Sinus of Valsalva aneurysm – myocardial infarction perpetrator or silent bystander?

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SOUHRN

Úvod: V některých případech nemusí infarkt myokardu vzniknout na podkladě aterotrombózy, ale může mít mnoho jiných příčin. Jednou z nich je embolizace do koronárního řečiště. Tato kazuistika diskutuje možnost koronárního embolismu z aneurysmatu Valsalvova sinu (SOVA).
Popis případu: Čtyřiapadesátiletá pacientka byla referována pro infarkt myokardu s elevacemi úseku ST (STEMI) spodní stěny. Byla provedena urgentní koronarografie s nálezem uzávěru ramus interventricularis posterior, dle intervenčního kardiologa suspektně embolizačního, řešeného perkutánní koronární intervencí (PCI). Při echokardiografickém vyšetření byla vyslovena suspekce na aneurysma pravého Valsalvova sinu. Nález byl potvrzen CT angiografií srdce. V odstupu byla provedena perikardiální záplata aneurysmatu. Avšak několik týdnů po výkonu došlo k recidivě bolestí na hrudi s konečnou diagnózou postperikardiotomického syndromu, kterou potvrdila magnetická rezonance srdce. Byla zahájena farmakologická terapie s dobrým klinickým efektem.
Diskuse a závěr: V diskutovaném případě nás suspektní embolizační uzávěr a nově zjištěné aneurysma Valsalvova sinu vedou k podezření považovat SOVA za zdroj embolizace. Dostupná literatura se zmiňuje o čtyřech případech systémové embolizace ze SOVA, ale o žádné do koronárního řečiště. Pravděpodobnost SOVA jako zdroje embolu je zvýšena umístěním aneurysmatu těsně pod odstupem pravé koronární tepny i přesto, že nedisponujeme průkazem trombu v aneurysmatu. Vzhledem k tomu, že neexistují oficiální guidelines ani stratifikační kritéria posuzující trombogenní potenciál SOVA, zůstává zde mnoho prostoru k diskusi a případně dalšímu výzkumu. Tento případ je tedy prvním publikovaným případem suspektní embolizace z aneurysmatu Valsalvova sinu do koronárního řečiště.

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ABSTRACT

Background: In some cases myocardial infarction is not associated with atherothrombotic coronary artery disease and can be caused by many different mechanisms. One of these situations is a coronary artery embolism. This case report discusses the possibility of coronary embolism from newly diagnosed sinus of Valsalva aneurysm (SOVA), which is a rare clinical abnormality that can be clinically silent or symptomatic in varied ways.
Case presentation: A 54-year-old woman presented with ST-segment elevations myocardial infarction of left ventricle inferior wall. We performed emergent coronary angiography where occlusion of the posterior descending artery was established. This finding was according to the interventional cardiologist's suspicion of embolic etiology. Primary percutaneous coronary intervention was performed. Transthoracic echocardiography suspected of an aneurysm of the right sinus of Valsalva presence. We added coronary computed tomography angiography with confirmation of the SOVA with no thrombi inside. Cardiac surgery with a pericardial patch was performed to solve the SOVA. Unfortunately later postpericardiotomy syndrome appeared which was confirmed by cardiac magnetic resonance. We initiated the therapy of pericarditis with a good effect on the patient's clinical state.
Discussion: In this case angiographic suspicion for coronary embolism in association with newly diagnosed sinus of Valsalva aneurysm led us to consider SOVA as the origin of the embolus. There have been four cases of systemic embolism from SOVA in so far published data mentioned, but no case of embolism from SOVA to coronary circulation has been described. Probability of the embolus origin from SOVA in this case is increased by localization of SOVA beneath the right coronary artery ostium even if we have no evidence of thrombi inside of SOVA. Because there are no official guidelines of SOVA management and there is no stratification scheme of potential SOVA thrombogenicity, there remains a large space for discussion. SOVA thrombogenicity criteria could be a subject for future research. This is the first published case of presumed coronary embolism from SOVA.

