DOI: 10.15386/cjmed-510

PANNUS-RELATED PROSTHETIC VALVE DYSFUNCTION. CASE REPORT

MARIA-SÎNZIANA MOLDOVAN[1], DANIELA BEDELEANU[1,2], EMESE KOVACS[1], LORENA CIUMĂRNEAN[2,3], ADRIAN MOLNAR[2,4]

[1]Cardiology Department, Niculae Stăncioiu Heart Institute, Cluj-Napoca, Romania
[2]Iuliu Hatieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania
[3]Internal Medicine Department, CF University Hospital, Cluj-Napoca, Romania
[4]Clinic of Cardiovascular Surgery, Niculae Stăncioiu Heart Institute, Cluj-Napoca, Romania

Abstract

Pannus-related prosthetic valve dysfunction, a complication of mechanical prosthetic valve replacement, is rare, with a slowly progressive evolution, but it can be acute, severe, requiring surgical reintervention. We present the case of a patient with a mechanical single disc aortic prosthesis, with moderate prosthesis-patient mismatch, minor pannus found on previous ultrasound examinations, who presented to our service with angina pain with a duration of 1 hour, subsequently interpreted as non-ST segment elevation myocardial infarction (NSTEMI) syndrome. Coronarography showed normal epicardial coronary arteries, an ample movement of the prosthetic disc, without evidence of coronary thromboembolism, and Gated Single-Photon Emission Computerized Tomography (SPECT) with Technetium (Tc)-99m detected no perfusion defects. Transthoracic echocardiography (TTE) evidenced a dysfunctional prosthesis due to a subvalvular mass; transesophageal echocardiography (TOE) showed the interference of this mass, with a pannus appearance, with the closure of the prosthetic disc. Under conditions of repeated angina episodes, under anticoagulant treatment, surgery was performed, with the intraoperative confirmation of pannus and its removal. Postoperative evolution was favorable. This case reflects the diagnostic and therapeutic management problems of pannus-related prosthetic valve dysfunction.

Keywords: prosthetic valve dysfunction, acute coronary syndrome, pannus, echocardiography, surgery

Introduction

Over the past decades, improvements have been made in the design and hemodynamics of mechanical valve prostheses in order to reduce valve dysfunction and increase durability [1]. However, prosthetic stenosis due to thrombus or pannus is still one of the most frequent complications and it can be severe, evolving into cardiogenic shock [2-7]. Pannus is a non-immune inflammatory reaction of the body to the valve prosthesis, a proliferation of fibroelastic tissue and collagen, with a starting point in the suture area and subacute or chronic centripetal evolution [8]. It usually proliferates on the ventricular side of aortic prostheses and is associated with certain risk factors such as operative technique, characteristics of the prosthesis, size of valves in patients with a small valve ring, young age, female sex, pregnancy, low cardiac output, turbulent flow, infection, inadequate anticoagulation [1,8,9]. The differential diagnosis of pannus-related prosthetic valve dysfunction with other causes of prosthetic valve dysfunction, mainly thrombotic involvement, is frequently difficult but essential, because therapeutic approaches are different [10,11,12].

Manuscript received: 09.07.2015
Received in revised form: 28.09.2015
Accepted: 29.09.2015
Address for correspondence: moldovansinzi@gmail.com

Case report

A 54-year-old patient from an urban area was brought by the ambulance to the service of cardiology for typical angina pain with onset about an hour before, after effort, without remission following nitroglycerin administration, associated with profuse sweating, nausea and vomiting.

We mention that the patient was known to the service of cardiology, as he had had a mechanical aortic prosthesis in place for 14 years, for aortic stenosis of rheumatic cause on a bicuspid valve, and ascending aortic aneurysm. The patient was last evaluated 3 years before, under the conditions of a similar angina episode, interpreted as non-ST segment elevation myocardial infarction (NSTEMI) syndrome, investigated by coronarography, with angiographically normal coronary arteries. At that time, echocardiography evidenced increased transprosthetic gradients: maximum pressure gradient(maxG)=88 mmHg, mean pressure gradient(meanG)=50 mmHg(normofunctional prosthesis range:<20 mmHg), maximum velocity(vmax)=4 m/sec(normofunctional prosthesis range: <3 m/sec), a moderate mismatch-indexed aortic valve orifice area=0.82 cm2/m2(moderate mismatch range: 0.65-0.85 cm2/m2)[13], and a small size mass with a pannus appearance on the ventricular side of the prosthesis, which did not interfere with the disc movement. The current episode occurred in the context of repeated chest pain episodes over the past month, which were of lower intensity compared to the current episode, with a variable duration, during effort and rest, with spontaneous remission, accompanied by a decreased effort tolerance, the patient being asymptomatic until one month before. Daily medication at home consisted of oral anticoagulant (international normalized ratio (INR) values unchecked for the past 2 months), aspirin, converting enzyme inhibitor.

