



Complex Cause of Ischaemic Stroke in Patient after Mitral Bioprosthesis Implantation - A Case Report

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Abstract

We present the history of 60-years-old woman with mitral and aortic bioprosthesis that have been implanted together with left atrial appendage closure. The patient had been treated with VKA and low dose of acetylsalicylic acid since the surgery. On 28th day of postoperative period an ischemic stroke occurred. In transoesophageal echocardiography we have discovered non-obstructive thrombosis of the artificial mitral annulus and a leakiness in left atrial appendage (LAA) closure. There was rapid outflow through the gap in closure directing to mitral annulus in close proximity to visible thrombus. The pathologic flow might have had the contribution in thrombus mobilization and stroke occurrence. We have decided to change anticoagulation to low molecular weight heparin. In control echocardiography, during heparin therapy, thirteen days later, there was remarkable worsening of annulus thrombosis with multiple thrombi present. Re-exchange anticoagulation to warfarin yielded in almost complete resolution of thrombosis after eleven days. We decided to continue anticoagulation over recommended three months not only due to incident of thrombosis but also suspicion of history of atrial fibrillation and presence of disrupted LAA closure.

Case Summary

60-years-old woman with a history of caseous mitral annulus calcification, severe mitral regurgitation, moderate aortic stenosis and regurgitation, have had implantation of mitral and aortic bioprostheses together with left atrial appendage closure. In postoperative period a typical anticoagulation was implemented - low molecular weight heparin followed by vitamin K antagonist under international normalized ratio (INR) control. Due to coexisting coronary artery disease and venous graft implanted an antiplatelet treatment (75 mg of acetylsalicylic acid) was also administered. On 28th day after the surgery symptoms of right disparetion, ocular ataxia, facial numbness and balance disorders occurred. Ultrasonography and computed tomography angiogram (angi-oCT) revealed an obstruction of right vertebral artery. No visible ischemic focus was discovered in brain computed tomography. INR slightly exceeded therapeutic range [1-3]

In transthoracic echocardiography we have discovered turbulent flow through mitral prosthesis with no other dysfunction. Appearance and function of aortic bioprosthesis and other heart structures were normal. In transoesophageal imaging there

was small thrombus in lateral part of mitral annulus. Due to incomplete left atrial appendage closure there was a flow through the leak. This outflow was rapid, turbulent and directed straight to mitral annulus, in close proximity to present thrombus. We suspect mobilization of a thrombus resulting in stroke by rapid left atrial appendage outflow. After the diagnosis of non-obstructive prosthetic thrombosis, anticoagulation with low molecular weight heparin was implemented. In control echocardiography, thirteen days later, we have confirmed not only previously diagnosed thrombus but also four new mobile thrombi. Our next decision was to implement vitamin K antagonist (VKA) with rigid INR control. After two weeks of the VKA therapy we have observed almost complete resolution of annulus thrombosis. We have continued VKA with target INR 3,0 together with low dose of acetylsalicylic acid for three months. After the three months the anticoagulation was switched to rivaroxaban. Control TOE revealed worsening of thrombosis after one week of rivaroxaban treatment, so we decided to continue further anticoagulation with VKA permanently. Subsequent TOE confirmed the accuracy of VKA treatment, there was only very small, residual thrombus visible.

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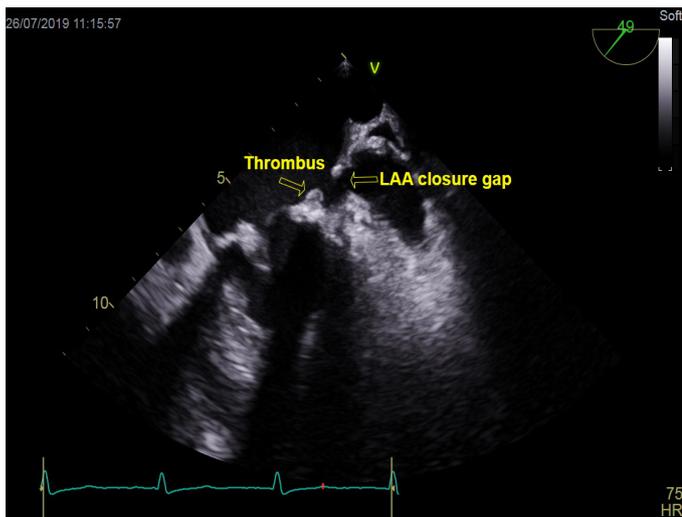


Figure 1: Thrombus on mitral annulus and gap in LAA patch.