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Introduction

The fourth universal definition of myocardial infarction (MI) divides MI to five different types. Type 1 MI develops on the basis of atherothrombotic coronary artery disease, usually in connection with unstable atherosclerotic plaque disruption. In type 2 MI myocardial ischemia is caused by e.g. anemia, coronary spasm, spontaneous coronary artery dissection, hypotension or embolism to coronary circulation.[1] There are several major potential origins of thrombus formation – left atrial appendage, valve prosthesis, left ventricular aneurysm, paradox embolism in presence of the patent foramen ovale.[2] Except these, some unconventional sources of thrombus formation exist – for example sinus of Valsalva aneurysm (SOVA). In literature four cases of unruptured SOVA have been reported as a presumed source of systemic embolism (in all cases to the central nervous system).[3–6]

SOVA is a rare abnormality (incidence 0.1–3.5% of all congenital heart defects) and can be inherited or acquired.[7] In congenital defects histologically we can detect a weakness of the juncture between aortic media and annulus fibrosus most often.[8] Acquired aneurysms can develop in relation to some types of connective tissue disorders as Marfan's syndrome, Ehlers–Danlos syndrome, or furthermore may be caused by syphilis, tuberculosis, cystic medial necrosis and atherosclerosis.[9] A part of SOVA cases is clinically silent and diagnosed incidentally, on the other hand some of them manifest themselves clinically. Possible symptoms of SOVA are varied – ruptured aneurysm toward cardiac chambers can lead to aorto-cardiac shunting, and cardiac failure, in extreme cases they can result in sudden cardiac death,[10,11] further unruptured SOVA can cause coronary artery compression and subsequent myocardial ischemia.[12] Another possibility is thrombus formation in unruptured aneurysm and its embolism to the systemic circulation.[3–6]

Case presentation

A 54-year-old female without any past medical history activated emergency service due to rapid onset of intense dull retrosternal chest pain 7/10 in severity. After its arrival a 12-channel electrocardiogram (ECG) was performed and transmitted to the coronary unit. ST-segment elevation myocardial infarction (STEMI) of the left ventricle (LV) inferior wall was diagnosed. Immediately acetylsalicylic acid 250 mg i.v., unfractionated heparin 7.500 I.U. i.v., 180 mg loading dose of ticagrelor p.o. and 50 μg of fentanyl i.v. were administered by the emergency service. The patient was immediately transferred to the cardiocenter by a helicopter.

On arrival chest pain lessened to 2/10 in severity, the patient was hemodynamically stable, blood pressure 120/80 mmHg, normal blood oxygen saturation, on ECG monitor sinus rhythm 50 bpm was present. Emergency coronary angiography by radial access was performed (see **Fig. 1**). During examination a smooth left coronary artery was described. Right coronary artery was smooth as well, however, its branch – the posterior descending artery (PDA) was occluded. Percutaneous coronary intervention (PCI) was indicated – first thromboaspiration

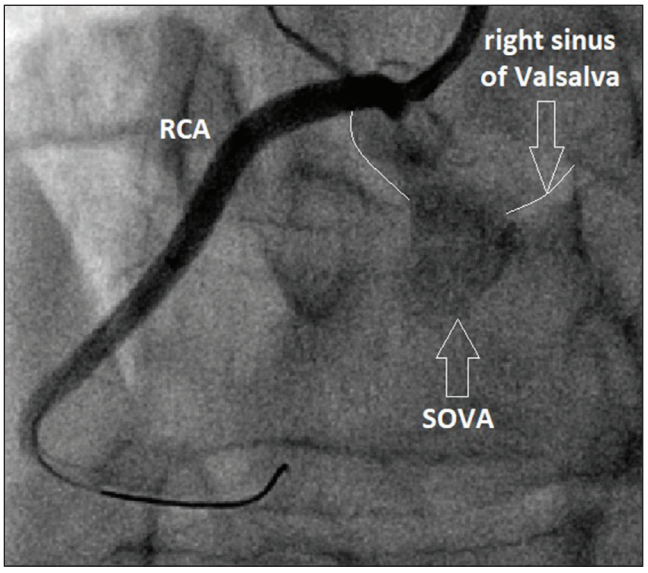

Fig. 1 – Coronary angiography. RCA – right coronary artery; SOVA – sinus of Valsalva aneurysm.

