

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 inter-observer 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 subsequent embolus 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 anti-coagulants (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.

## REFERENCES

1. Shanes JG, Ghali J, Billingham ME. Inter-observer variability in the pathologic interpretation of endomyocardial biopsy results. *Circulation* 1987, 75: 401-405.
2. Furlan AJ, Cracium AR, Raju NR. Cerebrovascular complications associated with idiopathic hypertrophic subaortic stenosis. *Stroke* 1984, 15: 282-284.

3. Friedman AR. Myocarditis. *In: Principles and Practice of Pediatrics*, Eds Oski FA, De Angelis CD, Feigin RD, Warshan IB. Philadelphia, J.B. Lippincott Co, 1990, pp 1459-1467.
4. Pavlakis SJ, Gould RJ, Zito JL. Stroke in Children. *In: Advances in Pediatrics*. Ed Barness AL. St. Louis, Mosby-Year Book Inc, 1991, 38: 151-179.

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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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tic brain involvement lesions demonstrated by computed tomography or more recently by magnetic resonance imaging (MRI)(1,2). MRI has surpassed or is at least equal to computed tomography in most of the central nervous system diseases and tuberous sclerosis should be no exception. We describe MRI findings in seven patients of tuberous sclerosis of pediatric age group.

### Material and Methods

Seven patients of tuberous sclerosis diagnosed by MRI at our centre over last four years formed the basis of the study. There were four females and three males with age ranging between 3 to 12 years. All the patients had presented with generalized seizures, three had mental retardation while adenoma sebaceum were seen in six patients.

All examinations were performed on 1.5 Tesla Scanner Magnetom (Siemens) having superconducting magnet. The scans were obtained in supine position in head coil. Coronal, axial and sagittal scans were obtained in all cases with slice thickness of 5 mm and interslice gap of 0-50%. Spin echo sequence with a repetition time (TR) or 700 msec and an echo time (TE) of 22 msec was used for T1 weighted images and TR of 2000 msec and TE of 22/90 msec was used to obtain T2 weighted images. Images were reconstructed on 256 × 256 matrix.

### Results

MRI revealed subependymal nodules in all, tubers in six, dilated lateral ventricles due to foramen of Monro block in 1 and calcification in cerebellar hemisphere in two cases.

Subependymal nodules (*Figs. 1A & B*) were seen in the form of small soft tissue

masses either projecting into the lateral ventricle or embedded in the caudate nucleus. In short TR images (T1 weighted images), they were iso or mildly hyperintense while in long TR images they appeared low intense.

Long TR images (T2 weighted images) demonstrated tubers in six of the seven patients as areas of hyperintensity (*Fig. 2*). On short TR images they could be appreciated only in two patients as zone of relatively low signal occupying the inner core of a gyrus. Tubers were localized to supratentorial compartment in four patients, while in two cases they could be seen in both supra and infra tentorial compartment. In two patients calcified lesions in cerebellum were seen (*Fig. 3*).

Dilated lateral ventricles due to foramen of Monro block by a large variegated mass with areas of calcification was seen in one patient. Biopsy revealed it to be a tuber.

### Discussion

For definite diagnosis of tuberous sclerosis, it is essential to demonstrate changes of brain involvement at CT or more recently MR(2,3). Calcified subependymal nodules are readily detected and have been the single most specific lesion seen on CT with parenchymal areas of hyperintensity, peripheral calcifications, giant cell astrocytoma and ventricular dilatation being the other important features(4).

McMurdo *et al.*(2) in an analysis of 15 patients of tuberous sclerosis examined by 0.35-0.5 Tesla MR scanner found lesions of prolonged T2 in cortical and subcortical regions in all and subependymal nodules in 11. Similar findings were observed by Roach *et al.*(3), in addition they also demonstrated cerebellar lesions. Nixon *et al.*(5) found subependymal nodules in 17 of 19

