Case Report

Signs of hemodynamically significant myocardial ischemia as a complication of transseptal puncture in catheter ablation of atrial fibrillation

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A B S T R A C T

Introduction: Transseptal puncture is a commonly used method which we can see being applied nowadays predominantly in electrophysiological studies. This interventional method has been very successful at experienced electrophysiological laboratories and has a low risk of potential complications. One of the described complications is emergence of hypotension or bradycardia or transient ST segment elevation on the basis of parasympathetic activation or air embolism into the coronary artery.

Patient, methods: In the case history we present a substantial response in the context of transseptal puncture during electrophysiological examination with pulmonary vein isolation in a patient with persistent atrial fibrillation. This patient subsequently developed bradycardia and there was no response to atropine so he needed temporary cardiac pacing. Furthermore, a stenocardia was observed, as well as ECG elevations of ST segment in II, III and aVF leads and even in the chest leads, and also a severe hypotension with the need for catecholamine support. Echocardiography examination excluded pericardial effusion, and urgent coronary angiography showed normal findings on coronary arteries. Problems gradually subsided and further course did not require pacing and catecholamine support, and subsequently the performance was completed without any other problems.

Discussion and conclusion: Literature describes individual cases with signs of ischemia in the inferior wall after the transseptal puncture. It has always been a temporary complication, and often with a decline of problems after intravenous administration of calcium channel blockers or nitrate. It was therefore suspected that this is a transient coronary artery spasm due to irritation of the autonomic nervous system with vagus nerve activation. Prompt administration of atropine, and if the problems persist also administration of norepinephrine, leads to a decline of problems. It becomes apparent that the need for timely treatment of this complication is necessary. After remission of symptoms and at the
1. Introduction

Transseptal puncture is a commonly used method, which we see today mostly in electrophysiological studies. The description of the transseptal puncture technique already appeared in 1959 [1], when this method was used for measuring hemodynamic parameters in left heart sides. Since then the use of this technique has greatly expanded largely because of the rapid development of electrophysiology in recent years due to more frequently performed procedures in left heart sides (therapy of atrial fibrillation, left-sided accessory pathways and ventricular tachycardia from the left ventricle). Furthermore, this approach is applied in techniques used for left atrial appendage closures. Less frequent indications currently include a mitral valvuloplasty or measurement of hemodynamic parameters in the presence of artificial aortic valve.

At experienced workplaces this method has had a very high success rate performance and a low risk of potential complications, which onset is often associated with possible anatomical varieties of interatrial septum (hypermobility or septum thickening, aneurysmal sinus, individual heart chambers dilation, atypical heart position, prior transseptal puncture) [2]. The described complications include punctures of coronary sinus, aortic root, free right wall or left wall atrium with the risk of pericardial effusion up to tamponade [3], then the emergence of reflex vasovagal hypotension or bradycardia, transient ST segment elevation in leads II, III, and VF with possible stenocardia from temporary coronary arteries spasm because of parasympathetic irritation. Another possible etiology, in our opinion less likely, is an air embolism into the coronary artery, which was already previously described in transseptal puncture at radiofrequency ablation of atrial fibrillation and accessory pathways [4–6]. Incidence of all complications at transseptal puncture is below 1% and the incidence of the above described complication with hypotension and bradycardia is to 0.6% [7].

In our case history we present a substantial response in the context of transseptal puncture during electrophysiological examination with emergence of stenocardia, hypotension, bradycardia and ST segment elevation with the need for temporary pacing and catecholamine support.

2. Case history

A 61-year-old patient was admitted to our department for persistent atrial fibrillation, manifested by impaired heart performance and breathlessness. Pharmacological antiarrhythmic therapy had no effect and repeatedly performed electrical cardioversions were always with a short-term maintenance of sinus rhythm. In addition, relapsing epistaxis appeared during a long-term use of warfarin. Heart echo showed a good left ventricular systolic function and already a slightly enlarged left atrium.

The patient was indicated for catheter ablation in the left atrium for persistent atrial fibrillation.

Before electrophysiological study the patient underwent transesophageal echocardiography, intracardiac thrombi was excluded. The examination did not confirm a patent foramen ovale. In the electrophysiological laboratory a 7F sheath was introduced into the right femoral vein and through the 7F sheath a decapolar catheter was inserted into the coronary sinus. Then using the right femoral vein an uncomplicated transseptal puncture under a skiascopic control and also a checkup by local control sprays of contrast iodine agent and invasive measurement of pressure at the needle tip were performed and after a successful transseptal puncture an Agilis 8.5 F steerable introducer (St. Jude Medical, USA) was introduced into the left atrium. The same method was applied during the second transseptal puncture with the introduction of 8F SL1 sheath (St. Jude Medical, USA).

