrokardiogram

A 62-year-old female patient was admitted to our hospital after DC cardioversion because of complaints of palpitation, dizziness and presyncope and wide QRS tachycardia on her electrocardiography. Her past medical history revealed hypertension for 20 years. She was on medical treatment with metoprolol and ramipril. Her blood chemistry was in normal range. Transthoracic echocardiography revealed normal left ventricular ejection fraction, left ventricular hypertrophy, enlarged left atrium, moderate mitral regurgitation, moderate tricuspid regurgitation with a pulmonary artery pressure of 50mmHg. Electrocardiography (ECG) showed short PR interval, an early transition zone in the precordial leads, increased notched p wave duration in inferior leads. During follow-up atrial flutter episodes were detected. Wide QRS tachycardia episodes with positive concordance and capture beats were also recorded (Fig. 1). An ECG revealed single and salvos of wide QRS complexes and narrow QRS beats with different morphology and axis (Fig. 2). An electrophysiological study was planned. The rhythm was sinus. Wide QRS complexes with positive concordance similar to clinical ECG with wide QRS were detected during programmed atrial pacing. Tricuspid and mitral annulus mapping was done to find accessory pathway. Accessory pathway was detected in left lateral position. After successful ablation (Fig. 3), normal atrioventricular conduction was detected. ECG revealed correction in precordial derivations and normal transition (Fig. 4).

We presented an electrocardiogram with intermittent minimal and maximal preexcitation in the absence of overt accessory pathway,



Figure 1. ECG showing atrial flutter rhythm, maximal preexcitation and normal conduction (arrow)

ECG - electrocardiogram

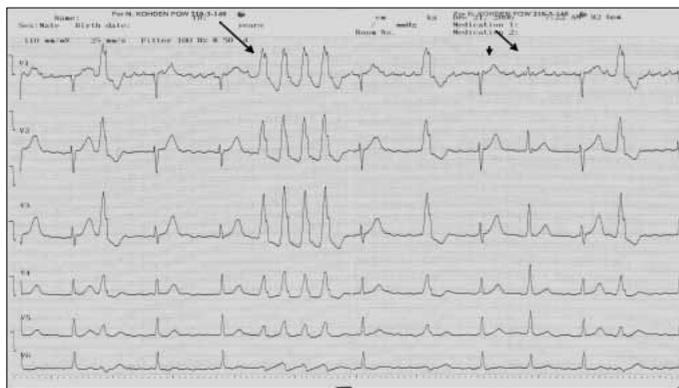


Figure 2. ECG showing atrial flutter rhythm, maximal preexcitation (large arrow), normal conduction (arrow head) and minimal preexcitation (small arrow)

ECG - electrocardiogram