EMBOLIC STROKES DUE TO LEFT CARDIAC THROMBI

Kristina Lazarovska
General hospital the 8th of September, Skopje, North Macedonia, dr.tinalazarovska.dr@gmail.com
Aleksandra Gulevska Vucinic
General hospital the 8th of September, Skopje, North Macedonia, gulevska_aleksandra@yahoo.com

Abstract: Introduction: Cardio-embolic strokes occur in about 20% of stroke patients. Left ventricular thrombi come second to atrial thrombi from atrial fibrillation as a cause of cardioembolic embolization. Myocardial infarction and dilated cardiomyopathy are most frequently associated with left ventricular thrombi formation. Dyskinetic segments that remodel into aneurysms and left atrial appendage remain chronic foci for thrombosis. Large size infarcts are known to be associated with high risks for hemorrhagic transformation.
Material and methods: In our case we presented a middle-aged patient, 42 years old, male, from the emergency department with acute onset of inability to speak and right sided weakness. Initial CT brain showed a small post ischemic zone in the left parietal lobe. A repeated CT brain after 48 hours showed a large left middle cerebral artery territory infarct with focal areas of hemorrhagic transformation corresponding to the neurological symptoms presented. We used the importance of echocardiography and transoesophageal echocardiography (TOE) in identifying the etiology and clinical evaluation of a ischemic embolic stroke with hemorrhagic transformation. The patient has comorbidities (ischemic cardiomyopathy, hypertension, dyslipidemia) and had a previous cerebral ischemic event. He was on antiplateleted therapy (Clopidogrel 75mg/day) because of previous cardiac ischemic event (myocardial infarction). Initially the patient was observed and treated by a multidisciplinary team consisting of a cardiologist, neurologist and transfusiologist who opted not to initiate full anticoagulation in the beginning. Instead the patient received low molecular weight heparin (enoxiparin) in the acute phase of the illness, the first three weeks and after that was continued with lifelong oral anticoagulation therapy (according to CHA2DS2-VASc 4 and HAS-BLED score 2). The therapy with antiplateled therapy was discontinued because of high risk of hemorrhagic transformation and reinitiated after 3 months. On discharge from the hospital the patient had mild neurological deficit, hemodynamically compensated, stable, without hemorrhagic event with recommendation for oral anticoagulant and antiplateled therapy.
Conclusion: Early and prompt cardiac evaluation using echocardiography and TOE in the emergency department is necessary in order to detect the etiology of embolic strokes. Multidisciplinary team is extremely important for the evaluation and guidance the patients with this kind comorbites.
Keywords: left ventricular and atrial thrombi, echocardiography, TOE, embolic strokes.

1. INTRODUCTION

Left cardiac thrombi is associated with significant risk of ischemic stroke and peripheral embolization (Aloysius Sheng-Ting Leow et al, 2019). They come second to atrial thrombi from atrial fibrillation as a cause of cardioembolic embolization. Post myocardial infarction left ventricle thrombi increases the risk of embolization significantly (Bhatnagar UB et al,2018), particularly if anticoagulant treatment is not used. For these patients choosing the best treatment strategy has always been challenging (Habash & Vallurupalli, 2017).

2. MATERIALS AND METHOD

A 40-year-old man came to the emergency department presented with acute onset of inability to speak and right sided weakness. His previous medical history was hypertension, hyperlipidemia, ischemic cardiomyopathy with preserved ejection fraction and anterior ST-elevation myocardial infarction (STEMI) with primary PCI: PTCA/Stent of proximal LAD two years ago. The patient was on two antihypertensive medications, beta-blocker, sinus rhythm1. with heart rate 95 beats per minute, central right facial palsy, aphasia, with right sided hemiplegia; laboratory findings (cardiac enzymes, PT-INR, d-dimmers) were unremarkable. Initial CT brain showed a small post-ischemic zone in the left parietal lobe. Doppler ultrasound of carotid arteries was unremarkable. The echocardiography in the emergency department revealed normal dimension of the cavities, mildly reduced ejection fraction, remodeling of the left ventricle with septo-apical aneurysm with akinetic walls (apical segment of the septum and lateral wall, apex), a mobile hypoechoic thrombus in the apex with great thromboembolic potential. The transoesophageal echocardiography (TOE) confirmed the apical thrombus and revealed a thrombus in the left atria appendage, without any defects of the interatrial septum. A repeated CT brain after 48 hours showed a large left middle cerebral artery territory infarct with focal areas of hemorrhagic transformations. Multidisciplinary team consisting of a cardiologist, neurologist and transfusiologist discussed over the decision what kind of anticoagulant to use. Preliminary study

suggests that low molecular weight heparins are well tolerated and efficient in terms of left ventricular thrombi disappearance or size reduction (Meurin F.,2005). So we opted not to initiate full anticoagulation. Instead, the patient received low molecular weight heparin (enoxaparin) 60mg+40mg per day. Three weeks later the control transoesophageal echocardiography demonstrated a complete regression of the thrombus in the left ventricle and in the left appendage, without any evidence of further systemic embolization. A control MRI brain scan after 3 weeks showed a complete resolution of the cerebral hemorrhage and oral anticoagulant (NOAC-Rivaroxaban 20mg) was initiated. Because the coronarography made 2 years ago, showed diffuse coronary artery disease of the arteries (stenosis lower than 50%), our patient needed an antiplatelet therapy (Clopidogrel 75mg.), which was reinitiated after 3 months, in consultation with neurologist (Parker & Storey,2021). During the hospitalization the patient was monitored all the time, and there were not paroxysmal arrhythmias noted. Additional transfusiological analysis that were made were unremarkable. On discharge from the hospital the patient had mild neurological deficit, was hemodynamically compensated, stable, without hemorrhagic events. The recommended therapy was direct oral anticoagulant, angiotensin-converting enzyme inhibitor, beta-blocker, antilipemic and proton pump inhibitor, antiplatelet therapy after 3 months. On the first control visit, TTE was performed and control coronarography scheduled. The patient was followed up regularly -after 1 month, after 3 months and after 6 months, with no hemorrhagic events notified.

3. DISCUSSIONS

For patients with confirmed left cardiac thrombi, current guidelines recommend starting anticoagulant plus antiplatelet therapy for 3-6 months; if there is evidence of resolution of the thrombi, oral anticoagulant can be discontinued. However, in clinical practice, many patients encountered left cardiac thrombi recurrence after anticoagulants discontinuation. Currently, there are limited research data on the incidence of recurrence of the left cardiac thrombi and great difference in different research reports. The issue of anticoagulant strategies for patients at high risk for left cardiac thrombi recurrence after thrombus regression was not specified in current guidelines, which may expose these patients to fatal embolism consequences (Sun W et al, 2021). Here we suggest that more attention should be given to this topic by multidisciplinary clinicians, and for certain patients (as described above in our case), it is still necessary to continue using anticoagulants (even for a lifetime) after thrombi regression (Merkler et al, 2018). Although long-term anticoagulant therapy may benefit patients, it is worth noting that the implementation of anticoagulant therapy should be individualized and evaluated according to the risk stratification and anticoagulant tolerance (McCarthy CP et al, 2018).

4. CONCLUSION

The case we presented showed the importance of using a multidisciplinary approach to every patient. Diagnostic tools like echocardiography and transoesophageal echocardiography are of great importance in patients with neurological pathology so we can determine the etiology and treatment of patients with this kind comorbites (Stollberger C. et al, 2017).

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