FREE FLOATING THROMBUS IN THE NON-ATHEROSCLEROTIC, NON-ANEURYSMAL ASCENDING THORACIC AORTA – A RARE ENTITY

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SLOBODNI FLOTIRAJUĆI TROMB U NEATEROSKLEROTSKI, NEANEURIZMATSKI IZMENJENOJ ASCENDENTNOJ AORTI – REDAK ENTITET

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ABSTRACT

Free floating thrombus (FFT) attached to the healthy aortic wall in ascending aorta is a very rare condition discovered more frequently with introduction of new imaging modalities. One of the main causes of this condition is hypercoagulable conditions and the main manifestations are distal embolisations.

Method We report a case of 59 years old male who suffered from acute ischemia of lower limb due to emboli from floating thrombus in ascending thoracic aorta. Embolus in lower limb arteries was detected first by digital subtraction angiography and FFT later on by computer tomography. Anticoagulation therapy was started immediately. After surgical removal of embolus in lower leg, hypercoagulable disorder was revealed. On control computer tomography scan, conducted after 10 days, no sign of thrombus in ascending aorta was seen.

Discussion We believe that initial treatment strategy for FFT should be conservative anticoagulate therapy that in many cases would be also the optimal solution, however no studies confirmed this, nor a medical treatment algorithm is suggested.

Key words: aorta, thoracic; thrombosis; anticoagulants.

INTRODUCTION

Thrombus in ascending aorta is a rare condition, and it is usually found in an aorta with diffuse atherosclerotic or aneurysmal changes. Thrombus in a non-atherosclerotic and non-aneurysmal ascending aorta is an even rarer condition. It is usually found during the diagnostic evaluation conducted because of distal embolisations. We report a case of a free-floating thrombus, formed in the ascending thoracic aorta that was morphologically normal, with peripheral embolism that caused acute ischemia of lower limb.

CASE REPORT

A 59 years old man was hospitalised in July 2015, because of acute ischemia of his left leg that started 3 to 4

SAŽETAK

Pojava slobodnoflotirajućeg tromba (SFT) u morfološki zdravoj ascendentnoj aorti veoma je retko stanje, koje se s pojavom savremenih dijagnostičkih metoda znatno češće otkriva. Jedan od glavnih uzroka ovog stanja jesu hiperkoagulabilni poremećaji, a glavne manifestacije su distalne embolizacije.

U ovom radu prikazujemo slučaj pacijenta muškog pola, starosti 59 godina, sa znacima akutne ishemije donjeg ekstremiteta, uzrokovane embolusom iz slobodnog flotirajućeg tromba u ascendentnoj aorti. Embolija je najpre detektovana digitalnom subtrakcionom angiografijom, a kasnije i SFT, kompjuterizovanom tomografijom. Odmah je započeto sa antikoagulantnom terapijom. Nakon hirurškog odstranjenja embolusa iz magistralnih arterija noge, otkriven je hiperkoagulabilni poremećaj. Na kontrolnom CT pregledu nakon 10 dana, nije bilo naznaka trombnih masa u ascendentnoj aorti.

Smatramo da je brzo započinjanje antikoagulantne terapije izuzetno značajno i da je u mnogim slučajevima to optimalan način tretmana ovakvih pacijenata sa SFT. Ipak, još nisu sprovedene studije ni istraživanja koja bi ovo potvrdila, niti postoji algoritam postupaka za tretman ovakvih pacijenata.

Ključne reči: aorta, torakalna; tromboza; antikoagulansi.

hours before the first medical observation. He had no palpable pulses on left popliteal and tibial arteries. The pulse was detected on the left femoral artery in inguinal region, and he also had normal pulses on magistral arteries on the other leg. Motor function of the left foot was unchanged, but the loss of finest tactile sensations was detected. If we look for cardiovascular risk factors, he had a history of 20 years of cigarette smoking and untreated hypercholesterolemia. He had no other history or risk factors for atherosclerotic disease, and also no previous medical history for diabetes mellitus, arrhythmias, ischemic heart disease or stroke. Initial laboratory testing was conducted, with elevated levels of thrombocytes ($520 \times 10^9/L$), total Cholesterol (5.9mmol/l, reference values under 5.2mmol/l), LDL (5.2mmol/l) and fibrinogen

(5.65 g/L, normal: under 3.5g/L). All other biochemical laboratory parameters were within the reference limit. His chest X-ray and electrocardiogram were normal. Medical therapy was started immediately after the hospitalisation. Heparin was administrated in bolus 7000ij, and after that 400ij per kg on 24h (30000 ij. i.v.) in continuous infusion. An emergency DSA (digital subtraction angiography) was conducted (Figure 1).

Oclusion and signs of embolisation of the left popliteal artery were detected. No heavy signs of atherosclerotic lesion were detected on infrarenal aorta, iliac and other magistral arteries on both lower extremities. On the basis of clinical and angiografic findings urgent surgical operation was indicated. Transpopliteal embolectomy through infragenicular approach was performed with Fogarty catheters No II, III and IV. Pedal pulses were

present after the operation was conducted. After the operation low molecular weight heparin was administrated (Enoxaparin 80mg on 12 hours).

In searching for a source of embolisation transthoracic echocardiogram was done and an echogenic lesion of irregular margins was detected in the ascending aorta on that examination. Multislice computed tomography angiography (MSCTA) scan was performed and it showed a mass attached to an anteromedial wall in the ascedent aorta (fig. 2).

The tests for coagulation and autoimmune disorders were conducted as well. Deficit of protein C and protein S (35% and 45%) was detected. On a control MSCTA scan ten days after, we found complete resolution of free-floating thrombus in the ascedent aorta and no signs of distal embolisation (Fig. 3).

