



Unusual non-valvular left ventricular endocarditis presenting as multiple brain embolism

Aleksandar Redzek , Andrej Preveden , Svetlana Ruzicka Kaloci , Golub Samardzija , Mihaela Preveden , Miodrag Golubovic & Lazar Velicki

To cite this article: Aleksandar Redzek , Andrej Preveden , Svetlana Ruzicka Kaloci , Golub Samardzija , Mihaela Preveden , Miodrag Golubovic & Lazar Velicki (2020): Unusual non-valvular left ventricular endocarditis presenting as multiple brain embolism, *Acta Clinica Belgica*, DOI: [10.1080/17843286.2020.1789273](https://doi.org/10.1080/17843286.2020.1789273)

To link to this article: <https://doi.org/10.1080/17843286.2020.1789273>



Published online: 30 Jun 2020.



Submit your article to this journal 

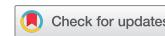


View related articles 



View Crossmark data 

CASE REPORT



Unusual non-valvular left ventricular endocarditis presenting as multiple brain embolism

Aleksandar Redzek^{a,b}, Andrej Preveden^{a,b}, Svetlana Ruzicka Kaloci^{a,c}, Golub Samardzija^{a,b}, Mihaela Preveden^b, Miodrag Golubovic^{a,b} and Lazar Velicki^{a,b}

^aFaculty of Medicine, University of Novi Sad, Novi Sad, Serbia; ^bClinic for Cardiovascular Surgery, Institute of Cardiovascular Diseases Vojvodina, Sremska Kamenica, Serbia; ^cClinic for Neurology, Clinical Center of Vojvodina, Novi Sad, Serbia

ABSTRACT

Introduction: Masses inside the heart can cause serious and life-threatening effects to the cardiovascular system, mainly because of hemodynamic obstruction of the blood flow, either in the heart cavities themselves or remotely due to embolization. In this paper, we report a case of left ventricular tumor mass which presented with neurological symptoms due to multiple brain embolism.

Case report: A 35-year-old female patient presented with right hemiparesis and dysarthria. Seven days prior to admission she had elevated body temperature and started taking antibiotics. Inflammatory markers were not elevated, and blood cultures were negative. Computerized tomography (CT) and magnetic resonance imaging (MRI) of the brain revealed multiple acute ischemic lesions. Echocardiography showed the presence of a lobular mass inside the left ventricle, which was attached to the basal segment of the lateral left ventricular wall. Based on the laboratory results and additional heart imaging (CT and MRI) the mass was primarily suspected to be a tumor. It was surgically removed. Microscopic analysis of the removed tissue revealed a non-specific endocardial inflammation with formed fresh fibrin thrombi on the surface. During the postoperative recovery intense physical rehabilitation was being performed, so the initial neurological deficit was completely withdrawn.

Conclusion: Intracardiac masses can cause serious and potentially fatal complications that often present with dramatic clinical symptoms. Despite the comprehensive clinical, laboratory, and imaging investigations, intracardiac masses can be hard to distinguish until the definite microscopic analysis. However, with the right approach and multidisciplinary collaboration, they can be successfully managed.

KEYWORDS

Heart tumor; left ventricular mass; blood culture-negative endocarditis; mural endocarditis; stroke; brain embolism

Introduction

Masses inside heart chambers are not a common finding. Based on histology, intracavitory masses can be classified as tumors, thrombi, vegetations, and calcific lesions [1]. Regardless of the structure, these masses can cause serious and life-threatening effects to the cardiovascular system, mainly as a result of hemodynamic obstruction to the blood flow, either in the heart cavities themselves or remotely due to embolization.

In this paper, we report a case of left ventricular tissue mass which presented with neurological symptoms due to multiple brain embolism.

Case report

A 35-year-old female patient presented with right hemiparesis and dysarthria. Seven days prior to admission the patient had elevated body temperature and started taking antibiotics. Upon admission she was conscious, body temperature was 36.4 °C, respiratory rate and blood pressure were normal. Electrocardiogram (ECG) showed normal sinus rhythm with 90 beats per minute.

Laboratory blood tests revealed mild leukocytosis (13.2 $\times 10^9/l$), while other inflammatory markers were normal (C-reactive protein 2.3 mg/l; procalcitonin 0.05 mg/ml).