Objectively, the patient at admission was afebrile, dyspneic, with angina, blood pressure of 90/50 mmHg, ventricular rate of 70/min, audible prosthetic murmur, no pathological pulmonary changes, diffuse subendocardial injury on electrocardiogram(ECG) (Figure 1), emergency echocardiography: non-dilated left ventricle, with preserved ejection fraction, without significant regional kinetic disorders, dilated ascending aorta, without signs of dissection within 2 cm, increased transprosthetic gradients, biologically: unreacted myocardial enzymes in the first set(CK=142 UI/L, normal range: 10-190 UI/L; CK-MB=18 UI/L, normal range: 0-25 UI/L), normal aspartate aminotransferase(ASAT)(17 UI/L, normal range: 5-40 UI/L), alanin aminotransferase(ALAT)(17 UI/L, normal range: 5-41 UI/L), leukocytosis (12.42*103/μL, normal range: 4.0-9.0*103/μL), hyperglycemia (215 mg/dl, normal range: 74-106 mg/dl), INR value of 2.04 (therapeutic range: 2-3).

Coronarography evidenced a normal appearance of epicardial coronary arteries, similarly to previous coronarography, and an ample movement of the prosthetic disc. Aortic dissection was excluded by CT Angiography (CTA), infectious endocarditis and myocarditis were excluded clinically, biologically, echocardiographically. Gated Single-Photon Emission Computerized Tomography (*SPECT*) with Technetium (Tc)-99m evidenced hypokinesis of the inferior wall and of the interventricular septum, without perfusion defects or non-viable areas.

With anticoagulant, anti-aggregant, calcium blocker, antiarrhythmic treatment during hospitalization, the patient continued to present frequent pain episodes of very high intensity, which were relieved by major analgesics or sometimes remitted spontaneously, accompanied by reversible ECG changes (Figures 2 and 3).

Myocardial enzymes were reacted (peak values: CK-MB=318.9 UI/L; CK=2755 UI/L; Troponine I=2.59 ng/ml(normal range: ≤0.014 ng/ml), AST=243 UI/L, ALT=47 UI/L, subsequently on a continuous decrease. Leukocytes gradually normalized, the other tests being within the limits of reference values.

Repeated transthoracic echocardiography (TTE) was performed, which evidenced increased transprosthetic gradients consistent with severe aortic stenosis (maxG=70-92.7 mmHg, meanG=38-55.5 mmHg, vmax=4.2-4.9 m/sec, aortic regurgitation(AR) grade II due to intraprosthetic leak), moderate prosthesis-patient mismatch (0.67-0.8 cm2/m2), intermittent kinetic disorders in the inferior territory, and an echodense mass attached to the ventricular side of the prosthesis. Subsequently, transesophageal echocardiography (TOE) was performed, which allowed to visualize the interference of the mass with the closure of the prosthetic disc (Figure 4).

The patient was transferred to the service of cardiovascular surgery, where the stenosing subvalvular mass with a macroscopic pannus appearance, subsequently confirmed by microscopy (Figure 5), was surgically removed in extracorporeal circulation.

Postoperative evolution (at 2 months) was favorable, without the recurrence of pain complaints and the restoration of effort tolerance. TTE showed an improvement of transprosthetic gradients: maxG=55 mmHg, meanG=28 mmHg, vmax=3.7 m/sec, with the persistence of AR grade II, hemodynamic insignificant. No regional kinetic disorders were evidenced.

