Matters arising

Rapid development of basal ganglia hyperdensity caused by anoxia

Sir: We read with interest the report of Iwasaki, et al 1 which described the rapid development of hyperdensity of the basal ganglia on CT scan. The authors ascribed this appearance to calcification occurring in the presence of anoxia and metabolic acidosis. However, our recent experience suggests that this appearance might be caused by diffuse ganglionic haemorrhage.

A 23 year old male with insulin dependent diabetes mellitus of 8 years duration, presented to the emergency department with diabetic ketoacidosis (blood glucose 68 mmol/l, sodium 117 mmol/l, potassium 7.6 mmol/l, bicarbonate 3.5 mmol/l, arterial pH 6.675, Paco $^2$ 2.44 kPa). He was alert with no neurological deficit. Treatment was begun with intravenous insulin and 0.9% saline. Thirty minutes later he had a cardiorespiratory arrest. He had a series of low output cardiac dysrhythmias. When sinus rhythm was re-established after 20 minutes he was unresponsive and it was assumed that some anoxic cerebral damage had occurred. Further cardiac dysrhythmias requiring multiple DC cardioversion occurred and a dopamine infusion was required to keep the diastolic blood pressure above 40 mmHg once he was back in sinus rhythm. The metabolic abnormalities were corrected, the potassium being normal within 4 hours, the blood gases within eight hours and the sodium within 24 hours, by which time the blood glucose was in the range 15–20 mmol/l. He was paralysed and ventilated electively for 24 hours.

Two days later he was alert and orientated, though with slow mentation. At this time he had no focal neurological deficit. During the following evening the insulin requirement increased with blood glucose levels up to 28.5 mmol/l, whilst the arterial pH dropped to 7.18. This was corrected by an increase in the intravenous fluids and insulin. The next morning there had been a notable change in his neurological condition. He lay with his eyes open, moaning but not making any coherent sounds. His back was arched and periodic flexor spasms of all limbs were noted though the tendon reflexes were not particularly brisk and the plantar responses remained flexor. He had a divergent strabismus.

CT 4 days later showed diffuse punctate hyperdensity in the basal ganglia bilaterally (fig. a). Initially, this was considered to be due to calcification though since there were no scans prior to this event haemorrhage could not be ruled out. The neurological features remained unchanged and six weeks later other CT now showed bilateral hypodensities in the same area of the basal ganglia (fig. b). In view of this it was concluded that diffuse petechial haemorrhage rather than calcification had been responsible for the appearance on the initial scan.

The clinical features of the two cases are strikingly similar with symmetrical neurological damage occurring in the presence of anoxia and metabolic acidosis. Infarcts of the basal ganglia and particularly the putaminal region are well recognised after anoxia. 2 Metabolic disturbances due to ingestion of foreign substances 3 or intrinsic defects may also cause basal ganglia necrosis. In cases due to carbon monoxide poisoning the ganglionic lesions may be haemorrhagic. 3 When CT scans have been performed in such cases they have usually shown bilateral ganglionic hypodensities though there has often been a delay between the cerebral insult and the scan. In our case, the detection of ganglionic hyperdensity on the CT four days after a major deterioration in neurological function with a change to low density after six weeks would be compatible with haemorrhagic pathology. 4 A similar CT pattern has been reported in a patient with uraemia, the original hyperdensity becoming hypodense by day 14. We do not know whether our patient’s lesion was haemorrhagic from the outset but we postulate that significant haemorrhagic transformation of a previously bland infarct resulted in the major neurological damage and temporary deterioration in his metabolic control. Although no definite reasons for the haemorrhagic transformation could be identified from his records, the symmetrical changes suggest that a factor such as an unrecognised surge in systemic blood pressure might have been responsible.

References


Focal epilepsy in India

Sir: We were interested to read the report of Dr RS Wadia et al in the October issue of the J Neurol Neurosurg Psychiatry on ring and disc lesions in focal epilepsies. Of their 150 cases, 39 showed a ring or disc lesion, after contrast injection, on CT scan. Of those 39 cases, 10 cases had tuberculosis elsewhere in the body, three had a past history of tuberculosis and four others had a history of "close contact" with a case of tuberculosis. It is not mentioned whether the three cases with a past history had been treated for tuberculosis or not. The other 22 cases had no tuberculosis elsewhere but were also treated with anti-tuberculosis therapy. However, of these 39 cases only two were histologically proven tuberculomas.