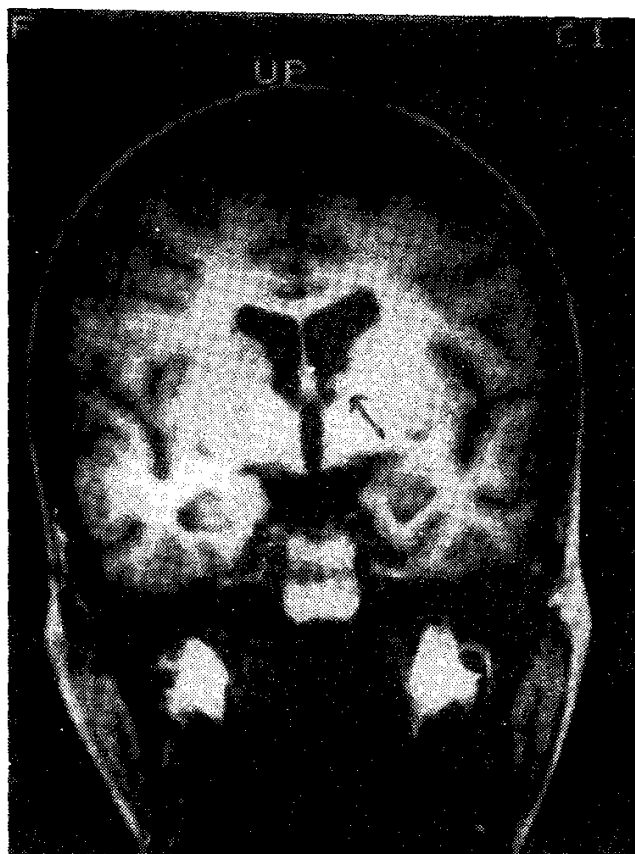
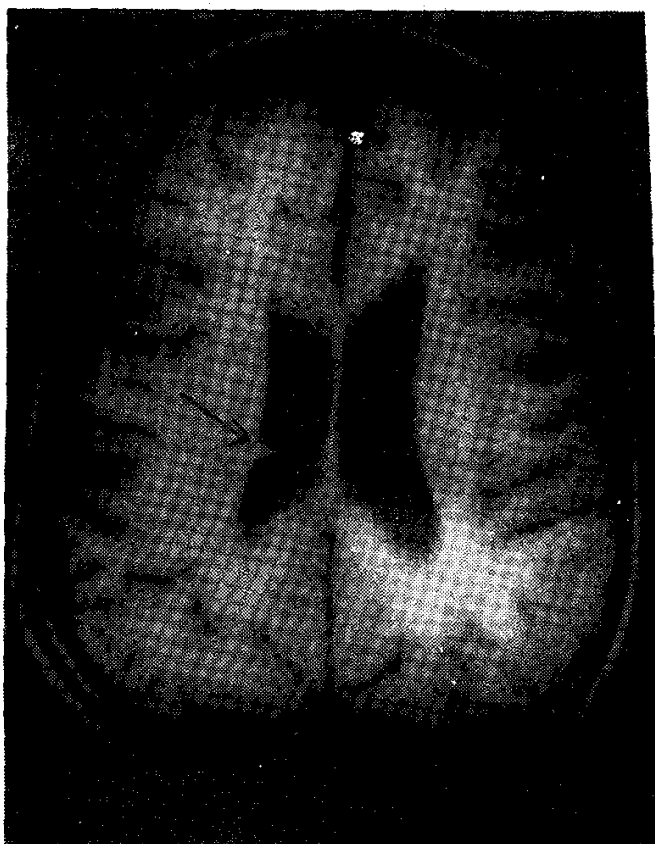


Fig. 1A & 1B. Axial (1A) and coronal (1B) T1 weighted images (TE/TR- 22/700 msec) showing subependymal nodules (arrows) as mildly hyperintense soft tissue masses.

clinically diagnosed patients of tuberous sclerosis. Cortical tubers were detected in 17, cerebellar in 3 and deep periventricular white matter lesions in 14 patients. They were of the opinion that some of the deep periventricular white matter lesions might have resulted from sectioning across the base of an affected gyrus, thus producing a false appearance of separation of lesions, others probably represent the white matter nodule or cluster of abnormal giant cell.

