considered by some to be interstitial edema and by others to be a mucopolysaccharide due to affinity for metachromatic stain. The dermal appendages are normal but owing to the thickening of the dermis appear to lie more superficially(5,6). In the mid and upper dermis some workers have found a mild perivascular inflammatory infiltrate. An electron microscopic study showed that the collagen fibrils of diseased skin were thinner than normal and showed a tendency to fractures and there was an excess of interfibrillary cement(5).

There are numerous theories about the etiopathogenesis of this condition. These include an immune mechanism, direct action of a bacterial toxin, and effect of adrenal steroids released due to infection(5). Circulating paraproteins have been found in patients with scleredema. Ohta *et al.* put forward the theory that these may enhance the synthesis of extracellular macromolecules by dermal fibroblasts, thus providing a mechanism for dermal fibrosis(7).

Although there are no controlled trials, therapy has been largely ineffective in this disorder. Bradford *et al.*(8) observed that children receiving steroids seemed to recover relatively quickly. This may be the cause for the rapid improvement in the present case also. Therapy is, however, not generally recommended because of the benign and self limited nature of the disorder.

REFERENCES

1. Heilbron B, Saxe N, Derm FF. Scleredema in an infant. Arch Dermatol 1986, 122: 1417-1419.
2. Greenberg LM, Geppert C, Worthen HG, Good RA. Scleredema 'adultorum' in children. Pediatrics 1963, 32: 1044-1054.
3. Deorari AK, Arora NK, Singhal P, Lata M, Kalra V. Scleredema in a child. Indian Pediatr 1986, 23: 560-563.
4. Nanda S, Tewari AD, Dhatt PS. Scleredema. Indian Pediatr 1987, 24: 1056-1057.
5. Carrington PR, Sanusi ID, Winder PR, *et al*. Scleredema adultorum. Int J Dermatol 1984, 23: 514-522.
6. Robinow M. Scleredema adultorum: A children's disease. Am J Dis Child 1963, 105: 265-274.
7. Ohta A, Litto J, Akarinen AI, *et al*. Paraproteinemia in patients with scleredema. J Am Acad Dermatol 1987, 16: 96-107.
8. Braford WB, Cook CD, Vawter GF, Berenberg W. Scleredema of childhood. J Pediatr 1966, 68: 391-399.

Intracranial Tuberculoma with Cutaneous Miliary Tuberculosis

V. Puri
R.K. Gupta
V. Malhotra

Tuberculomas constitute upto 15% of space occupying intracranial lesions in India(1). Tuberculomas result from heamtogenous spread from a primary

From the Departments of Neurology and Pathology, G.B. Pant Hospital, New Delhi 110 002, and NMR Research Center, Institute of Nuclear Medicine and Allied Sciences, Delhi.

Reprint requests: Dr. Vinod Puri, Associate Professor, Department of Neurology, G.B. Pant Hospital, New Delhi 110 002.

Received for publication December 9, 1990; Accepted March 11, 1991

focus, evident or dormant elsewhere in the body. The MR (magnetic resonance) appearances of intracranial tuberculoma are specific(2,3) in contrast to nonspecific CT scan findings(4,5). However both serial MR(3) and CT scan(5-7) allow an objective assessment of the effect of medical treatment on both the tuberculoma and the surrounding edema. Acute miliary tuberculosis of the skin is quite rare(8-10) and results from hematogenous spread. Both, intracranial tuberculoma and cutaneous miliary tuberculosis can occur together; however, we have not come across any such report in the literature, since the CT era. The present case is unique with the presence of both intracranial tuberculomas and acute miliary tuberculosis of the skin.

Case Report

A 9-year-old girl presented with simple partial motor seizures of left side (with no march) and followed by post ictal weakness of 7 days duration. During some attacks she had loss of consciousness. Some seizures were only sensory (numbness) of left side. She had never experienced any seizure prior to this onset. She had low grade fever of 3 weeks duration and already received several oral antibiotics (erythromycin, chloromycetin, trimethoprim). There was no family history of seizures, any other neurological illness or tuberculosis. Examination revealed a well nourished, average built child with multiple subcutaneous nodules without exanthematous reaction of the overlying skin. She had no lymphadenopathy or visceromegaly. The detailed neurological examination including fundii was not contributory except the transitory post ictal weakness (power 3/5) of left upper and lower limbs. Her hemogram, blood biochemical profile for sugar, urea, calcium, phosphorus, liver function tests, electrolytes, urinalysis was normal. Culture of urine and blood grew no organism. The roentgenogram of chest, skull, arms, legs was normal. CT scan head showed an enhancing ring lesion with perifocal edema in right parietal area, a disc lesion in left occipital and hypodensity (edema) in right occipital region. Her CT thigh and legs had multiple well defined subcutaneous hyperdense lesions while some of these had in addition hypodensity in the centre. On T2 weighted MR imaging of head (*Fig. 1*) she had multiple hypointense lesions with hyperintense center and surrounding hyperintensity of perifocal edema, while the T1 weighted imaging these lesions had hypo to isointensity. T2 weighted MR of the forearms (*Fig. 2*) showed multiple subcutaneous hyperintense lesions which were hypointense on T1 images. On biopsy of the skin nodule, pus like material came out of the nodule which later on grew tubercle bacilli on culture. Histopathological examination showed caseating tuberculous granulomas with epitheloid and Langhen's giant cells. CSF examination was normal and negative on ELISA for cysticercosis. Mantoux test was negative.