After the introduction of the second sheath into the left atrium the patient experienced severe chest pressure, chest pain, nausea and hypotension. On the new surface ECG, ST segment elevations appeared in leads II, III, and VF and subsequently also elevations in chest leads V1-V6, then a significant bradycardia and hypotension (50/30 mmHg) emerged. Therefore an ablation catheter was introduced into the right ventricle pacing by frequency of 70/min (Figs. 1–3). During this stimulation, the blood pressure value was 70/50 mmHg. When trying to switch the stimulation off, the patient had atrial fibrillation with significant bradycardia, transiently even asystoles. Elevations at the lower and the front wall persisted. Therefore the stimulation continued and two minutes after the onset of problems, Atropine i. v. was repeatedly administered without any effect. Due to nausea, Torecan i. v. was repeatedly administered.

Cardiac echo was performed, with no evidence of pericardial effusion; moderately significant depression of LV function was found without any clear segmental kinetics defects. Due to the ongoing bradycardia, hypotension and stenocardia we arranged an acute coronary angiography. Since symptomatic hypotension persisted, norepinephrine (2 ml/20 ml NS–4 ml/h) was administered 17 min after the onset of the difficulties and subsequently there was an increase in blood pressure up to 100/60 mmHg and the patient’s own rhythm was restored—atrial fibrillation with ventricular response 80–100/min. Angiography was performed in case of persistent symptoms and persistent ST segment elevation in leads II, III, and VF and V1-V6 which showed smoothwalled coronary arteries with TIMI III flow (Fig. 4a and 4b). Chest pain gradually declined and ST elevations decreased both in the limb and chest leads. After 35 min from the beginning of the
difficulties and when blood pressure values returned to normal, norepinephrine was discontinued (Fig. 5).

In the further course the patient was already without any difficulties and after the introduction of lasso catheter and ablation catheter into the left atrium we performed a complex radiofrequency ablation in the left atrium (pulmonary vein isolation, roof line and linear lines to mitral anulus) with the support of NavX system without any further problems or complications. In the interval of 4 and 12 h after completing the intervention we took cardiospecific markers (CK, CK-MB, troponin-T) with maximum values of CK 7.57 ukat/l, CKMB 0.43 ukat/l and troponin-T (cTnT) Ultras 1.0700 ug/l. These values do not exceed usually observed range, which occur after radiofrequency ablation within the complex interventions in the left atrium [8]. Before the discharge the patient his heart echo showed normal systolic function of the left ventricle, without evidence of pericardial effusion. Thus the patient was discharged to the outpatient care.

Fig. 1 – Initial 12-lead ECG.

Fig. 2 – The 12-lead ECG—ST elevation in leads II, III, aVF.
3. Discussion

In the past, we could read descriptions of individual cases with signs of ischemia in the lower wall after transseptal puncture. Transient ST segment elevations in limb leads II, III, and VF are quoted in the literature in the volume of about 0.6% of the total amount of procedures [9]. It was always about a temporary complication, and therefore it was suspected that this is a transient coronary artery spasm arising from the parasympathetic stimulation. This theory is supported by the results of angiography, which were performed when ST segment elevations were present. During examination of the coronary arteries no significant stenosis or the

![Fig. 3 – The 12-lead ECG—ST elevation in leads II, III, aVF, V1-V6.](image1)

![Fig. 4 – Result of the coronarography at the moment of patient's problems: (a) A coronary angiogram that shows the left main coronary artery (LMCA), the left circumflex artery (LCX) and the left anterior descending artery (LAD) without spasm or slow flow phenomenon. (b) Normal coronary angiogram of the right coronary artery (RCA).](image2)
presence of thrombus were observed; only exceptionally we observed insufficient flow in the right coronary artery without any clearly visible blood vessel spasm [10]. With regard to this finding, hypothesis also considered a possible air embolism into the coronary artery. Most common are ischemic changes above the inferior wall. This would correspond with a higher likelihood of air embolism into the right coronary artery due to the more anterior-positioned right aortic cusp in the supine position it is more likely that the air would be accumulated in this place with the subsequent air embolism into the right coronary artery. Often the problems were resolved after an intravenous administration of calcium channel blockers or nitrate [11], but this medication is problematic both in patients with hypotension and bradycardia, and also in normotensive patients. If the caused condition is air embolism into the coronary artery, then the administration of nitrates and calcium channel blockers can cause worsening of clinical condition and can deepen difficulties. Therefore, if this complication occurs, atropine should be administered, and even if after this the problem still persists, then the cause of this difficulty is more likely to be air embolism into the coronary artery. Subsequent and timely administration of norepinephrine may be the only therapy that leads to a rapid solution of this complication and to the decrease of a patient’s problems. Cases described in the literature achieved complete normalization of the ST segment, stenocardia resolved and the scheduled electrophysiologic examination was completed in most cases [12]. This was also our case, even though the symptoms were extremely significant and were manifested as an acute myocardial infarction with cardiogenic shock and bradycardia. Administration of atropine in our patient did not improve his clinical condition, and an earlier administration of norepinephrine would most likely lead to a more rapid retreat of problems which would have probably also evolved less significantly. It turns out that after timely treatment of this complication and if the problems persist even after excluding other possible complications it is then possible to complete electrophysiologic study after the symptoms have resolved.

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References


