Matters arising


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.1 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, not 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 epileptics with a positive ring or disc lesions with another (for example those with complex partial or generalised 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 al2 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 antituberculosis 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.3 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 non-specific finding caused by a variety of diseases like tuberculosis, supratentorial gliosis, focal encephalitis, gliomas, secondary deposits or granulomas due to other infective causes like larva migrans, cysticercosis 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.4

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.

PG DIVATE
CA APTE
Division of Neurosciences,
King Edward Memorial Hospital,
Pune, India

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 lungs 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 tuberculosis meningitis with an excess of lymphocytes in CSF I am sure Drs Diwate and Apte consider that tuberculosis 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.12 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.3

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.4 The incidence of tuberculosis in gliomas should be similar. Our incidence of 10 in 39 is nearly 12 times