She was put on antitubercular treatment (rifampicin + INH + pyrazinamide) alongwith carbamazepine. Rifapicin had to be withdrawn (due to intolerance) and replaced with streptomycin. Initially she was intolerant to INH and pyrazinamide (alone or together) but later on it improved. Streptomycin was withdrawn after 90 injections while INH and pyrazinamide were continued. About 3 weeks after on regular antitubercular treatment the subcutaneous nodules lessened numerically to disappear after another two months. The intracranial lesions also decreased in size to disappear

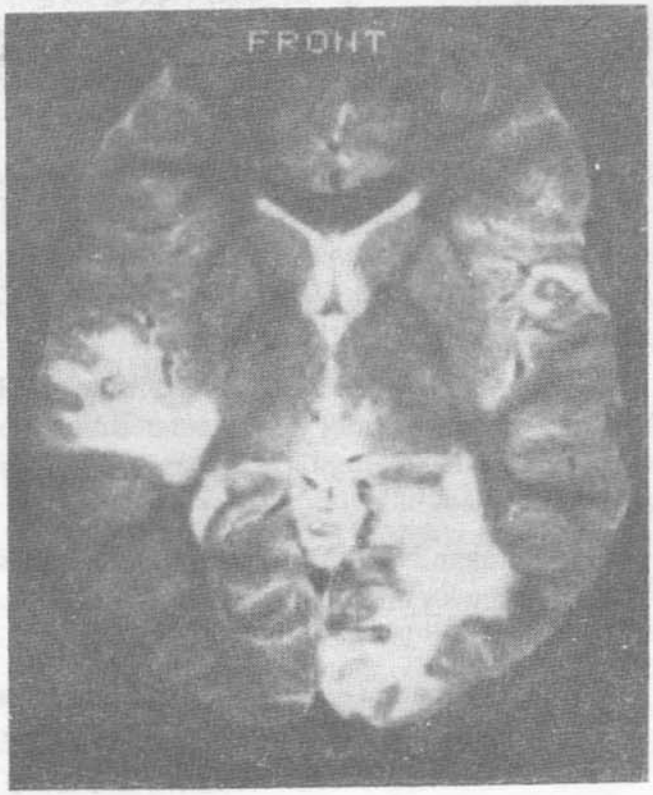

Fig. 1(a). T2 weighted axial image shows a hypointense lesion with hyperintense central area, surrounded by hyperintensity of edema, in both hemispheres.

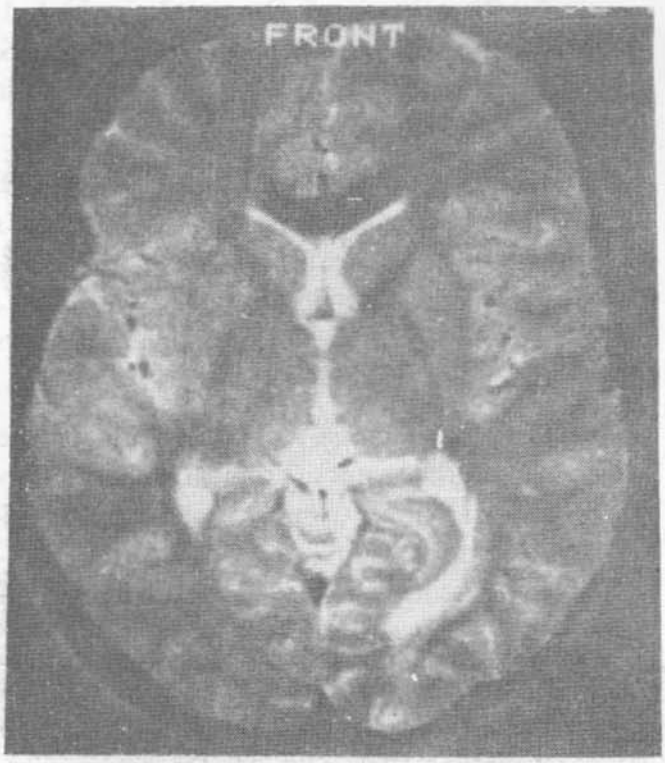

Fig. 1(b). Follow up T2 weighted image after 4 months of antitubercular treatment showing regression of the lesions.