Figure 1. DSA images showing non-atherosclerotic abdominal aorta and magistral arteries of lower limbs (A) and occlusion of left popliteal artery-embolus (B).

Figure 2. Thoracic CT angiography demonstrating a FFT in the ascending aorta (white arrow). CT, computed tomography; FFT, free floating thrombus.

Figure 3. Thoracic CT angiography demonstrating a complete resolution of FFT in ascending aorta. CT, computed tomography; FFT, free floating thrombus.

The patient was discharged with vitamin K antagonist (phenocumarol, Farin) for secondary prevention of thromboembolism and with atorvastatin 10mg per day. On this therapy regimen the patient was asymptomatic with no recurrence of FTT and distal embolisation at the six-month follow up.

DISCUSSION

The primary source of distal arterial embolisms are cardiac cavities in more than 85% of cases. In recent years non-cardiac causes have been detected with increasing frequency due to new imaging techniques, such as transesophageal echography (TEE), computed tomography (CT) and magnetic resonance (MR). Among these non-cardiac causes of distal embolisations, the aorta has been reported in up to 5% of cases (1), and most of them are aneurysmal and atherosclerotic lesions, dissections, penetrating ulcers or traumatic lesions of aorta(2).

Hypercoagulability, endothelial injury of vessel wall and stasis of blood flow are suggested by Virchow's triad as main factors that promote a prothrombotic state. In atherosclerosis an aortic thrombus occurs when plaques are eroded and disrupted (3), and in aneurismatic formations there is relatively stagnant and turbulent blood flow that may cause thrombus formation.

Embolisation from non-atherosclerotic, non-aneurysmal thoracic aorta is a very rare event(4). Only few case reports and a small series of patients with thrombus in non-atherosclerotic, non-aneurysmal aorta have been published (5). The causes of thrombus in NANA thoracic aorta are often more systemic. Several factors are mentioned that may increase the risk of formation of thrombus in the ascending aorta, such as underlying malignant disease, hypercoagulable disorders, steroid and estrogen usage and primary endothelial disorders (6). Eguchi and associates (7), reported a case of a patient with protein S deficiency and acute myocardial infarction caused by thrombus in right sinus of Valsalva. Ito et al.(8) reported a case of the women receiving hormone replacing therapy, acute myocardial infarction and aortic thrombus located near the left coronary ostium. Several studies reported cases of aortic thrombus without any known etiology (9-11).

In our case we have a patient with aorta of normal dimensions, and also free of any visible intimal lesions noticed by conducted diagnostic examinations (ultrasonography, MSCT). The prevalence of Protein C and protein S deficiency is about 1 in 1000 patients (12), and in the series of Lapersche et al.(13), 2 of 23 cases of aortic arch thrombosis had protein C deficiency. In one cohort family study, the arterial events were diagnosed in 8% of the 144 subjects with protein C or S deficiencies (14). We speculate that protein C and S deficiency could be the main reason of thrombosis in our case. Additive prothrombotic effects of hypercoagulability, due to the decreased level of

Protein C and S (35% and 45%), and also an increased level of cholesterol and excessive smoking seem most likely to be the cause of thrombus formation in this case.

Free Floating thrombus is usually discovered when the source of distal embolisation is evaluated, although it can be also an incidental finding (15,16, 4,5). The best possible diagnostic strategy for detecting the thrombus in thoracic aorta is synergistic use of transesophageal echosonography (TEE) and CT or MRI (17).

Various treatment modalities have been described in different forms and with variable success including thromboaspiration and surgery (5, 9, 17), thrombolysis (18), anticoagulate therapy alone (13). Exclusion of FFT with endovascular stent graft as a minimally invasive therapeutic option has been recently reported (20,21), however its role has not been fully established yet, regarding the long-term outcome.

With this patient we performed an transpopliteal embolectomy before knowing the exact source of peripheral embolisation. When the MSCT was conducted on the fifth postoperative day and the free floating thrombus was revealed in the ascending aorta, low molecular weight heparin anticoagulation was continued. At the end of the second week after initial operation, control CT was performed, and the complete resolution of thrombus was found.

Therefore, in this case the anticoagulant therapy appeared to be the optimal and the most logical therapeutic strategy for FFT. Ryan S.Turley et al. (22) recently described better results of non-operative management of aortic thrombus in NANA thoracic aorta over the surgical solutions. However, sometimes, despite the anticoagulant therapy, persistence of thrombus is recorded. In those cases, and in the presence of large hypermobile thrombus or in recurrent embolic events some of surgical interventions should be considered.

CONCLUSION

Thrombus in non-atherosclerotic and non-aneurysmal ascending thoracic aorta, represents a rarely diagnosed medical entity. Thrombophilia studies should be performed and malignancy excluded in searching for the underlying reason.

We report an unusual case of peripheral embolisation caused by thrombus in the ascending aorta, contributed by protein C and S deficiency, surgical resolution of embolus and a good response of FFT on anticoagulated therapy. The optimal therapeutic treatment for these patients are still questionable, and mostly depend on the location and morphology of the lesion, the symptoms, general condition of patients and the experience of the medical unit that is dealing with this rare condition. We think that the best first line strategy for FFT is long-term anticoagulation therapy, and surgery (endovascular or open) should be reserved for patients with contraindications to anticoagulation therapy,

for the patients who fail to conservative treatment, and for patients with recurrent embolic events. In cases with mobile thrombus, modality treatment should be considered firmly because of the high risk of embolism. If no exact source of distal embolisation was found after standard exams, we think that MSCT of aorta should always be done.

ABBREVIATIONS

CT (computed tomography),
FFT (free floating thrombus),
MRI (magnetic resonance imaging),
NANA (non atherosclerotic, non aneurysmal).

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