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Short Communication

Cardiac Manifestations on Anti-Phospholipid Syndrome

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SHORT COMMUNICATION

Antiphospholipid syndrome may present in various ways from cutaneous manifestation, obstetric complications, neurological manifestation, and cardiac manifestation to renal involvement. There are many cardiac complication of anti-phospholipid syndrome, among them are valvular dysfunction, pulmonary hypertension, myocardial infarction, intracardiac thrombi, and ventricular dysfunction [1]. The most common cardiac manifestation is valvular abnormalities ranging from 11.6-32% [2-5].

Cardiac thrombus is an important sequela of APLS. In general, the exact mechanism of intracardiac thrombus formation in APLS is unclear. Julio and Carmen suggested that the endocardial surface might be an important site for thrombus formation in patients with circulating aPL, because these antibodies, in the presence of other hemostatic defects, will abolish the balance between thrombosis and fibrinolysis, and might change the endocardial surface factors so that clot formation is promoted [6]. Coppock, et al. Speculated that an abnormal intracardiac blood flow pattern might contribute to thrombosis [7], and Kaplan, et al. hypothesized that diffuse ventricular dysfunction might predispose to the formation of intracardiac thrombus [8].

Intracardiac thrombosis is a potentially life threatening as it can cause pulmonary and systemic embolic events; however, it is treatable condition. 50% of APLS patients are at risk of developing recurrent embolic events [9], so there must be a delicate balance between this event as well as risk of bleeding from anticoagulation.

Other cardiac manifestations that has been reported to be associated with APLS are valvular lesions, pseudoinfective endocarditis, myocardial infarction, cardiomyopathy and pulmonary hypertension [10].

Valvular lesions has been reported in SLE and APLS patients. Recently; a prospective cross sectional study done showed that positive anticardiolipin, lupus anticoagulant and anti-beta 2 glycoprotein were associated with mitral valve regurgitation; thus the need to perform transthoracic echocardiogram for early detection and management plan even in asymptomatic patients [11]. APLS may also be presents with Libman-Sacks endocarditis which may occur with a prevalence of 11% in SLE. Recently, Kotkar reported on a APLS patient with pulmonary edema with Libman-Sacks endocarditis who required mitral valve replacement [12].

Myocardial infarction occurs in 5% of aPL positive patients [10]. The presence of LA and IgG anticardiolipin antibodies at medium or high titers helps to identify APLS patients at risk for thrombosis. A research done in Tel Aviv University among 214 patients showed that 6.9% with acute myocardial infarction has elevated

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antiphospholipid antibodies. Three out of 7 of them did not have risk factors of Ischaemic heart disease [13]. Young patients with vascular thrombosis and myocardial infarction with normal coronaries should be investigated for APLS [14].

Cardiomyopathy as a primary cause from APLS may be difficult to diagnose in a patient with multi-organ involvement. However, Antiphospholipid antibodies may be a useful marker. Leung et al reported that 4 out of 5 patients with isolated left ventricular dysfunction had +ve aPL. The presence of aPL were significantly associated with left ventricular dysfunction [15].

With regards to the management, aggressive treatment of all risk factors and liberal use of folic acid and vitamin B has been advocated. The use of hydroxychloroquine has been shown to have cardioprotective effect as it has suggested that it has anti-atherogenic effects [16,17]. Generally, the treatment for myocardial infarction, pulmonary hypertension and valvular disease are similar as those without APLS.

For APLS patients with thrombotic events, the duration of treatment is according to the patient's clinical situation and the size, shape, and location of the thrombus. Lifelong warfarin may be an option in the presence of underlying cardiomyopathy which may predispose to further intracardiac thrombosis. A randomized clinical trial done comparing high intensity warfarin (INR 3.0-4.0) versus conventional antithrombotic therapy for the prevention of recurrent thrombosis in patients with antiphospholipid syndrome showed that high intensity warfarin was not superior and is associated with increased risk of minor hemorrhagic complications [18]. A current review states that for patients with definite APLS with first venous thrombotic event, prolonged use of anticoagulation to maintain target INR of 2.0-3.0 is recommended. For those with arterial thrombosis, proposed therapies include: anticoagulation with target INR 3.0-4.0; antiplatelet therapy alone; anticoagulation with target INR 2.0-3.0, or combination of antiplatelet or anticoagulation therapy with target INR 2.0-3.0 [19].

This manuscript illustrates the various cardiac presentations of APLS. The occurrence of thrombotic phenomena, especially in young patients without risk factors, should prompt further investigation towards APLS, i.e. aPL antibodies. Estimation of aPL in cardiological practice assumes considerable importance today.

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