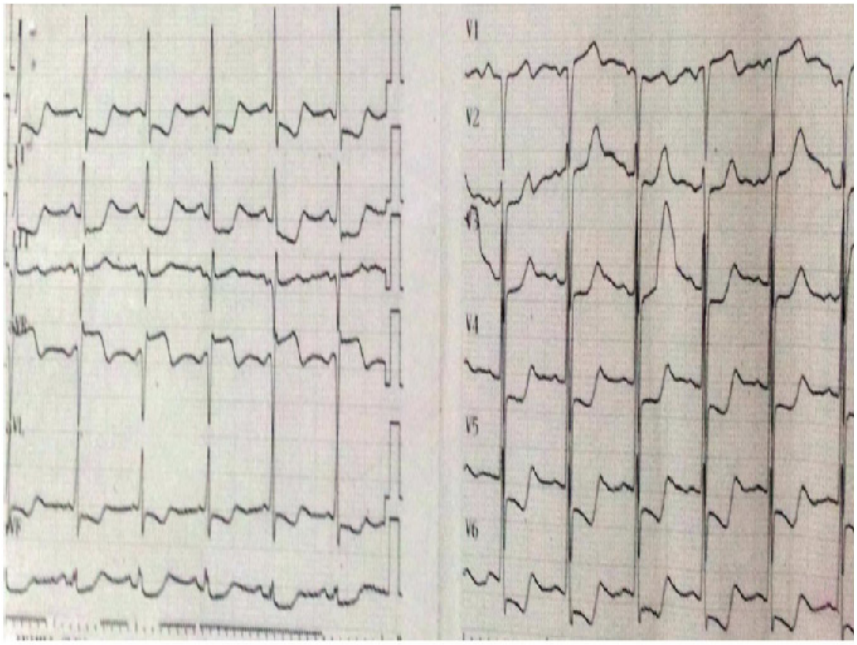

Figure 1. ECG at presentation, during chest pain, showing sinus rhythm with ST depression in leads V3-V6, DI, DII, aVF, AVL and ST elevation in aVR.

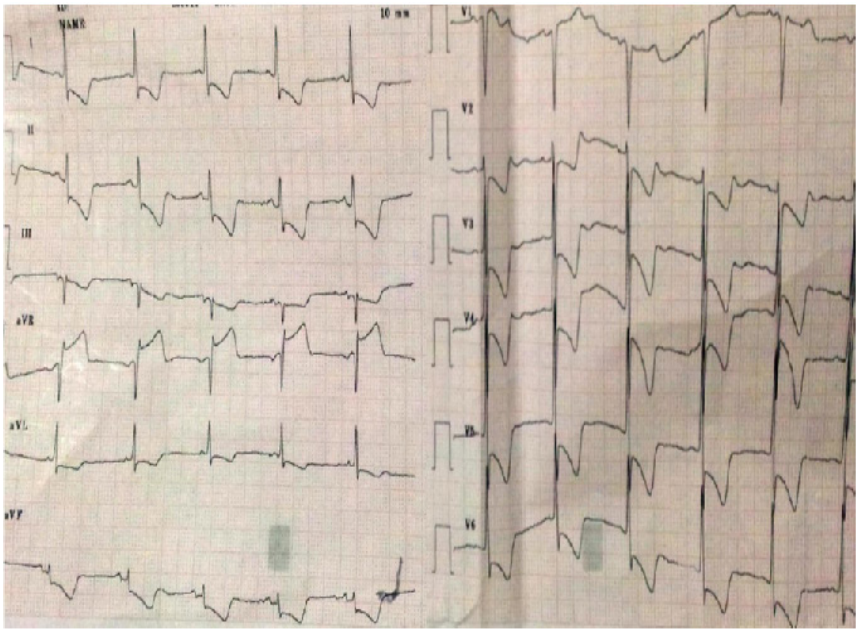

Figure 2. ECG during chest pain, showing remarkable ST changes-ST depression and T wave inversion, including leads V2 and DIII.

