

Nonbacterial thrombotic endocarditis as a surgical complication in a patient status post mitral valve repair with patent foramen ovale closure

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Citation

C Mihos, R Artola, A Torres. *Nonbacterial thrombotic endocarditis as a surgical complication in a patient status post mitral valve repair with patent foramen ovale closure*. The Internet Journal of Cardiology. 2009 Volume 9 Number 1.

Abstract

First described in the 1800s, nonbacterial thrombotic endocarditis (NBTE) was initially dismissed as clinically insignificant, a post-mortem diagnosis or complication seen in patients suffering from malignancies, hypercoagulable states or autoimmune disorders. While occasional studies and reports over the last several decades have proven it to be a major clinical problem with serious sequela, it remains underdiagnosed and underreported. We present the case of a 44 year-old male with recurrent transient ischemic attacks who developed NBTE status post surgical repair of a patent foramen ovale and mitral valvulopathy, illustrating a unique presentation and etiology that has rarely been mentioned or described in medical literature.

CASE PRESENTATION

Patient X was a 44 year-old male with a past medical history of mitral valve regurgitation and patent foramen ovale status post surgical correction of both conditions two months prior to admission. He presented with an approximately two month history of intermittent aphasia, clumsiness and left facial parasthesias that began shortly after his surgery. On the day of admission he noticed increasing difficulty with coordinating his movements, permanent facial parasthesia and new onset ataxia, all on the left side of his body. Past medical history included well-controlled hypertension and family history significant for embolic CVA of unknown origin in his mother. No history of coronary artery disease, malignancy, connective tissue disease or coagulopathies existed in his family. His social history was unremarkable except for rare social alcohol consumption. On physical exam his vitals were: temperature 98.2, pulse 93, respirations 20 and blood pressure 151/94 (subsequent readings were 137/84 and 114/93, without any medical intervention). Neck was supple with no carotid bruits appreciated or thyromegaly. His cardiovascular exam was significant for a II-III/VI blowing holosystolic murmur best heard at the apex and radiating to the axilla. S1 and S2 were present with a regular rate and rhythm – there was no S3, S4, rubs or gallops. Musculoskeletal exam revealed 4/5 muscle strength in left upper and lower extremities without any right-sided deficits. A thorough neurological exam

demonstrated: cranial nerves II-XII intact grossly, with mild left-sided weakness when the patient was prompted to shrug his shoulders to test cranial nerve XI; mild ataxia; intact sensory discrimination; negative Romberg's, pronator drift, rapid alternating hand movements, finger to nose test and heel to shin test. Laboratory values were all within normal limits. EKG showed normal sinus rhythm at 92 bpm and borderline left atrial hypertrophy. In light of the patient's symptoms a CT of the brain was performed and revealed an acute to subacute ischemic infarct of the lateral right frontal lobe in the distribution of the M2-M3 branch of the right middle cerebral artery. This was subsequently confirmed by non-contrast MRI of the brain. The patient was placed on TIA/stroke protocol, including antiplatelet therapy with 325mg aspirin daily, and by the following morning all neurological symptoms had spontaneously resolved. With preliminary blood cultures negative, the patient was started on IV heparin as the presumptive diagnosis was cardioembolic TIA. A transesophageal echocardiogram was performed and revealed an ejection fraction of 65%, negative bubble study for PFO, a small mobile mass on the mitral valve leaflets and moderate mitral valve regurgitation. (Note: post-operatively was said to have mild mitral regurgitation.) Workup for hypercoagulable states, including autoimmune and rheumatological disorders that increase the risk of clotting, was negative. Bacterial endocarditis was unlikely, with five sets of blood cultures revealing no growth and no recent antibiotic use. The possibility of culture-