Head computerized tomography (CT) on the admission revealed a discrete ischemic lesion in the left cerebral hemisphere with suspected occlusion of the left middle cerebral artery (MCA). Further evaluation included microemboli detection with transcranial color Doppler ultrasonography (TCD). During a 40-minute examination, a total of 71 microemboli in both hemispheres were registered (Figure 1(a)).

For a more detailed evaluation of the ischemic changes of the brain, magnetic resonance imaging (MRI) of the brain was performed (Figure 1(b)). MRI detected multiple acute infarcts of the left cerebral hemisphere dominantly in the MCA basin, but also acute microinfarcts in the right frontal lobe and the left cerebellar hemisphere. Such a presentation with multiple bilateral ischemic lesions of the brain detected by both CT and MRI confirmed the embolic etiology of the lesions.

Echocardiography was performed in search of the origin of the emboli. A tissue formation measuring

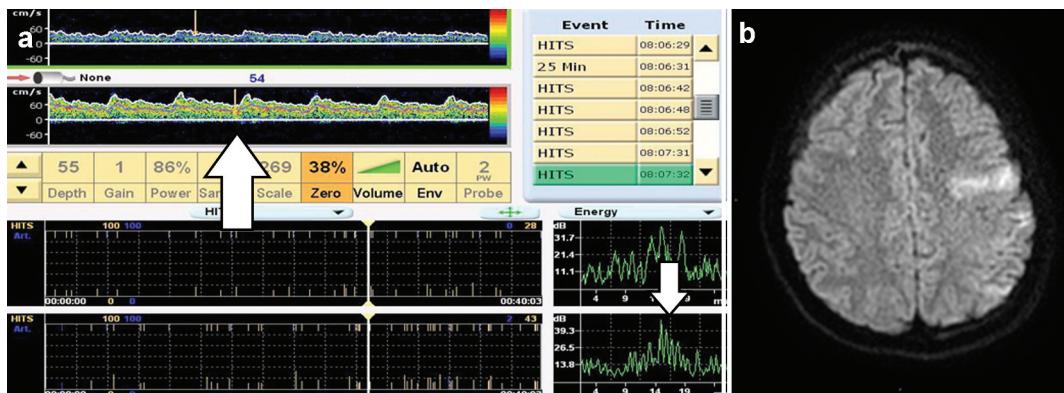


Figure 1. Multiple ischemic lesions detected by brain imaging: (a) – Transcranial color Doppler ultrasonography microembolus detection: Both medial cerebral arteries (MCA) were insonated for 40 minutes. The frequent high-intensity transient signals (HITS) were recorded on both sides with the characteristics of solid microemboli. MES display in the MCA spectrum (large arrow) and duration of the signal in ms (small arrow); (b) – Brain magnetic resonance imaging: multiple acute infarcts of embolic etiology in both cerebral hemispheres.

19 × 14 mm was registered inside the left ventricle widely attached to the basal segment of the lateral wall in the region of the posterior mitral ring (Figure 2(a-b)). It demonstrated a high embolization potential. However, based on its echocardiography features, it could not be determined whether this was a tumor, vegetation, or a thrombus. The mitral valve was competent without any direct contact with the observed mass. Patent foramen ovale (PFO) was also found. Other heart valves were competent. No regional wall motion abnormalities were detected, and the systolic function of the left ventricle was preserved (ejection fraction 64%).

Further diagnostics included cardiac CT and MRI to differentiate the structure of the detected mass and to get a better insight into its relation to the surrounding cardiac structures. These imaging methods confirmed the presence of a lobular mass measuring 15 mm in size inside the left ventricle attached to the basal segment of the lateral wall, in close contact to chordae tendineae of the anterolateral papillary muscle, without direct contact to the mitral valve (Figure 2(c)). Characteristics of the observed mass were not

distinctive enough, so the diagnosis remained uncertain, with a tumor, vegetation, and thrombus all being suspected as possible. However, based on its radiological features on both CT and MRI (interpreted by two different radiologists), the mass primarily corresponded to the papillary fibroelastoma, which was adopted as a working diagnosis for the moment.

Since it was determined that it was a mass inside the left ventricle with high embolic potential, surgical treatment was indicated by the multidisciplinary team consisting of cardiologists, cardiac surgeon, anesthesiologist and neurologist.

