A Rare Case of Minocycline Induced Eosinophilic Myocarditis

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BACKGROUND

- Eosinophilic myocarditis (EM) is a rare form of myocarditis characterized by myocardial inflammation associated with elevated levels of blood eosinophils greater than 1.500.

CASE PRESENTATION

- A 36 year old female with newly diagnosed nodular cystic acne on oral minocycline, presented to the emergency department with suspicion of an acute CVA due to asymmetric upper extremity weakness, paresthesias, and acute onset chest pain.
- MRI Brain demonstrated bilateral watershed infarcts involving the anterior cerebral arteries, middle cerebral arteries, and posterior circulation.
- She was found to have hyper eosinophilia on initial labs, for which she was treated with Aspirin, Decadron, and Colchicine due to suspected myocardial involvement on exam and mildly elevated troponin.
- Coronary angiography revealed no significant disease.
- Within 24 hours of treatment, her eosinophil count was undetectable.

RESULTS

- Cardiac MRI (CMR) with contrast demonstrated sub endocardial delayed gadolinium enhancement in a pattern consistent with eosinophilic myocarditis.
- The diagnosis was determined to be drug induced SLE with E, as evidenced by her peripheral eosinophilia, elevated troponin, CMR findings of delayed gadolinium enhancement, and clinical improvement following corticosteroid therapy and withdrawal of the offending agent (minocycline).

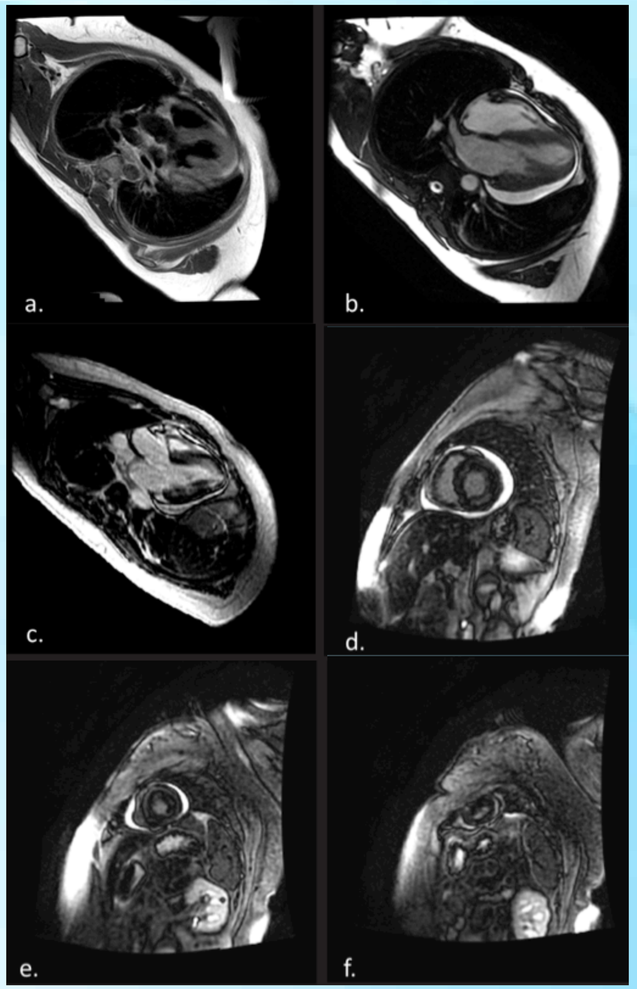

Figure 1: Cardiac MRI for patient with diagnosed eosinophilic myocarditis. (a.) T2-weighted sequence in the 4-chamber view demonstrating myocardial edema. (b) T1-weighted images in the 4-chamber view demonstrating increased global myocardial early gadolinium enhancement. (c) Post-contrast T1-weighted images demonstrating focal late-gadolinium enhancement in non-ischemic pattern. (d-f) T1-weighted images demonstrating subendocardial delayed gadolinium enhancement involving the basal inferior, apical inferior and apical lateral walls.

DISCUSSION

- Eosinophil mediated heart damage may present with varying symptoms including chest pain, arrhythmias, heart failure, or death.
- EM is usually associated with and secondary to an underlying cause such as hypersensitivity to drugs, infections, autoimmune disease, or malignancy.
- The mechanism of myocardial damage in EM is thought to be a delayed type hypersensitivity reaction due to drug haptens binding to myocardial collagen fibrils.
- A definitive diagnosis is made by endomyocardial biopsy, however the utility of invasive testing is limited by the patchy infiltration of eosinophils necessitating multiple tissue samples.
- Noninvasive modalities like echocardiogram and gadolinium enhanced CMR have gained favor in the diagnosis of EM.
- A CMR study is consistent with the presence of myocarditis if at least two of the three following criteria are present: regional or global myocardial signal increase in T2-weighted images, an increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium enhanced T1-weighted images, and at least one focal lesion with nonischemic regional distribution in inversion recovery-prepared late gadolinium enhanced T1-weighted images.
- EM is often associated with subendocardial late gadolinium enhancement, which can be patchy or diffuse.
- A failure to diagnose EM and the delay of therapy may lead to the progression to fulminant myocarditis and irreversible myocardial damage.

CONCLUSION

- This case demonstrates a patient presenting with an acute CVA and heart failure syndrome, and highlights the role of noninvasive imaging in the diagnosis of eosinophilic myocarditis.

Author has nothing to disclose.

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