Löffler Endocarditis
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Clinical History
A 50-year-old female with essential thrombocytosis, chronic anemia and protein S deficiency presented with shortness of breath. She was in heart failure, with elevated troponin T of 0.15 ng/mL. She had anemia and leukocytosis (15,000/mm³) with markedly increased peripheral eosinophils of 20%. Her increased peripheral mast cells was suggestive of a myeloproliferative disorder and a diagnosis of chronic eosinophilic leukemia was confirmed with bone marrow biopsy. A transthoracic echocardiogram showed an ejection fraction of 58%, with segmental wall motion abnormalities in the septal, anterior and apical territories. There was restrictive mitral inflow pattern on the Doppler recordings. A large echogenic mass was seen adjacent to the mid to distal anterior ventricular wall that raised the possibility of a thrombus or infiltrative mass (Figure 1) and was suggestive of eosinophilic myocarditis or Löffler endocarditis. Further studies were obtained to characterize the left ventricular mass.

Findings
Cardiac CT showed large masses within the left ventricular (LV) cavity that involve multiple coronary territories. The masses were isoattenuating to myocardium and showed no delayed enhancement. (Figure 2) The underlying myocardium was morphologically normal with preserved LV function. This attenuation pattern was suggestive of thrombus in absence of infarction. Cardiac MR showed circumferential T2 hyperintensity and delayed hyperenhancement in the LV endocardium, supportive of an inflammatory and fibrous disease process (Figure 3). Large amount of LV thrombus was seen involving the anteroseptal, anterolateral and inferolateral segments, leaving a tunnel-like communication to residual LV cavity space at the apex (Figure 4). The presence of thrombus and endocardial fibrosis at multiple LV segments, without any evidence of obstructive coronary disease, is suggestive of a diagnosis of Löffler endocarditis.

Discussion
Eosinophilia from hypereosinophilic syndrome, eosinophilic leukemia, infection-related or drug-induced settings can lead to end organ dysfunction from eosinophilic infiltration. Löffler endocarditis is a form of restrictive cardiomyopathy, as a result of eosinophilic penetration of the cardiac myocytes leading to a fibrotic thickening of subendocardium of the heart. Layered thrombus commonly forms due to the damaged endocardium by eosinophilic degranulation. This patient’s symptoms of congestive heart failure were attributed to restrictive physiology due to endomyocardial fibrosis from her systemic eosinophilia. She was started on corticosteroids for her peripheral eosinophilia, and ultimately on therapy for her chronic eosinophilic leukemia.

REFERENCES