The surgery was performed in general endotracheal anaesthesia through a median sternotomy. After heparinization, standard cardio-pulmonary bypass with ascending aortic and right atrial cannulation was initiated. Cardioplegia was introduced anterogradely through the ascending aorta. Right atriotomy with transseptal approach was used to access the left atrium. The mitral apparatus including leaflets, chordae tendineae and papillary muscles appeared normal with preserved morphology. The tissue mass was found inside the left ventricle just below the posterior mitral leaflet in

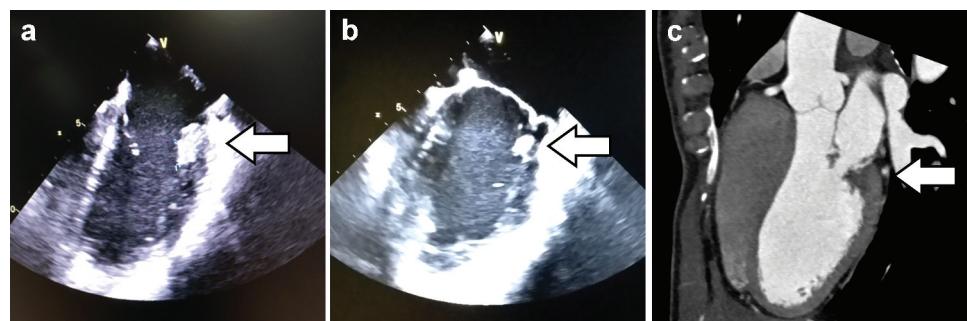


Figure 2. Tumor mass inside the left ventricle detected by heart imaging: (a-b) – Transesophageal echocardiography (diastole [a] and systole [b]): Below the mitral apparatus there is a hyperechogenic formation attached under the posterior mitral cusp without direct contact to the mitral apparatus; (c) – Cardiac computerized tomography: Hypodense formation with lobular edges inside the left ventricle in close contact with chordae of anterolateral papillary muscle and the basal segment of the left ventricular lateral wall.

the area of P1 and P2 scallops (Figure 3). The mass was completely removed, with no injury to the mitral apparatus. The PFO was closed. Standard draining, temporary pacing, and closing technique were used. After the operation, the patient was transferred to the intensive care unit.

The immediate postoperative course was uneventful. On the first postoperative day, the patient developed sinus tachycardia, and on one occasion non-sustained ventricular tachycardia was recorded, so beta-blocker therapy was introduced which lead to the heart rhythm stabilization. The patient was hemodynamically stable during the whole time. She received a transfusion of red blood cells due to anemia, while all other laboratory findings were normal.

Microscopic analysis of the removed tissue mass showed a non-specific endocardial inflammation with thick inflammatory cell infiltrates, and with fresh fibrin thrombi formed on the surface (Figure 4). Such a structure primarily resembled vegetation in infective endocarditis and was not at all suggestive of papillary fibroelastoma or any other tumor.

During the postoperative recovery intense physical rehabilitation was being performed, so the initial neurological deficit was completely withdrawn. The patient was discharged on the fifth postoperative day in good general condition with acetylsalicylic acid and bisoprolol in therapy.

The patient came to check-up one month after discharge. She was free of symptoms and without a neurological deficit. A 24-hour ECG Holter monitoring recorded normal sinus rhythm with a mean heart rate of 75 beats per minute, without ventricular arrhythmias.

Discussion

We report a case of intracardiac tissue mass with a dramatic neurological presentation due to multiple brain embolism. After the initial clinical evaluation and thorough non-invasive investigations, the origin of the emboli was located inside the left ventricle.

Apart from comprehensive brain imaging including CT, MRI, and TCD, which revealed the cause of the