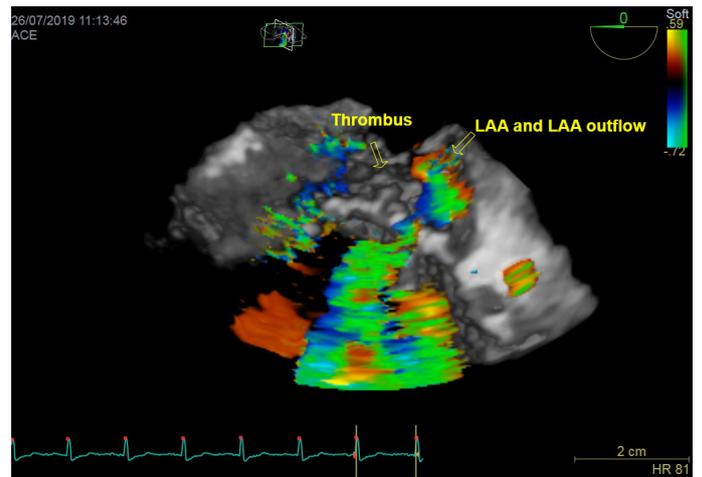


Figure 4: 3D reconstruction of lateral part of mitral annulus.

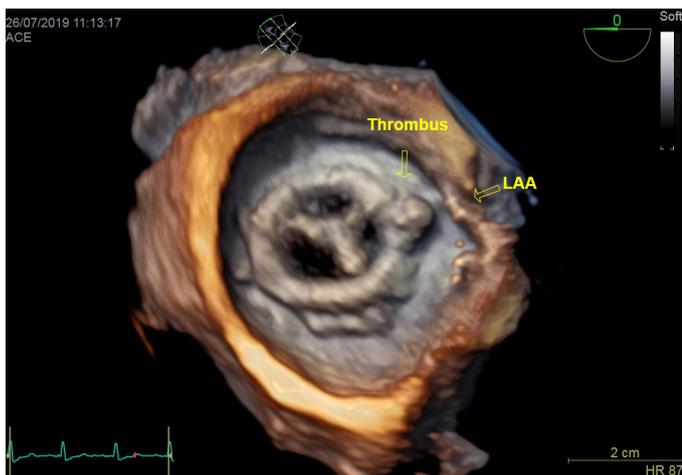


Figure 2: Thrombus on mitral annulus close to LAA in 3D.

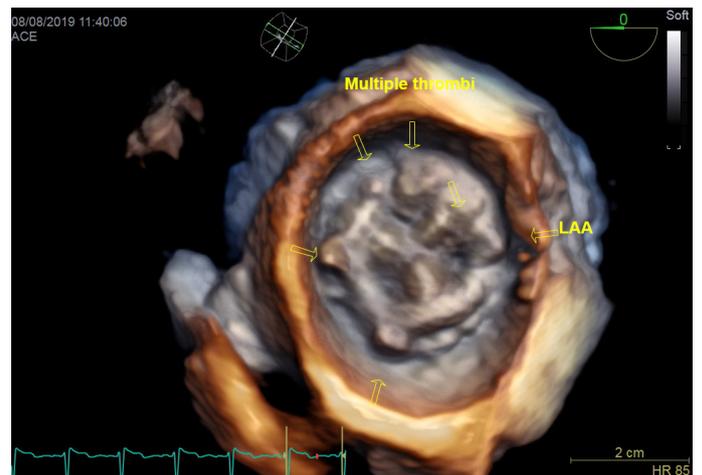


Figure 5: Worsening of annulus thrombosis on low molecular weight heparin therapy.

