ABSTRACT #9
CLINICAL VIGNETTE/CASE REPORT/CLINICAL REVIEW
CARDIOVASCULAR DISEASE

CONCURRENT ACUTE MYOCARDIAL INFARCTION AND CEREBROVASCULAR ACCIDENT CAUSED BY LIBMAN-SACKS ENDOCARDITIS IN A PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Presented at the **Society of General Internal Medicine Mid-Atlantic Regional Meeting**; Pittsburgh, Pennsylvania, United States held on November 15, 2019.

CASE PRESENTATION: A 36-year-old African American female with a 3-year history of SLE (+ANA/anti-Smith/anti-dsDNA/RNP, low C3/C4, +anticardiolipin IgM/Lupus anticoagulant, +Coombs, +pericarditis, +synovitis, +nephritis) on prednisone and plaquenil but noncompliant with medical therapy, recent lupus flare, and newfound aortic valvular thickening refusing further workup presented with left chest pressure. Patient was hemodynamically stable. On exam, she had a positive systolic murmur. EKG showed acute STEMI. Patient underwent urgent cardiac catheterization which showed single-vessel CAD with RCA total occlusion with evidence of spasm and thrombus. PCI was performed with multiple thrombectomies with red thrombus specimens removed. TTE showed a 2.0 x 1.2 cm mass on the aortic valve, moderate aortic regurgitation, LVEF 40-45%, and severely hypokinetic inferior and inferoseptal walls. Blood cultures were negative. Brain MRI showed 5-6 small acute right insular cortex and parietal cortical infarcts likely cardioembolic with a normal neurological exam. Libman-Sacks endocarditis (LSE) was diagnosed clinically. Aortic valve replacement was initially planned but later aborted due to the need for triple valve surgery given TEE findings of worsening valvular diseases such as severe MR, severe TR, and moderate to severe RV dysfunction. Patient remained stable with systematic anticoagulation, dual antiplatelet, and lupus management. She left against medical advice on hospital day 19 with further management as an outpatient.

DISCUSSION: Libman-Sacks endocarditis (LSE) is a form of noninfectious endocarditis which is characterized by the deposition of sterile platelet and fibrin deposition on the heart valves. It is a rare condition and is often associated with malignancies, SLE, and antiphospholipid syndrome[1]. LSE were present in up to 50% SLE patients on autopsy[2]. In a transesophageal echocardiographic study of valvular disease associated with SLE, valvular vegetations were present in 43% patients initially and 34% at follow up[3]. TEE is superior to TTE for detection of LSE[4]. LSE can be complicated by embolic cerebrovascular disease, peripheral arterial embolism, and superimposed infectious endocarditis[1]. Acute myocardial infarction caused by LSE vegetation in SLE patients was rarely reported. The treatment for LSE includes systematic anticoagulation, surgery, and management of underlying disease[1]. LSE should be strongly suspected when valve dysfunction develops or abnormal cardiac exam presents during the course of SLE. Appropriate and timely anticoagulation needs to be initiated to prevent mortality related to cardiac and CNS embolic events as in our patient[5].

References:
1. Ibrahim, et al. *Libman Sacks Endocarditis*. StatPearls. 2018.
2. Bulkley, et al. “The heart in SLE and the changes induced in it by corticosteroid therapy.” *Am J Med*.1975; 58(2):243-64.
3. Rolden, et al. “An echocardiographic study of valvular heart disease associated with SLE.” *NEJM*. 1996; 335:1424-1430.
4. Roldan, et al. “TTE vs TEE for detection of LSE: RCT study.” *J Rheumatol*. 2008;35(2):224.
5. Lopez, et al. “Nonbacterial thrombotic endocarditis.” *Am Heart J*. 1987; 113(3): 773-84.