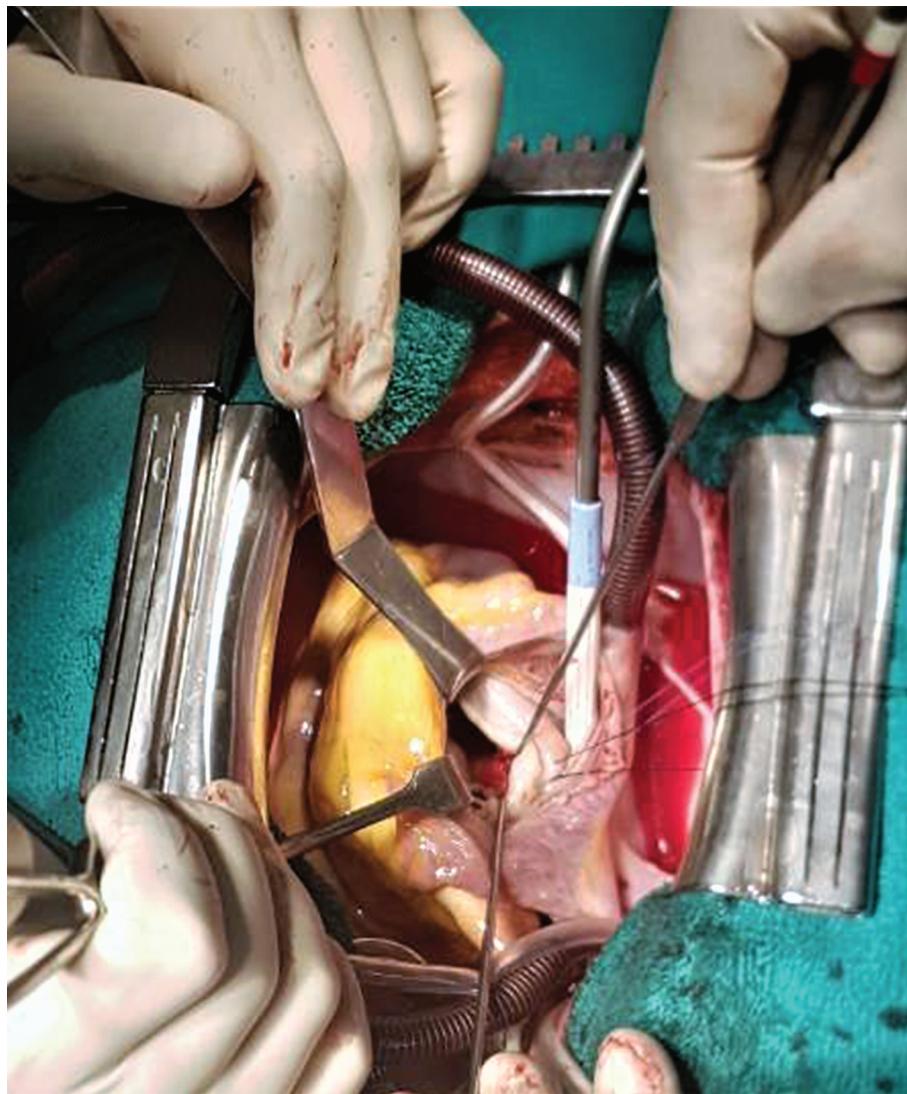


Figure 3. Intraoperative finding of the tissue mass inside the left ventricle.

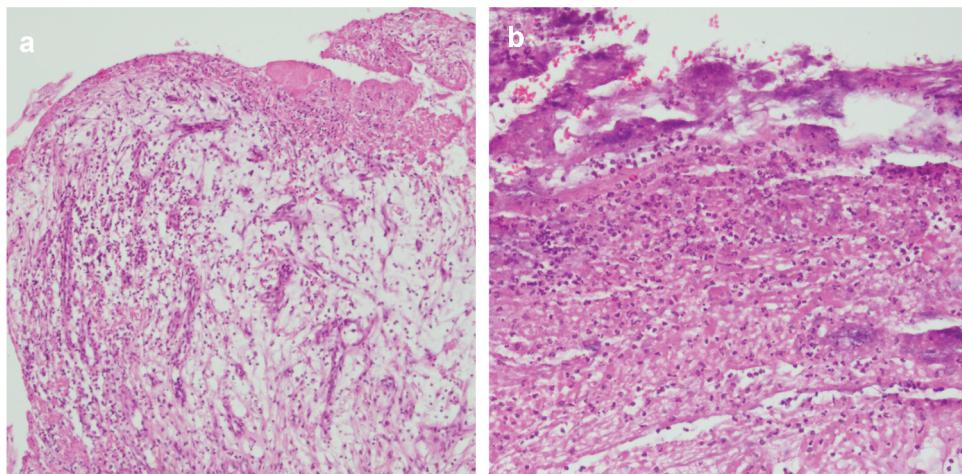


Figure 4. Pathohistological finding of the extracted tissue ((a) – H&E, 10x; (b) – H&E, 20x): Fragments of young granulation tissue with numerous blood vessels of the swollen endothelial cells, infused with thick inflammatory cell infiltrates, including lymphocytes, plasma cells, neutrophilic and eosinophilic granulocytes. Some fragments of the tissue partly have the myxoid appearance and are partly built of hyalinized connective tissue. On the surface of the described fragments of the granulation tissue and separately, there are fresh fibrin thrombi made from a network of fibrin fibers mixed with numerous neutrophilic granulocytes.

neurological deficit, the crucial role in the final diagnosis was delivered by echocardiography. Initially, transthoracic echocardiography revealed a hyperechogenic formation inside the left ventricle. However, a more detailed evaluation regarding its exact location, attachment point and morphology was possible only by transesophageal echocardiography, which is the superior method for the characterization and assessment of intracardiac masses [2]. The findings of the intraoperative transesophageal echocardiography can often influence the surgical technique itself and force a modification of the planned procedure in cases of intracardiac mass removal [3].