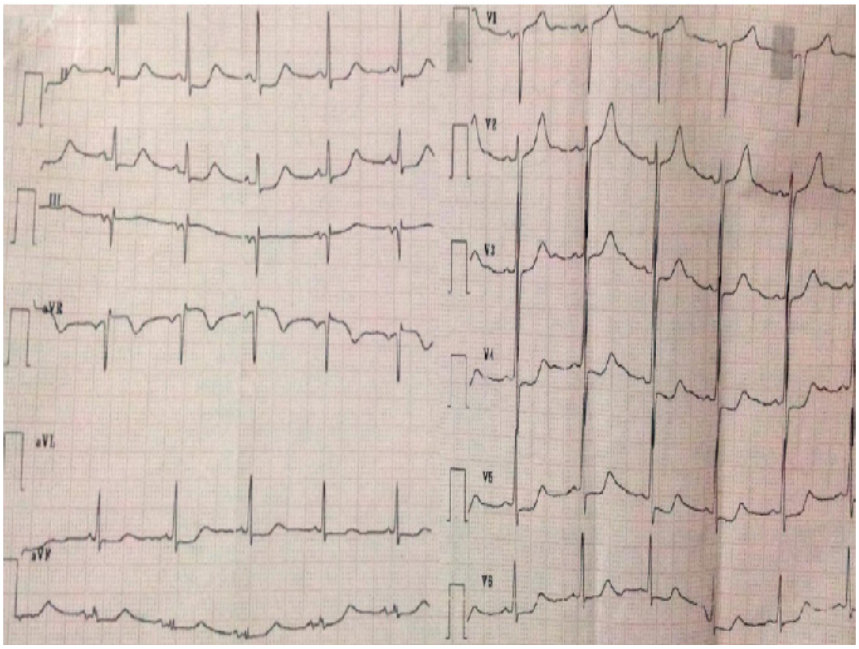

Figure 3. ECG after chest pain remission.

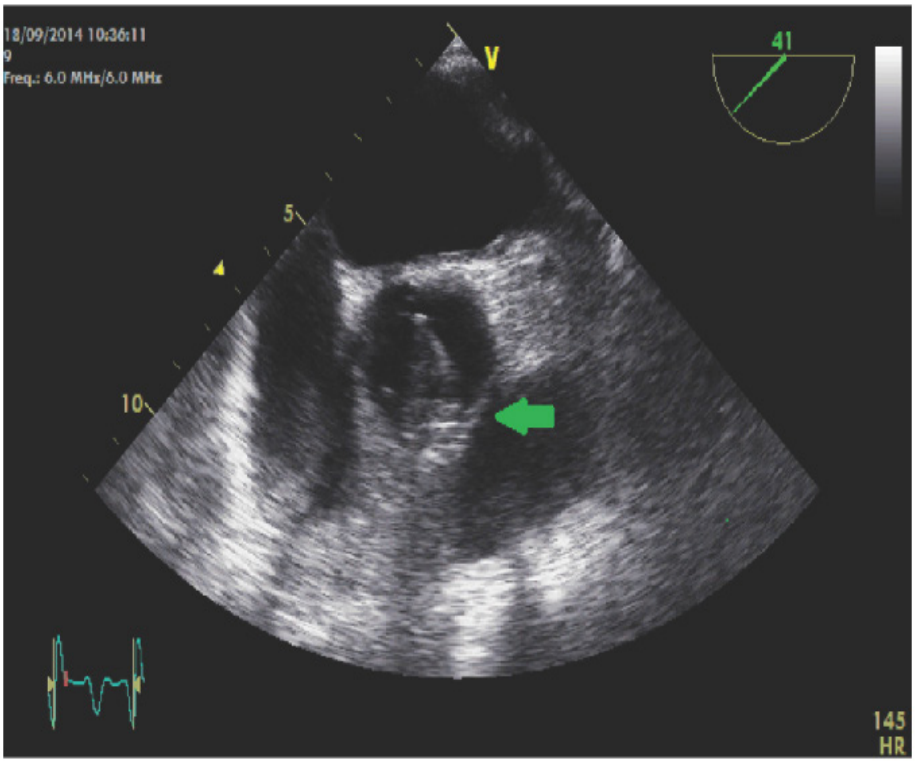

Figure 4. Transesophageal echocardiography. Interference of the mass with the closure of the prosthetic disc.

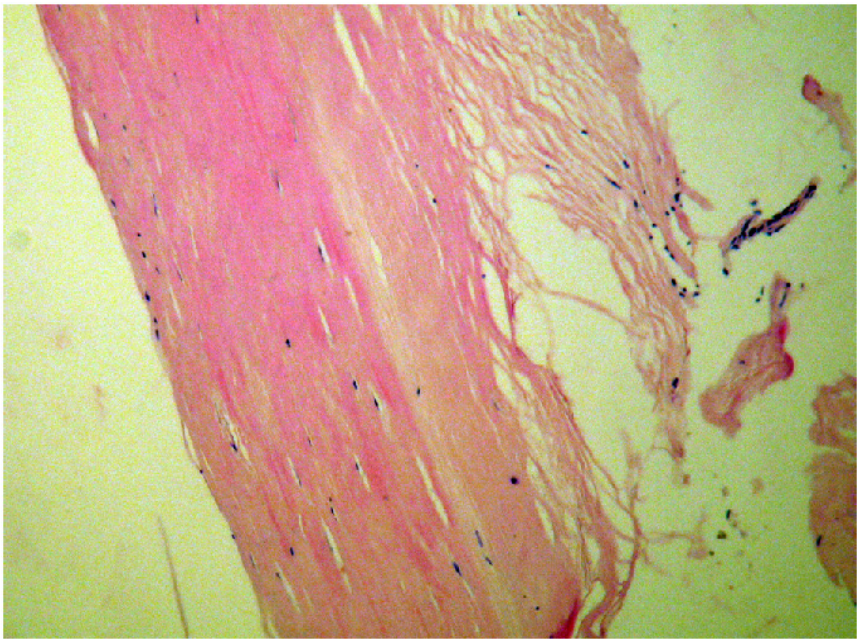

Figure 5. Hematoxylin and eosin staining, obx10. Microscopic aspect suggestive of pannus

