TOXOPLASMA MYOCARDITIS PRESENTING AS MYOCARDIAL INFARCTION

Sir,

Autopsy profile of immunocompromised patients in India revealed that myocarditis caused by *toxoplasma gondii* is relatively uncommon. Clinically, diagnosis of myocarditis remains a challenge owing to the nonspecific pattern of clinical presentation and lack of universally accepted and standardized diagnostic criteria. The spectrum of clinical presentation is broad and ranges from an asymptomatic state to fulminant condition causing sudden death. Herein we report a case of myocarditis caused by toxoplasma, resembling myocardial infarction in an immunocompromised host.

A 30-year-old male was brought for forensic autopsy. History revealed that he was treated for antero-septal myocardial infarction with cardiogenic shock. In the past 2 months, he was admitted thrice. On first admission, he was complaining of chest pain and was in cardiogenic shock, with his electrocardiogram and enzyme studies showing features of antero-septal myocardial infarction. He was treated for the same and discharged. He had subsequent admission within a fortnight with similar complaints. He was in cardiogenic shock, and electrocardiogram (ECG) revealed Q in V1-V4. He was discharged on the 10th day. Again within a week, he was admitted with pain and vomiting and was in persistent shock.

Autopsy findings showed 100 ml straw-colored pericardial effusion. The coronaries were patent. Both lungs were congested and edematous. Significant pathology was not noted on gross examination of rest of the organs. The blood was reactive for HIV-1 by tridot card method. Microscopic examination of brain showed necrotic areas with histocytes studded with bradyzoites and...
tachyzoites of toxoplasma gondii and vascular proliferation [Figure 1]. Section from myocardium showed edema, inflammatory cell infiltrate comprising of lymphocytes and histocytes. Myocytes showed degeneration and necrosis with focal areas of fibrosis [Figure 2]. Lung showed interstitial pneumonia with edema and cells comprising of intranuclear basophilic inclusion suggestive of cytomegalovirus inclusions. Liver, spleen, kidneys and pancreas showed congestion and vascular proliferation.

In the present case, the person was an immunocompromised host, and reactivation of latent toxoplasma infection is common in such patients. The factors necessary to control the spread of tachyzoites are lacking; cardiac toxoplasmosis occurs during the course of multivisceral dissemination. The present case showed features of toxoplasma myocarditis.

The clinico-pathological correlation suggests that an erroneous initial diagnosis of acute myocardial infarction had been made. The patient was admitted thrice without alleviating the symptoms. The ECG changes of ischemia were not seen subsequently. The coronaries were normal. No histological changes suggestive of ischemic heart disease were found. Nevertheless, presence of lymphocytic inflammatory infiltrate and myofiber necrosis was detected in this case, and it was therefore unlikely that they represented the histological manifestations of myocardial infarction.

Differentiation between toxoplasmic myocarditis and myocarditis relating to other causes can be done by doing endomyocardial biopsy and demonstrating the parasite. The sensitivity of the procedure may be further increased by application of immunohistochemical and molecular biological techniques.

In view of the findings of the present case, it can be suggested that it would be useful to consider toxoplasmic myocarditis in the differential diagnosis of immunocompromised patients presenting with chest pain.

REFERENCES
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