Although the patient was afebrile on admission, with just slightly elevated leukocytes and normal inflammatory markers, infective endocarditis was suspected based on the echocardiography finding and the anamnestic information about elevated body temperature and the use of antibiotics several days prior admission. However, blood cultures that were taken upon admission remained negative, and the subsequent additional imaging of the heart with CT and MRI implied that the mass primarily resembled papillary fibroelastoma. Cardiac CT and MRI are synergistic methods that are often used in addition to echocardiography for the evaluation of complex and confusing intracardiac masses [4].

Given all the available clinical, laboratory, and imaging data at that moment, infective endocarditis was ruled out and a tumor, most likely papillary fibroelastoma, was adopted as the primary suspect. Papillary fibroelastoma is a very rare cardiac tumor, most commonly of valvular origin, with an incidence of about 5.9% among primary benign cardiac tumors [5]. Myxoma, on the other hand, is the most common

tumor of cardiac origin, accounting for as much as 50% of cases [6].

Nonetheless, microscopic analysis of the surgically removed tissue from the left ventricle ultimately showed that it was not a tumor, but a combination of inflamed endocardium and thrombus instead.

This is not the first case of this kind in the literature. Raut et al. [7] and Dinesh Kumar et al. [8] recently published two cases with similar neurological symptoms and a suspected tumor inside the left ventricle based on echocardiography finding. However, microscopic analysis of the removed tissue after the operation in both cases revealed that it was not a tumor, but a thrombus instead.

Thrombus is, as a matter of fact, the most common cause of left ventricular mass [1]. It is usually associated with complications of coronary artery disease, such as wall motion abnormalities and left ventricular aneurysm [9], although these were not present in our patient as observed by echocardiography. On the other hand, systemic embolism does represent a common clinical manifestation of a thrombus [10].

Such a microscopic finding could be explained by blood culture-negative endocarditis, which is, in fact, not that rare and can account for as much as 31% of total infective endocarditis cases [11]. Blood culture-negative endocarditis can be caused by rare pathogens such as fungi and fastidious slow-growing bacteria, although it is also associated with antibiotics use before blood sampling [12,13], which could be the possible explanation in our case.

Endocarditis without valvular involvement is extremely rare, however, there are a few case reports describing mural localization of vegetation inside the left ventricle without direct contact to the mitral valve

[14–18], similar to our finding. Interestingly, in all of these cases, *Staphylococcus aureus* was identified as the responsible pathogen, whereas our patient had negative blood cultures.

According to the modified Duke criteria for the diagnosis of infective endocarditis [19], our patient classifies as 'possible endocarditis', with having one major (possible vegetation on echocardiography) and two minor criteria (elevated body temperature and major arterial emboli). Unfortunately, due to the strongly suspected diagnosis of heart tumor at the moment of the operation, the surgically removed tissue mass was sent only to microscopic analysis and not to microbiology analysis, so the culture, which might have provided some crucial information for the final diagnosis, was not performed.

Tahara et al. [18] analyzed 19 cases of mural endocarditis without valvular involvement. Based on their finding, the left ventricle is indeed the most commonplace of mural endocarditis (13/19 cases), however, it is associated with risk factors including immune deficiency, intravenous drug use, and recent cardiac surgery, out of which our patient had none. On the other hand, it is noted that mural endocarditis usually presents with only mild inflammation in laboratory analyses. Considering that our patient had only mild leukocytosis and normal levels of C-reactive protein and procalcitonin, these findings could also go in favor of mural endocarditis as a definite diagnosis.

A ventricular septal defect is reported as the most frequent congenital heart disease associated with infective endocarditis, however, it can also be observed in patients with PFO, patent ductus arteriosus and atrioventricular septal defect [20]. We hypothesize that the PFO in our patient might have played a crucial role in the pathophysiology of endocarditis by transporting infective agents from venous circulation to systemic circulation and the left ventricle.