Cortical tubers are pathologically considered to be hamartomas(6,7). On MR imaging they show prolonged T1 and T2. Nixon *et al.*(5) opined tubers presumably contain more unbound water than normal brain does, but it is uncertain whether this

is because of greater production or greater retention of free water or interstitial fluid within tubers. There is no apparent breakdown of the blood brain barrier within cortical tubers as is evidenced by their lack of enhancement by contrast on CT.

We strongly feel that MR is a sensitive imaging modality in detecting brain lesion in patients of tuberous sclerosis especially in demonstrating tubers. The only limitation seems to be in picking up small calcifications and CT may be more helpful in detecting intracranial calcifications than MR. MR can be of real help in screening family members of a patient of tuberous sclerosis. However, its specificity is less apparent. Certainly a subependymal nodule is a reli-

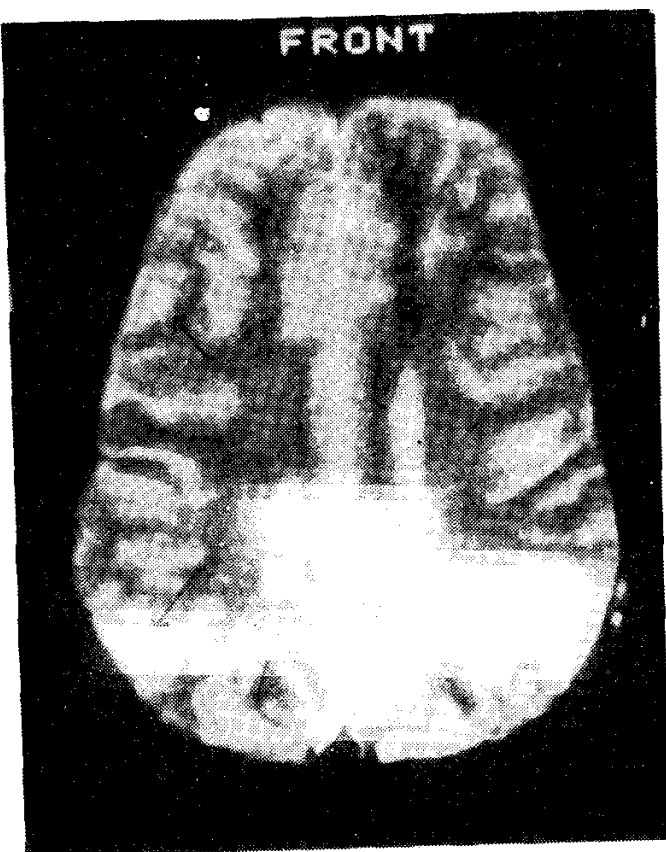


Fig. 2. Axial T2 weighted images (TE/TR - 90/2000 msec) showing hyperintense lesions occupying the inner core of gyri in the parietal lobe (arrow) --Tubers.



Fig. 3. T1 weighted axial scans showing calcified lesions in the cerebellar hemisphere.

able sign if identified confidently as a soft tissue mass projecting into the lateral ventricle to make a diagnose of tuberous sclerosis in a clinically suspected case

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#### REFERENCES

1. Gomez MR. Tuberous Sclerosis, 2nd edn. New York, Raven Press, 1988.
2. McMurdo SK J, Moore SG, Brant-Zawadzki M, *et al.* MR imaging of intracranial tuberous sclerosis. *AJNR* 1987, 8: 77-82.
3. Roach ES, Williams DP, Laster DW. Mag-

netic resonance imaging in tuberous sclerosis. *Arch Neurol* 1987, 44: 301-303.

4. Critchley M, Earl CJC. Tuberous sclerosis and allied conditions. *Brain* 1932, 55: 311-346.
5. Nixon RJ, Houser OW, Gomez MR, Okazaki H. Cerebral tuberous sclerosis: MR imaging. *Radiology* 1989, 170: 869-873.
6. Bender BL, Yunis EJ. The pathology of tuberous sclerosis. *Pathol Ann.* 1982, 17: 339-382.
7. Nardelli E, De Benedictis G, La Stilla G, Nicolardi G. Tuberous sclerosis: A neuropathological and immunohistochemical study. *Clin Neuropathol* 1986, 5: 261-266.