Matters arising

Focal epilepsy in India

Sir: We read with interest the article on focal epilepsy in India by Wadia *et al*.¹ Like other neurologists and neurosurgeons in India we have seen a considerable number of patients with focal epilepsy whose CT scans show small ring or disc enhancing lesions. We have done CT guided stereotaxic biopsies on six such lesions using the Brown-Roberts-Wells system (Radionics Inc. Burlington Mass, USA). In all these cases the biopsy was reported as "nonspecific chronic inflammatory lesion". These lesions are "focal encephalitis" whose cause has yet to be determined. The enhancing lesions are the cause of the epilepsy and is therefore not a post-ictal phenomena. Five of these patients were followed up with only anticonvulsant therapy and in all of them the lesions have disappeared or left a small calcified residue, within three months. We also think that the CT characteristics of these disappearing lesions are distinct from those of small tuberculomas. Therefore we disagree with the policy, advocated by Wadia *et al*,¹ of starting these patients on antituberculous therapy. We prefer to give only anticonvulsants as advised by Sethi *et al*.² A detailed report of these patients with the pathology and CT characteristics of these lesions will soon be published.

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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.
- 2 Sethi PK, Kumar BR, Madan VS, Mohan V. Appearing and disappearing CT scan abnormalities and seizures. *J Neurol Neurosurg Psychiatry* 1985;48:866-9.

Wadia *et al* reply:

We did not in fact state anywhere that we advocate the use of antitubercular therapy in all such cases. We did in the period 1982-85 use antituberculous therapy for all cases.

Towards the end of the study we also considered treatment of cases without such drugs. Unfortunately our first such trial was a near disaster. As evidence accumulated from other centres we now do not use anti-tuberculous treatment in all cases. We know that the vast majority of neurologists in India were doing as we were, in that period probably including the authors of the above letter. That some of these lesions are tuberculomas we have already proven unequivocally (histologically positive). The question of how many remains to be answered by accumulation of other consecutive studied cases in series such as ours.

The authors say they have biopsied six lesions. Were these consecutive cases? If they were the trial must have started after our paper was published. We see 1-2 cases a month in our relatively small hospital. If it was started before the paper was published then it is a totally selected set of biopsies and a large number of cases have not been biopsied and the significance of their finding is unclear. If the cases are consecutive cases would it be correct for us to state that the authors are now "advocating" that all centres in India do likewise or transfer these cases to centres that can do so?

At present we stick by our view as expressed. Biopsy at first diagnosis seems unnecessary for the lesions resolve with medical therapy. There is dilemma as to aetiology. Some of these are tuberculomas. Others may be of diverse aetiology of which focal encephalitis of unknown aetiology may be one. At the time we wrote the article evidence for this was only a hypothesis but the authors seem to show some more positive evidence of the same. However, we have heard good neuropathologists say clear diagnosis of tubercular infection from stereotaxic CT guided biopsy would be difficult. We had originally written that waiting without specific treatment may be hazardous if follow up is defective. This we have already shown may be so. It is clear now that our idea of the hazard was over-rated. A study from several centres in India where *all* cases are biopsied would teach us more on these lesions though we do not think (at present) that this would be the best way to manage these cases.

Carnitine palmitoyltransferase deficiency

Sir: In a recent issue of this journal Carey *et al*¹ reported a case of carnitine palmitoyltransferase (CPT) deficiency

presenting with abnormal mitochondria. This paper contains several points which require further comment.

First, there are two enzymes with CPT activity, CPT I and CPT II; they have different molecular weights and are located at different sites. CPT I is located in the outer mitochondrial membrane whereas CPT II is on the inner face of the inner membrane.² The assay used does not differentiate between the two types. This uncertainty is compounded by the absence of any information about the conditions used for centrifugation of the homogenate. We are not able to decide whether they measured CPT I, CPT II or total activity. We also feel that the use of an insensitive end-point assay is much less satisfactory than those which allow the measurement of the rate, and as these exist for the measurement of CPT I and CPT II activities their use is to be preferred.³

Second, the authors are correct in stating that morphological abnormalities have never been reported in CPT deficiency. However, they suggest that the apparent CPT deficiency is the cause of the morphological abnormalities demonstrated, without having excluded any of the more common causes such as defects of the respiratory chain, in particular cytochrome oxidase deficiency. It is entirely possible that the activity of affected mitochondrial enzymes was low in the fraction studied.

Third, measurement of ¹⁴CO₂ release from ¹⁴C-palmitate is not an accurate measure of palmitate oxidation. The ¹⁴CO₂ released is only a minor product and does not necessarily reflect the flux through beta-oxidation.^{4,5}

Fourth, the authors in table 1 have mentioned a mean value plus a range. From their figures it seems that they only determined the activity in two control subjects. If so, this should be stated, as it would then make the results quoted in this table meaningless since the figures for the controls are different by an order of magnitude.

We feel that the results shown in this paper do not confirm CPT deficiency as the primary biochemical defect. It is necessary to raise the above points as the diagnosis of mitochondrial disease is difficult and a more thorough and logical approach is necessary.

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