The authors assume that infections in the lungs or elsewhere at the time of fits, a past history or close contact with tuberculosis is sufficient evidence to call the ring/disc lesion a tuberculoma. It is, however, uncommon in this country to see a patient with an expanding lesion in a hemisphere or stroke to have tuberculosis in the lung, either active or burnt out. At necropsy as well, it is common to see old tuberculous lesions in patients dying of unrelated causes. Again, tuberculosis being a social stigma, is not uniformly notified; hence the term "contact" is, at best, puristic. Therefore unless one compares the incidence of extracranial tuberculosis (active or burnt-out) in a group of focal epilepsies with a positive ring or disc lesions with another (for example those with complex partial or generalised or simple partial epilepsy) but without a positive scan it is not possible to determine the statistical significance of the association of extracranial tuberculosis and the lesions revealed by CT.

Against the idea of disappearing lesions being those of focal encephalitis the authors cite the example of primary a lesion in the lung which clears on its own. We feel this comparison is erroneous as both the organs are structurally unrelated and, more so, immunologically.

After the advent of CT in India there was a wave of "tuberculomas" among focal epileptics. The present phase is one of introspection. After the publication of Sethi et al we have felt justified in treating patients with focal epilepsy and ring or disc enhancing lesions with no drug except anticonvulsants. Arbitrarily we decide to give anti-tuberculosis therapy after 3 months if the lesion does not disappear or the surrounding oedema increases, or the lesion expands.

We have seen earlier instances where the ring lesions were treated with anti-tuberculosis drugs. The repeat scan done after 3 months showed its disappearance, to reappear after fit while the therapy was continued.

A stereotactic biopsy of the lesion may certainly settle such issues. As experience increases, the mortality and morbidity of this procedure are negligible. It is also required to study the spectrum of focal encephalitis and the evolution of their CT appearances. It is possible that some of these lesions persist well beyond 3-4 months or increase and may still not be tuberculomas. In our opinion the ring or disc lesion is a nonspecific finding caused by a variety of diseases like tuberculosis, supratentorial gliosis, focal encephalitis, gliomas, secondary deposits or granulomas due to other infectious causes like larva migrans, cystercerosis or toxoplasmosis.

Short of biopsy one may have to, as a policy, delay the scan as far as possible after a focal fit. At the end of three months it may be possible to identify those lesions which were destined to disappear. In those that persist, one has to either adopt a policy of observation or stereotactic biopsy. It is also worth doing immunological studies of CSF to detect tuberculoprotein.

We do not deny that a small percentage of patients with focal epilepsy have a tuberculoma. But to what extent and which lesions constitute tuberculosis is, as yet, not known.

We share the dilemma and the agony of indecision with Wadia et al but take exception to their conclusion that one-third of ring/disc lesions are tuberculomas and certainly to the blanket policy of anti-tuberculosis therapy in all ring and disc enhancing CT lesions in India.

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References
1 Wadia RS, Makhale CN, Kelkar AV, Grant KB. Focal epilepsy in India with special reference to lesions showing ring or disc like enhancement on contrast computed tomography. J Neurol Neurosurg Psychiatry 1987;50:1298-301.

Wadia et al reply

We read the comments of Drs Diwate and Apte on our paper with interest. We were expecting comments from several colleagues from this country because this problem has been exercising all neurologists here. Our paper represented an attempt at outlining the problems.

We did not include cases with a past history of tuberculosis or close contact with tuberculosis, among those we thought certainly had tuberculomas. In that group we included the two histologically proven, the 10 with tuberculosis (active) elsewhere, and the one who developed meningitis. We feel that if a person with tuberculosis of the lung develops another lesion also known to occur with tuberculosis then the lesion is due to tuberculosis unless proved otherwise. If a case with pulmonary tuberculosis develops tuberculous meningitis with an excess of lymphocytes in CSF I am sure Drs Diwate and Apte consider that tuberculous meningitis is present and do not spend scarce resources and time in proving that aetiology, nor would they advocate biopsy of the meninges. Moreover when that lesion resolves on treatment we think the matter should be considered settled on circumstantial evidence. That a tuberculoma does have this CT appearance has been proven by us as well as by others. That it is a relatively frequent CT appearance in these cases is seen in cases of miliary tuberculosis and tuberculosis meningitis where we have seen similar lesions frequently. This has been reported by others also.

Drs Diwate and Dr Apte point out that in India a patient with glioma may also by chance have active or burnt out tuberculosis or a history of contact. As we said they misread our article and we only included the active cases. The incidence of active tuberculosis in India is high but official figures for our state put the incidence at just under 2% of population. The incidence of tuberculosis in gliomas should be similar. Our incidence of 10 in 39 is nearly 12 times