negative infective endocarditis was entertained, however, the patient was at very low risk of HACEK organism infection and Coxiella and Bartonella IgG/IgM were negative, with no recent travel history or exposure to infected animals. In light of the patient's presentation, symptomatology and workup, a diagnosis of nonbacterial thrombotic endocarditis was made. Reviewing the peri-operative phase of the patient's care and applying the historical evidence from NBTE literature, it is likely that our patient suffered injury to the endothelium of the mitral valve during his surgery that disrupted its structural integrity. The subsequent platelet-fibrin interaction then likely caused the formation of a sterile vegetation on the valve leaflet, which served as the source of the thromboemboli causing his transient ischemic attacks. Heparin therapy was bridged to warfarin with a target INR of 2-3 and recommendation for 3 months of anticoagulation with strict outpatient cardiology follow-up, including repeat echocardiogram to ensure resolution of the thrombus. Due to his age, lack of comorbidities and proper therapy, the patient had no permanent deficits with full resolution of his symptoms on discharge.

DISCUSSION

Nonbacterial thrombotic endocarditis is characterized by an aggregate of fibrin and platelets on a cardiac valve without an inflammatory or bacterial component. Studies have shown that approximately 1.2 to 9.3% of autopsies reveal these valvular thrombi [1]. Variable in size and friable, the thrombi are known to embolize easily and cause cerebral infarctions. It is most commonly associated with malignancies, especially primary lung adenocarcinoma [2,3], as well as hypercoagulable states, connective tissue disorders, autoimmune disorders, tuberculosis, uremia, AIDS, trauma (indwelling pulmonary catheter, CVC), snake bites and late effect of radiation therapy [3]. While the pathogenesis is not completely understood, experts agree that endothelial damage and subsequent exposure to circulating platelets are the most likely and important mechanisms in the development of thrombi. The most commonly cited factors implicated in aiding the development of verrucae are circulating immune complexes,

hypoxia, hypercoagulability and carcinomatosis [3]. Management is difficult with the main focus being to correct the underlying etiology of thrombus formation. Systemic anticoagulation is of utmost importance, with heparin and warfarin as the mainstays of treatment. However, warfarin therapy is contraindicated in patients with malignancy-associated NBTE due to recurrent thromboembolic events – the etiology behind this phenomenon remains unclear [1, 4, 5]. There are no guidelines on length of anticoagulation therapy or surgical intervention, as treatment course is tailored to each individual patient. It is worth noting that surgical therapy is usually not recommended unless the patient is in acute congestive heart failure [3]. The importance in our case lies with the clinical vigilance necessary to identify the development of NBTE in patients with recent cardiac surgery and no history of malignant neoplasms or infection. It is difficult to diagnose and treat and requires a high-index of suspicion. NBTE should be included in the differential diagnoses of all patients presenting with new-onset heart murmurs or neurological symptoms, especially in patients with underlying malignancies and autoimmune disorders or in cases where infectious etiologies have been ruled out. Finally, our case highlights NBTE as an important complication that may arise in patients undergoing cardiac surgery or procedures that require valve manipulation.

References

1. Lopez JA, Ross RS, Fishbein MC. Nonbacterial thrombotic endocarditis: A review. *Am Heart J* 1987; 113:773-84.
2. Guinn GA, Ayala A, Liddicoat J. Clinical and therapeutic considerations in nonbacterial thrombotic endocarditis. *Chest* 1973; 64:26-28.
3. Asopa S, Patel A, Khan OA, Sharma R, Ohri SK. Nonbacterial thrombotic endocarditis. *Eur J Cardiothorac Surg* 2007; 32:696-701.
4. Sack GH Jr, Levin J, Bell WR. Trousseau's syndrome and other manifestations of chronic disseminated coagulopathy in patients with neoplasms: Clinical, pathophysiologic and therapeutic features. *Medicine* 1977; 56:1-37.
5. Rogers LR, Cho ES, Kempin S, Posner JB. Cerebral infarction from non-bacterial thrombotic endocarditis: Clinical and pathological study including the effects of anticoagulation. *Am J Med* 1987; 83:746-756.

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