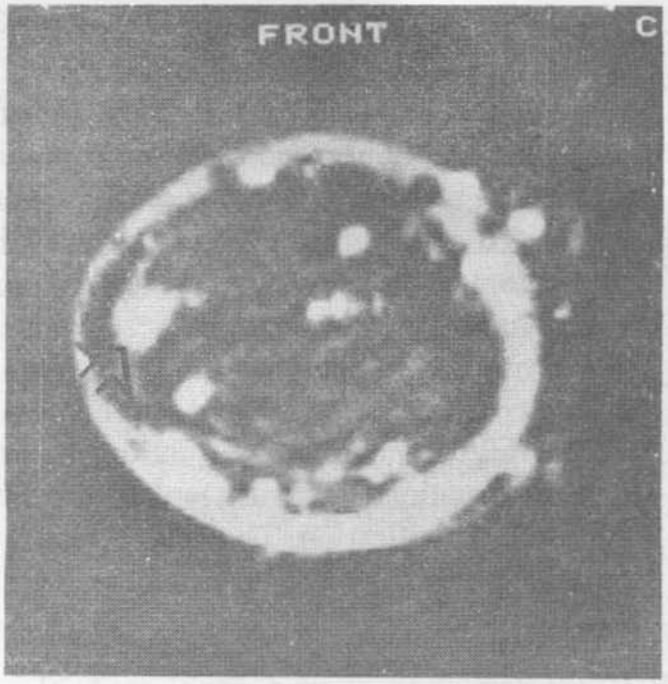

Fig. 2. T2 weighted axial image of left forearm through the nodule shows hyperintense lesions (open arrow) which were hypointense on T1 image. Rest of the bright spots in the muscles are flow artifacts.

on follow up CT and MRI scans. She had seizures twice during her follow up and required an increase of carbamazepine dosage.

Discussion

Tuberculosis of skin may be localized progressive or hematogenous form(9,10). The present case had multiple subcutaneous tuberculous nodules without any overlying exanthematous reaction, suggestive of hematogenous miliary spread of tuberculosis from the primary site. Miliary tuberculosis of skin usually occur alongwith tuberculous meningitis or generalised miliary tuberculosis or a post primary manifestation provoked by immunosuppresent disorder or illness. No such association was recorded in the present case; however she had also intracranial tuberculomas. Mantoux (tuberculin) test was negative as had

also been reported in miliary hematogenous tuberculosis of the skin(8-10).

Prior to the advent of CT, diagnosis of intracranial tuberculoma was usually confirmed at either surgery or autopsy. Computed tomography of intracranial tuberculomas show a variety of lesions corresponding to the pathological events of evolving granuloma(11-16). The edema and necrosis appear as low attenuation area, while granuloma formation show high attenuation, contrast enhancement and finally calcification. The central necrosis and peripheral organization produce ring appearances.

On MR, the caseous necrosis, liquefaction have bright signal on T2 weighted images and hypo to intermediate intensity on T1 images. The granulation area of tuberculoma is hypointense on T2 images and iso intense on T1 images. The surrounding edema, a sign considered towards immaturity of tuberculoma is hyperintense on T2 images and hypointense with T1 images(2,3).

The MR appearances of intracranial lesions in the present case were typical of tuberculoma(2,3). The cutaneous lesions were hyperintense on T2 images and hypointense with T1 images. This could be due to the perhaps presence of more caseous necrosis and liquefaction in the cutaneous lesions.

The development of caseous necrosis is considered an indication for surgical intervention by some(13,17) and not so by others(3). Serial MR showed disappearance of all cutaneous and intracranial lesions on antitubercular therapy.