performed, intracoronary bolus of IIb/IIIa inhibitor eptifibatide was administered and then two sirolimus-eluting coronary stents were implanted. According to the interventional cardiologist, the features of the coronary obstruction impress as coronary embolism.

Patient was admitted to the coronary unit. Blood samples were collected. As expected – troponin T (hs-cTnT) dynamically increased from 10 ng/l to 1311 ng/l (upper normal limit 14 ng/l). High-intensity statin therapy was initiated and dual antiplatelet therapy with acetylsalicylic acid 100 milligrams o.d. and ticagrelor 90 milligrams b.i.d. was started.

Transthoracic echocardiography (TTE) was performed. Hypokinesis of the LV inferior wall with normal global ejection fraction was described. Surprisingly in an apical 5-chamber (A5C) view and parasternal short axis (PSAX) view at the aortic level, atypic formation by the right sinus of Valsalva was seen (see **Fig. 2**).

Suspicion of SOVA was established. Coronary computed tomography angiography (CCTA) was performed and diagnosis was thus confirmed. CCTA described saccular SOVA sized 15 × 12 millimeters with 8 millimeter ostial diameter, but with no thrombi inside (see **Fig. 3**).

Due to the suspicion of embolic etiology and newly diagnosed potential embolic source a triple antithrombotic therapy was initiated. Rivaroxaban 20 mg o.d. was added to the therapy and ticagrelor switched to clopidogrel 75 mg o.d. The patient was persistently hemodynamically stable. During 5-day continuous ECG monitoring no arrhythmias were observed and sinus rhythm was present all the time. Patient was referred to a cooperating cardiac surgery center and an elective ambulatory control was arranged. Patient was discharged from hospital in a good clinical status.

Then cardiac surgeons indicated surgical repair for suspected thrombogenicity of SOVA and risk of its rupture. Five months after MI a surgical pericardial patch of the aneurysm was performed. The procedure was successful with no serious complications. Anticoagulation therapy was discontinued and dual antiplatelet therapy was kept in medication.

