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## **Embolic Stroke in Myocarditis**

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Almost all embolic strokes that occur in pediatric population beyond the perinatal period are the result of either congenital or acquired heart disease. Among acquired heart diseases, rheumatic heart disease is known to cause embolization to the central nervous system. However, acute myocarditis of non-rheumatic etiology with embolic stroke is a previously unrecognized association. Hence, we report a case of acute myocarditis of non-rheumatic etiology with embolization to the central nervous system.

## Case Report \*

A 5-year-old boy presented with complaints of right sided weakness of thirty six hours and inability to speak for twenty four hours duration. The past history was non-contributory except for a mild febrile illness seven days earlier. The child was appropriately immunized for age. The developmental history was normal.

On examination, the child was afebrile

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Received for publication: June 21, 1992; Accepted: December 24, 1992 but irritable. The pulse rate was 150/min and all the peripheral pulses were palpable. The blood pressure was 110/70 mm Hg and 120/80 mm Hg in left upper and lower limb, respectively. The jugular venous pressure was elevated 5 cm above the sternal angle. Cardiovascular examination revealed sinus tachycardia with S3 gallop and cardiomegaly. The liver was tender and enlarged 6 cm below the costal margin. Neurological examination showed right sided hemiparesis, ipsilateral upper motor neuron facial nerve pareses and motor aphasia; meningeal signs were absent. The musculoskeletal system was normal.

Investigations showed a hemoglobin of 12 g/dl, total leucocyte count of 9,600/mm<sup>3</sup>, ESR was 4 mm/h. Serum sodium was 133 mEq/L, postassium was 4.4 mEq/L, creatinine was 0.82 mg/dl. LDH was 784 $\mu$ /L, creatinine kinase was 32  $\mu$ /L. Blood culture was negative and ASO titre was 60 Todd U. ECG revealed sinus rhythm, heart rate of 150/min and left bundle branch block with mild prolongation of P-R interval.

Two dimensional echocardiogram showed a dilated globular left ventricle with an ejection fraction of 20%, the valves were normal with mild secondary mild regurgitation and there was a small clot in left atrium. Computerized tomography of brain showed left parietal, parieto-occipital and occipital hyperemic infarction due to embolism (Fig.).

A diagnosis of acute myocarditis was made. The child was treated with digitalis, intravenous furosemide and oral captopril. After the availability of echocardiogram report, heparin was used in a dosage of  $4500 \,\mu$ /day and aspirin in a dosage of  $150 \,\mathrm{mg/day}$ . Ten days later, heparin was gradually stopped and warfarin (1.25 mg/day in a single dose) was started, to main-



Fig. Contrast enhanced CT scan showing enhancing lesions in left parietal and parieto occipital and occipital regions.

tain the PT at 30 sec. Physiotherapy was initiated.

The patient's cardiac failure and hemiparesis improved over the next two weeks. The child was discharged on digitalis, furosemide and oral anticoagulants (warfarin and aspirin). On follow-up over 6 months the hemiparesis disappeared and the child was fast regaining speech. The patient's echocardiogram showed resolution of the left atrial thrombus and increase in the left ventricle ejection fraction to 52%.

### Discussion

In our patient, rheumatic etiology of the myocarditis was ruled out as the Jones criteria were not fulfilled. The acute presentation and prompt response to therapy with an improvement in the left ventricular ejection fraction and decrease in cardiac size on serial echocardiograms ruled out a chronic congestive cardiomyopathy. The diagnosis of viral myocarditis was made on clinical criteria. While endomyocardial biopsy is considered to be essential for diagnosis of viral myocarditis, the procedure is unreliable and subject to wide interobserver variation(1).

Hereditary cardiomyopathies may be associated with thrombus formation in the heart(2). In cases with acute myocarditis, stagnation of blood flow can give rise to thrombus formation in the heart and subembolus formation. Mural thrombi have been described in the left ventricles of some patients with myocarditis, and small emboli are occasionally seen in the coronary and cerebral vessels(3). Our patient showed a thrombus in the left atrium and evidence of an infarct in the left parietal, parieto occipital and occipital regions. Occurrence of the infarct was possibly related to the thrombus in the left atrium.

The drug therapy for childhood stroke includes platelet antagonists and oral anticoagulants (warfarin)(4). Our patient has improved dramatically while on low dose aspirin and warfarin and has remained free of the side effects of these drugs.

We report this case to stress that acute myocarditis can no longer be considered a benign condition and that embolic stroke can be one of its presentations in the acute phase.

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# Demonstration of Brain Lesions in Tuberous Sclerosis by Magnetic Resonance Imaging

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Tuberous sclerosis is an inherited autosomal dominant disease, clinically characterized by adenoma sebaceum, seizures and mental retardation. These features though suggestive are not specific of the disease unless accompanied by characteris-

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