REFERENCES

1. Bhatia S, Tandon PN. Solitary microlesions in CT. A clinical study and follow up. Neurol India 1988, 36: 139-150.
2. Gupta RK, Jena A, Sharma A, *et al*. MR imaging of intracranial tuberculomas. J Compt Assist Tomogr 1988, 12: 280-285.
3. Gupta RK, Jena A, Singh AK, *et al*. Role of magnetic resonance (MR) in the diagnosis and management of intracranial tuberculomas. Clin Radiol 1990, 41: 120-127.
4. Mayers MM, Kaufman DM, Miller MH. Recent cases of intracranial tuberculomas. Neurology (Minneap) 1978, 28: 256-260.
5. Rahman NU. Intracranial tuberculomas: diagnosis and management. Acta Neurochirugica 1987, 88: 109-115.
6. Hildebrandt G, Angoli AL. Differential diagnosis and therapy of cerebral tuberculomas. J Neurol 1982, 228: 201-208.
7. Harder E, Al-Kawi KZ Carney P. Intracranial tuberculoma: conservative management. Am J Med 1983, 74: 570-576.
8. Wilkinson DS. Tuberculosis of the skin. *In:* Text Book of Dermatology, Vol. 1, 3rd edn. Eds. Rook A, Wilkinson DS, Ebling FJG. Oxford, Blackwell Scientific publis. Vol. I, 1979, pp 677-699.
9. Domonkos AN, Arnold HL, Odom RB. Mycobacterial diseases. *In:* Andrew's Diseases of the Skin-Clinical Dermatology. Eds. Domonkos AN, Arnold HL, Odom RB. Philadelphia, WB Saunders Co. 1982, pp 404-420.
10. Gonzalez-ochoa A. Tuberculosis of the skin in the tropics. Tropical bacterial dermatoses II. *In:* Clinical Trophical Dermatology. Ed. Canizares O. Oxford, Blackwell scientific Publications. 1975, pp 134-141.
11. Price HI, Danziger A. Computed tomography in cranial tuberculosis. Amer J Roentgen 1978, 130: 769-771.
12. Welchman JM. Computerized tomography of intracranial tuberculomata. Clin Radiol 1979, 30: 567-573.

13. Bhargava S, Tandon PN. Intracranial tuberculomas: A CT study. Br J Radiol 1980: 53: 935-945.

14. Whelan MA, Stern J. Intracranial tuberculoma. Radiology 1981, 138: 75-81.

15. Vengsarkar US, Pisipaty RP, Parekh B, Panchal VG, Shetty MN. Intracranial tuberculoma and the CT scan. J Neurosurg 1986, 64: 568-574.

16. Draouat S, Abdenabi B, Ghanem M, Bourjat P. Computed tomography of cerebral tuberculoma. J Comput Assist Tomogr 1987, 11: 594-597.

17. Pandya SK, Desai AD, Dastur HM. Caseative liquefaction within brain stem tuberculoma under drug therapy with simultaneous regression of cerebral tuberculomas. Neurology India 1982, 30: 121.

Cranio-Cerebral Neuroblastoma

R.V. Phadke
V.K. Jain
D.N. Srivastava
P. Tandon
R.B. Gujral

Among the malignant tumors of childhood, neuroblastoma either from adrenal medulla or sympathetic chain with cranial metastases is a frequent occurrence. Primary intracerebral neuroblastoma is, however, very rare and presents some interesting features. Earlier workers have documented radiological characteristics of primary and metastatic neuroblastoma(1,2).

From the Departments of Radiodiagnosis, Neurosurgery and Pathology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow 226 001.

Reprint requests: Dr. Rajendra V. Phadke, Associate Professor, Department of Radiodiagnosis, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow 226 001.

Received for publication November 30, 1990; Accepted March 14, 1991

Case Reports

Case 1: A 10-year-old male patient presented with recent onset paraplegia, proptosis and swellings on the head of 4 months duration. Myelography revealed block at D 10 with patchy sclerosis of vertebral bodies. Plain films of skull showed sutural widening and sunburst periosteal reaction on left parietal bone. Calcification was noted in plain films of abdomen in right adrenal region. Ultrasound revealed right adrenal mass lesion and secondaries in liver. CT of skull showed extraconal left orbital mass with involvement of greater wing of sphenoid and extracranial soft tissue swelling (*Fig. 1*). All these soft tissue masses enhanced on contrast CT. Liver biopsy revealed metastatic neuroblastoma.

Case 2: A 6-year-old male child presented with one month history of headache, vomiting and left lower limb weakness. Bilateral papilledema and Grade II power in left lower limb was detected on examination. CT showed large isodense right parafalcial parietal mass lesion with slight peritumoral edema and heterogenous contrast enhancement and cystic component (*Fig. 2*). Plain *X*-ray, sonography of abdomen and chest *X*-rays were normal. Total removal of the neoplasm along with its attachment to falx, was done by right parietal craniotomy. Histopathology showed a highly cellular undifferentiated tumor with multifocal pseudocystic degeneration with necrosis and calcification. Tumour cells showed hyperchromatic densely speckled nuclei. In view of the