Discussion

The case has several particular features. The discordance between repeated severe symptoms, ECG and enzymatic changes on the one hand, and the coronarographic and particularly, scintigraphic appearance on the other hand should be mentioned. The first 3 elements allow the diagnosis of acute myocardial infarction [14]. Normal coronarographic appearance does not exclude the presence of endothelial erosions visible by intracoronary imaging methods such as intravascular ultrasound (IVUS) or optical coherence tomography (OCT) [15], methods that were not applied in this case. Coronary spasm cannot be excluded, but there are several counter-arguments: it is frequently accompanied by ST segment elevation [16], it was not evidenced during coronarography [17], a procedure performed during the crisis, no response to anticalcium drugs [18]. What was intriguing and made the diagnosis of infarction difficult was the almost white scintigraphic result, suggesting that the event that induced the enzymatic increase left no visible traces on scintigraphy. Transient kinetic disorders evidenced by some echocardiograms and by scintigraphy raised questions: interoperative variability or post-ischemic myocardial stunning.

While excluding other possible cardiovascular diseases and taking into consideration prosthetic valve dysfunction, which was the only abnormality found, with the remission of symptoms post-intervention, final differential diagnosis was required between the various causes of increased transprosthetic gradients: stenosis due to pannus and/or thrombus, prosthesis-patient mismatch (moderate, previously documented). Differential diagnosis between thrombus and pannus is made based on clinical, echocardiographic and morphopathological criteria (Table I) [10,11,12] and is essential because therapeutic approaches are different. If in thrombotic obstruction, thrombolysis can be considered, this is contraindicated in the case of pannus, where the only valid therapeutic option is surgery (valve replacement or pannus removal) [19]. In our case, pannus was supported by chronic evolution, effective anticoagulation, subvalvular location at the level of the aortic prosthesis, non-interference with the opening of the prosthetic disc. In contrast, the acute evolution of symptoms, with frequent episodes occurring during rest, under the conditions of transprosthetic gradients approximately similar to those determined 3 years before, was not characteristic of uncomplicated pannus. A possible thrombotic association, with repeated microembolizations, in the absence of imaging or morphopathological evidence, remained hypothetical.

Therapeutic approach in case of severe stenosis due to pannus involves surgical reintervention, consisting of valve replacement or pannus removal. Valve replacement, a more complex procedure, is recommended only in certain conditions such as extensive circumferential pannus or a significantly altered prosthesis, based on the assumption of a marked inflammatory reaction in the case of replacement [1]. In this case, pannus removal was the only option decided. An alternative could have been replacement with a larger size prosthesis, possibly with the previous enlargement of the aortic ring [1,20], and/or a newer generation prosthesis, with intrinsically reduced transprosthetic gradients.

In conclusion, the presented case illustrates the

problems that pannus-related prosthetic valve dysfunction, a late complication of mechanical prosthetic valve replacement, can pose in terms of clinical picture (severity, discordant elements), differential diagnosis, as well as optimal therapeutic approach.

Table I. Differential diagnosis of pannus and thrombus [10,11,12].

	Pannus	Thrombus
Chronology	Minimum 12 months from date surgery	Occurs at any time(if late usually associated with pannus)
Evolution of symptoms	Subacute/chronic	Acute
Relation with anticoagulation(low INR)	Poor relationship	Strong relationship
Location	More frequent involving the aortic valve Subvalvular	More frequent involving the mitral valve Supra or subvalvular
Morphology	Small mass, undetected at TEE (semi)circular mass which involve the suture line Centripetal growth Valve restriction can be absent	Larger mass than pannus,detected at TEE Irregular mass attached to valves /hinge point Centrifugal growth Valve restriction
Echo density	>0.7	<0.7 (PPV=87%)