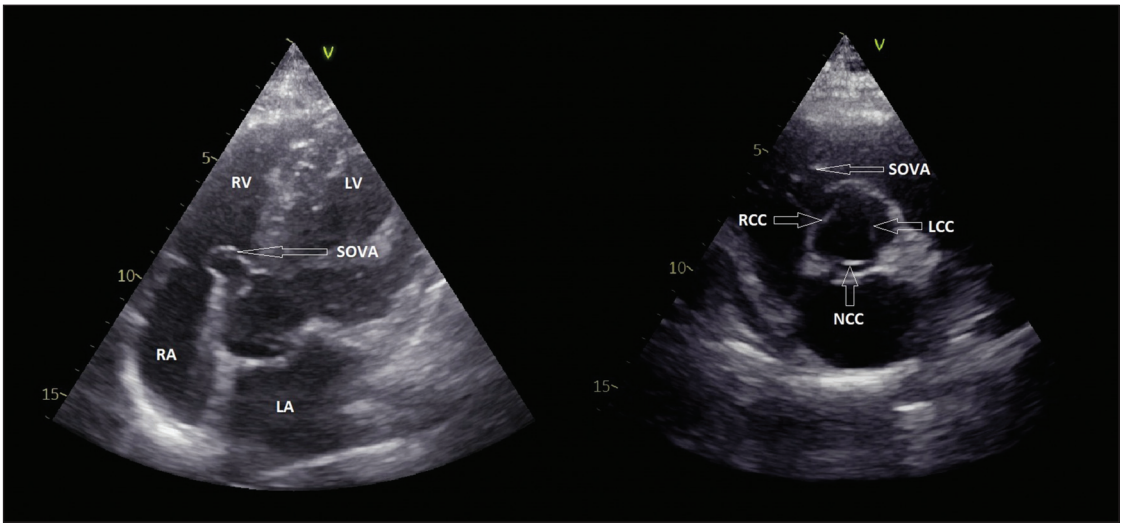

Fig. 2 – Transthoracic echocardiography (TTE). Left side: apical-5-chamber plane (A5C). Right side: parasternal short axis plane (PSAX). LA – left atrium; LCC – left coronary cusp; LV – left ventricle; NCC – noncoronary cusp; RA – right atrium; RCC – right coronary cusp; RV – right ventricle; SOVA – sinus of Valsalva aneurysm.

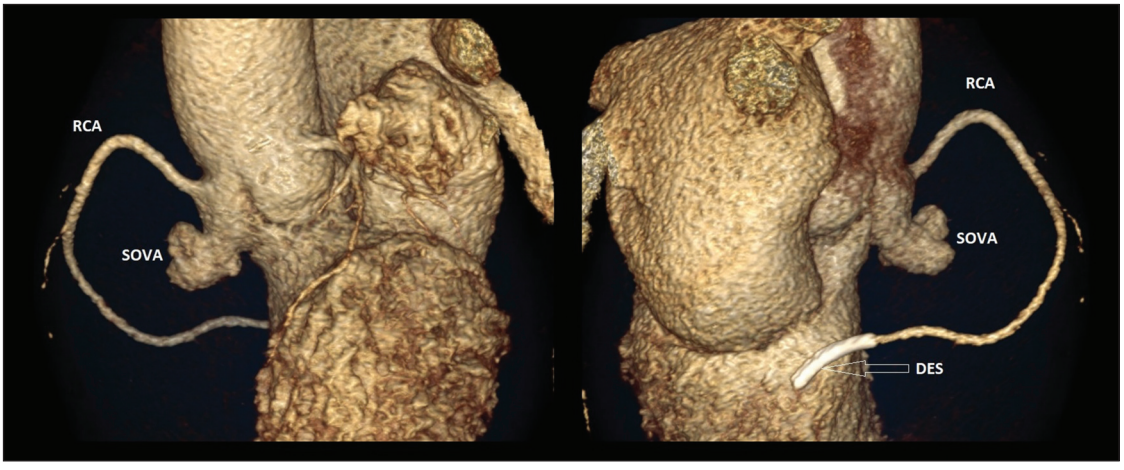

Fig. 3 – Coronary computed tomography angiography (CCTA). Left side: anterior view. Right side: right posterior view. DES – drug-eluting stent; RCA – right coronary artery; SOVA – sinus of Valsalva aneurysm.