In conclusion, it should be noted that intracardiac masses are capable of causing serious and potentially fatal complications that often present with dramatic clinical symptoms. Despite comprehensive clinical, laboratory, and imaging investigations, intracardiac masses can be hard to distinguish until the definite microscopic analysis and histology diagnosis. However, with the right approach and multidisciplinary collaboration, they can be successfully managed.

Disclosure statement

No potential conflict of interest was reported by the authors.

ORCID

Andrej Preveden  <http://orcid.org/0000-0003-3572-2604>
Lazar Velicki  <http://orcid.org/0000-0002-2907-819X>

References

- [1] Basso C, Rizzo S, Valente M, et al. Cardiac masses and tumours. *Heart*. 2016 Aug 1;102(15):1230–1245.
- [2] Mankad R, Herrmann J. Cardiac tumors: echo assessment. *Echo Res Pract*. 2016 Dec;3(4):R65–R77.
- [3] Dujardin KS, Click RL, Oh JK. The role of intraoperative transesophageal echocardiography in patients undergoing cardiac mass removal. *J Am Soc Echocardiography*. 2000 Dec;13(12):1080–1083.
- [4] Kassop D, Donovan MS, Cheezum MK, et al. Cardiac masses on cardiac CT: a review. *Curr Cardiovasc Imaging Rep*. 2014;7:9281..
- [5] Meng Q, Lai H, Lima J, et al. Echocardiographic and pathologic characteristics of primary cardiac tumors: a study of 149 cases. *Int J Cardiol*. 2002 Jul;84(1):69–75..
- [6] Araoz PA, Mulvagh SL, Tazelaar HD, et al. CT and MR imaging of benign primary cardiac neoplasms with echocardiographic correlation. *Radiographics*. 2000 Sep-Oct;20(5):1303–1319. .
- [7] Raut MS, Maheshwari A, Dubey S, et al. Left ventricular mass: myxoma or thrombus? *Ann Card Anaesth*. 2015 Jan-Mar;18(1):95–97..
- [8] Dinesh Kumar US, Shetty SP, Sujay KR, et al. Left ventricular mass: A tumor or a thrombus diagnostic dilemma. *Ann Card Anaesth*. 2016 Oct-Dec;19(4):728–732..
- [9] Sharma ND, McCullough PA, Philbin EF, et al. Left ventricular thrombus and subsequent thromboembolism in patients with severe systolic dysfunction. *Chest*. 2000 Feb;117(2):314–320..
- [10] Preveden A, Kovačević M, Šušak S, et al. Coronary embolism causing myocardial infarction after heart valve surgery. *Vojnosanit Pregl*. Online First November, 2018.
- [11] Brouqui P, Raoult D. Endocarditis due to rare and fastidious bacteria. *Clin Microbiol Rev*. 2001 Jan;14(1):177–207.
- [12] Houpikian P, Raoult D. Blood culture-negative endocarditis in a reference center: etiologic diagnosis of 348 cases. *Medicine (Baltimore)*. 2005 May;84(3):162–173.
- [13] Fournier PE, Thuny F, Richet H, et al. Comprehensive diagnostic strategy for blood culture-negative endocarditis: a prospective study of 819 new cases. *Clin Infect Dis*. 2010 Jul 15;51(2):131–140.
- [14] Litmathe J, Fussen R, Heinzel A, et al. An unusual agent for an unusual localization of infective endocarditis. *Perfusion*. 2017 Nov;32(8):691–694..
- [15] Shiono Y, Hirata K, Takemoto K, et al. Multiple mobile structures attached to the left ventricular wall in infective endocarditis. *Eur Heart J*. 2015 Jan 21;36(4):213.
- [16] Takemoto K, Hirata K, Kubo T, et al. Primary mural endocarditis with enormous vegetation in the left ventricle. *Eur Heart J Cardiovasc Imaging*. 2013 Jun;14(6):514..
- [17] Adel A, Jones E, Johns J, et al. Bacterial mural endocarditis. A case series. *Heart Lung Circul*. 2014 Aug;23(8):e172–9..
- [18] Tahara M, Nagai T, Takase Y, et al. Primary mural endocarditis without valvular involvement. *J Ultrasound Med*. 2017 Mar;36(3):659–664. .
- [19] Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. *Clin Infect Dis*. 2000 Apr;30(4):633–638. .
- [20] Knirsch W, Nadal D. Infective endocarditis in congenital heart disease. *Eur J Pediatr*. 2011 Sep;170(9):1111–1127.