late with sub-acute endocarditis evolving over months making it difficult to establish the exact onset of IE. We presume he developed MA later in the course of the disease and the delayed institution of appropriate antibiotic therapy could have been a factor responsible for the bleed.

The management approach depends on whether the aneurysm had ruptured or not, presence of mass effect and the site. Current recommendations regarding the management of MA have been divided into medical, endovascular and surgical therapies. They are known to resolve with medical therapy as was the case with our patient. The recommended treatment is intravenous antibiotics for 4 to 6 weeks. A repeat angiography is required to confirm resolution. Endovascular coiling has now evolved as a popular modality of treatment option. Surgical interventions are reserved for definitive indications.

Acknowledgments:
Authors thank Dr. V. B. Borade, Dean, Bharati Vidyapeeth Deemed University Medical College & Hospital, Sangli. for permission to publish this manuscript.
Authors Contribution:
SSD was involved in the management of the case. SMD did the cardiac evaluation. Both the authors were involved in the drafting of the paper. SSD will act as the guarantor.

Source of Funding:
None
Competing Interests:
None

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E-published: 1st February 2013
DOI No. 110.7199/ped.oncall.2013.8

Letter to the Editor (Viewer’s Choice)

CONCOMITANT RHEUMATIC FEVER AND DILATED CARDIOMYOPATHY: A CASE REPORT

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**Key words:** Dilated Cardiomyopathy, Acute Rheumatic Fever.

An 8 years old boy presented with pain and swelling in his knees for two weeks. He was advised ibuprofen. X-ray knees showed no abnormality. On admission, he complained of new onset of right ankle joint pain, fever, breathlessness, palpitation on exertion. Examination revealed temperature of 100.8°F, tender swollen ankle joints (right > left) and edema of the feet. On general examination, he had basal crepitations in both lungs, tender hepatomegaly and pansystolic murmur in mitral area. Investigations showed hemoglobin of 12.9g/dl, white blood cell count of 11,500 cells/cumm, neutrophils 9600 cells/cumm, erythrocyte sedimentation rate of 74mm at end of 1 hour and C-reactive protein of 90mg/l. Chest x-ray showed cardiomegaly with a cardiothoracic ratio of 0.59. Echocardiography revealed dilated cardiomyopathy (DCM) with left vertical dysfunction (ejection fraction of 39%) and global hypokinesia. In addition, there was mildly dilated left atrium. Left ventricular end diastolic dimension was 4.8. Pharyngeal throat swab culture showed streptococcus pyogenus. Anti-streptolysin antibody was raised, peaking at 6 weeks after onset.
and declining thereafter. The highest titer reached were antistreptolysin O 2000 unit, DnasB 1900 unit, and antihyaluronidase 1024 unit. This indicates recent streptococcal infection along with carditis, polyarthritis, fever and positive acute phase reactants suggestive of rheumatic fever. Echocardiography was suggestive of DCM. Patient was treated with oxygen, prednisolone (2 mg/kg/day for six weeks then gradually tapering for another 6 weeks), Benzyl penicillin (15 lac unit in four divided doses for 10 days), furosemide, enalapril and carnitine. Later on patient was put on benzathine penicillin prophylaxis, enalapril and carnitine were continued. After five months of treatment the patient remained well.

DCM is characterized by dilatation and impaired contraction of left ventricle or both ventricles. (1) In the present case DCM was associated with acute rheumatic fever (ARF). Such association is infrequently reported previously in the world literature though it has been reported with viral myocarditis. (2,3) The short term prognosis of myocarditis is usually good, but varies widely by cause. Those patients who initially recover might develop recurrent dilated cardiomyopathy and heart failure. (2) However in end stage dilated cardiomyopathy with clinically unsuspected acute rheumatic carditis, it can have a fatal outcome. (3) The diagnosis of isolated rheumatic fever was considered in our patient because of finding of very high anti-streptolysin O titre and lack of evidence of either viral disease, infection with group C and G streptococci or other cause of spuriously high titer of antistreptolysin O. (4)

Thus to conclude, the association between dilated cardiomyopathy and rheumatic fever may be more prevalent than recognized and further study into the association and the responsible pathogenic mechanism is warranted.

Contributors: KA and DPB were involved in patient management and review of literature. KA supervised the management and drafted the manuscript. KA should act as guarantor.

Funding - none

Competing interest - none

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