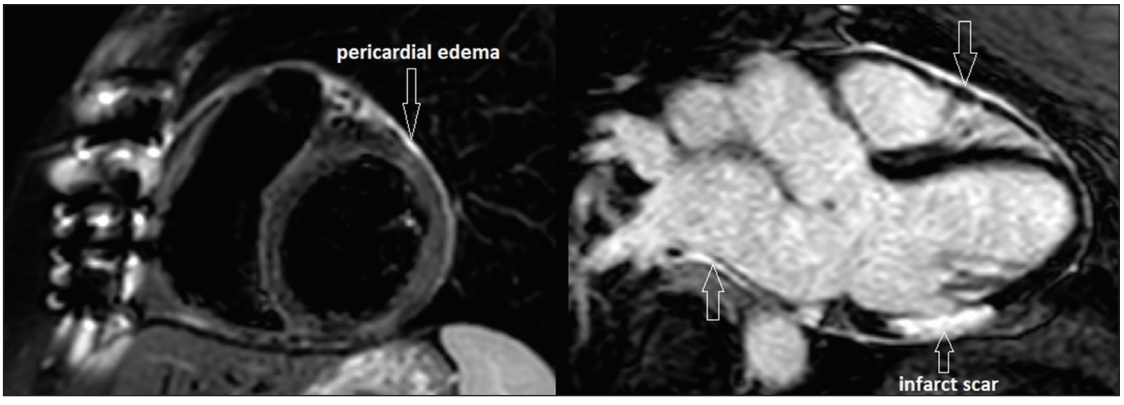

Fig. 4 – Cardiac magnetic resonance (CMR). Left side: T2-weighted imaging with depicted pericardial edema. Right side: late gadolinium enhancement (LGE) of inferior wall transmural infarct scar. Other 2 arrows show pericardial enhancement.

A few days after the discharge from cardiac surgery chest pain appeared. Patient was examined at the emergency department of the regional hospital, ECG showed pathologic Q-wave with non-diagnostic horizontal ST-segment elevations in inferior leads, thus reinfarction was suspected. The patient was immediately transferred

to our cardiac center. A second urgent coronary angiography was performed with smooth coronary arteries and optimal effect after previous PCI. As a part of the examination ventriculography was performed and LV inferior wall dyskinesis described. Serial troponin T (hs-cTnT) measurement was slightly abnormal with no dynamic in the context of recent cardiac surgery. During physical examination no pericardial friction rub was presented. TTE showed no pericardial effusion. Post-thoracotomy chest pain was suspected, analgesics were added and the patient was discharged home.

In a few weeks chest pain recurred. This time TTE showed pericardial effusion. Pericarditis diagnostic criteria were met (chest pain, pericardial effusion) and postpericardiotomy syndrome was suspected. Ibuprofen 400 mg t.i.d. was included in medication with good clinical effect. Cardiac magnetic resonance (CMR) was performed and showed signs of pericarditis, and further the left ventricle inferior wall transmural infarct scar was confirmed by late gadolinium enhancement (LGE) (see **Fig. 4**).

Discussion

The aim of the authors was to present the first case of SOVA suspect from coronary embolism. A pericardial patch of SOVA was performed, unfortunately later postpericardiotomy syndrome occurred.

In some cases a clinical diagnosis and its etiology is clear. But sometimes there can be hidden underlying conditions. In our case we had a suspicion of coronary embolism from coronary angiography, a known potential source of embolism and no other suspicious source of embolism in patient without a history of palpitations or atrial fibrillation. These three facts give us an argument to consider the sinus of Valsalva as symptomatic. Localization of SOVA below the right coronary artery ostium increases the probability of thrombus origin in SOVA. Nevertheless the lack of the proof of thrombus formation inside of the aneurysm makes this case not entirely clear and leaves it open for discussion.

There are no specific guidelines for SOVA management and treatment.[13] Surgery repair is a preferred method, but in literature several transcatheter closure procedures have been performed successfully.[14] Timing of the intervention depends on its symptomatology. In our case a surgery repair was performed with no in-hospital complications. Early after discharge from cardiothoracic surgery postpericardiotomy syndrome occurred. Ibuprofen has been started with a good clinical effect.

A cornerstone of the diagnostics was established by a carefully executed TTE. For diagnosis confirmation and more precise imaging of the anatomical relationships to surrounding structures we successfully used CCTA. Later in a differential diagnostics of the postoperative chest pain, CMR gave us a clear answer.

Conclusion

This is the first published case of presumed embolization from SOVA to coronary circulation resulting in STEMI. A multimodal cardiac imaging was used during diagnostics. Surgical treatment of SOVA was performed. Unfortunately later the postpericardiotomy syndrome occurred, but it was recognized properly and treated successfully.

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Conflict of interest
None declared.

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Informed consent
The authors confirm that written consent for publication of this case report has been obtained from the patient in line with COPE guidance.

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