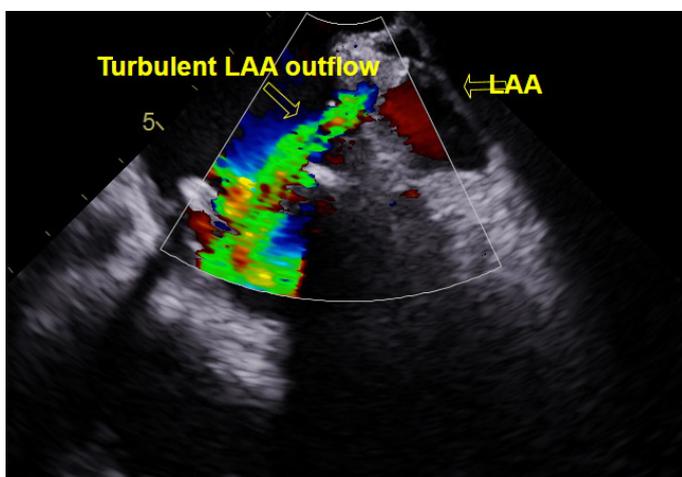


Figure 3: Turbulent LAA outflow directing to mitral annulus.



Figure 6: Resolution of thrombosis on vitamin K antagonist therapy.



Figure 7: Re-worsening on rivaroxaban 3 months later.

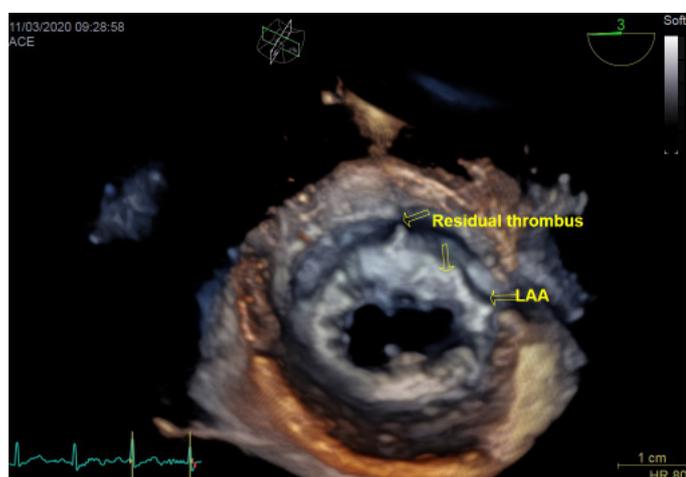


Figure 8: Improvement after returning to vitamin K antagonist.

Discussion

Patients after cardiac surgery are high risk group of ischemic stroke [4]. The risk is even higher after valve surgery [5]. Mitral valve replacement presents a 1,62% threat of stroke in postoperative period [1]. Current recommendations direct us to use vitamin K anticoagulants for three months after mitral valve replacement (bioprosthesis) [4,5].

Taking into account prevalence of atrial fibrillation in patients subjected to mitral valve surgery and specific surgical access, closure of left atrial appendage emerged as almost a routine [6].

Dispatching of the LAA closure is rare [7]. In our patient it took place probably due to high pressure generated by contracting LAA muscle. Unfortunately outflow through the crack was directing to mitral annulus and probably have mobilized thrombus previously present in this place.

The bare metal of mitral annulus is highly thrombogenic. After the period of epithelialization, (approximately 3 months), thromboembolic risk grows low and the anticoagulation may be stopped.

In this case we think, that continuing of anticoagulation permanently is a good choice. Arguments for further anticoagulation are: thromboembolic event in postoperative period, unclosed left atrial appendage, presence of malign LAA flow that probably interfere epithelialization of mitral annulus, calcification of native mitral annulus [2], and suspicion of atrial fibrillation. In this specific case there was no documented atrial fibrillation but the risk of the arrhythmia in patient with the history of severe mitral regurgitation is very high [3]. Vitamin-K-antagonists turned out to be better than other anticoagulants: temporary switching to both parenteral antiXa-antagonist (enoxaparin) and oral antiXa-antagonist (rivaroxaban) resulted in rapid worsening of thrombosis. In both cases, returning to VKA improved the state significantly.

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