PPV= positive predictive value

References

1. Sakamoto Y, Hashimoto K, Okuyama H, Ishii S, Shingo T, Kagawa H. Prevalence of pannus formation after aortic valve replacement: clinical aspects and surgical management. J Artif Organs. 2006;9(3):199-202.
2. Shiraishi M, Kimura C, Takeuchi T, Kanoh M, Muramatsu K, Yamaguchi A, et al. Pannus-Related Mechanical Valve Dysfunction Leading to Hemodynamic Shock. Arch Clin Exp Surg. 2012;1(1):50-53.
3. Melenovsky V, Al Hiti H, Lupinek P, Marek T, Veiser T, Skalsky I, et al. Intermittent cardiogenic shock in a man with mechanical prosthesis of the aortic valve. circulation. 2011;124(1):e1-e3.
4. Giroux SK, Labinaz MX, Grisoli D, Klug AP, Veinot JP, Burwash IG. Intermittent, noncyclic dysfunction of a mechanical aortic prosthesis by pannus formation. J Am Soc Echocardiogr. 2010;23(1):107:e1-e3.
5. Karagiannis SE, Karatasakis G, Spargias K, Louka L, Poldermans D, Cokkinos DV. Intermittent acute aortic valve regurgitation: a case report of a prosthetic valve dysfunction. Eur J Echocardiogr. 2008;9(2):291-293.
6. Sorgdrager BJ, Tavilla G. E. van der Wall EE, Scholte AJ. Paroxysmal mechanical aortic valve prosthesis dysfunction: capturing the right moment. Neth Heart J. 2011;19(10):440–441
7. Hiremath C, Gajjar T, Rao N, Desai N. Intermittent dysfunction of prosthetic aortic valve causing acute coronary syndrome. Türk Göğüs Kalp Damar Cerrahisi Dergisi. 2013;21(4):1050-1053.
8. Darwazah AK. Recurrent pannus formation causing prosthetic aortic valve dysfunction: is excision without valve re-replacement applicable?. J Cardiothorac Surg. 2012;7:62.
9. Misawa Y. Valve-related complications after mechanical heart valve implantation. Surg Today. 2015;45(10):1205-1209.
10. Barbetseas J, Nagueh SF, Pitsavos C, Toutouzas PK, Quiñones MA, Zoghbi WA. Differentiating thrombus from pannus formation in obstructed mechanical prosthetic valves: an evaluation of clinical, transthoracic and transesophageal echocardiographic parameters. J Am Coll Cardiol. 1998;32(5):1410-1417.
11. Tanis W, Habets J, van den Brink RB, Symersky P, Budde RP, Chamuleau SA. Differentiation of thrombus from pannus as the cause of acquired mechanical prosthetic heart valve obstruction by non-invasive imaging: a review of the literature. Eur Heart J Cardiovasc Imaging. 2014;15(2):119-129.
12. Venkatesan S. What is pannus formation in prosthetic valves? What is the clinical significance?. Available from:http://drsvenkatesan.com/2009/03/20/what-is-pannus-formation-in-prosthetic-valves-what-is-the-clinical-significance/
13. Popescu BA, Ginghina C. Ecocardiografia Doppler [Doppler echocardiography]. Bucureşti: Editura Medicala; 2011.
14. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al.; ESC Committee For Practice Guidelines(CPG). Third universal definition of myocardial infarction. Eur Heart J. 2012;33(20):2551-2567.
15. Kato K, Yasutake M, Yonetsu T, Kim SJ, Xing L, Kratlian CM, et al. Intracoronary imaging modalities for vulnerable plaques. J Nippon Med Sch. 2011;78(6):340-351.
16. Davies O, Ajayeoba O, Kurian D. Coronary artery spasm: An often overlooked diagnosis. Niger Med J. 2014;55(4):356-358.
17. Sreckovic MJ, Jagic N, Zdravkovic V, Nikolic D, Tasic M, Sreckovic AM, et al. Coronary spasm that caused non-ST elevation myocardial infarction appeared in cath lab due to vasovagal reaction. Postepy Kardiol Interwencyjnej.

2014;10(2):138-140.
18. Hung MJ, Hu P, Hung MY. Coronary artery spasm: review and update. Int J Med Sci. 2014;11(11):1161-1171.
19. Cardoso G, Trabulo M, Andrade MJ, Ribeiras R, Rodrigues R, Neves JP, et al. A rare case of two mechanisms of prosthetic valve dysfunction in the same patient. Rev Port Cardiol. 2013;32(12):1037-1041.
20. Kuniyoshi Y, Koja K, Miyagi K, Shimoji M, Uezu T, Arakaki K, et al. Pannus formation in aortic valve prostheses in the late postoperative period. J Artif Organs. 2003;6(3):179-182.