Correspondence

Reye’s syndrome: diagnosis by muscle biopsy?

Sir,

I read the paper by Shapira et al.1 with great interest as it was, to my knowledge, the first report of this condition. Although recurrent Reye’s syndrome has been reported,2 Pichichero and McCabe did not obtain liver biopsies in any episode. Shapira et al.1 noted the marked fat droplet infiltration in hepatocytes by electron microscopical examination but they did not mention any mitochondrial changes in their patients although they accepted the fact that primary generalised mitochondrialopathy is the pathophysiological basis of Reye’s syndrome. Were mitochondrial changes present in their case 3?

Shapira et al.1 pointed out the potential danger of bleeding in percutaneous liver biopsy, but in our experience of liver biopsies on at least 947 children (311 of them below age 2 years), this danger should not be exaggerated, provided the necessary precautions are taken.4

High levels of plasma lactate, alanine, and glutamine were accepted as indicating intramitochondrial decreased oxygenation of pyruvate as a result of primary generalised mitochondrialopathy. Although mitochondrial structural alterations appear to be similar in brain, muscle, heart, lung, and kidney, the activities of mitochondrial enzymes—such as citrate synthase, glutamic dehydrogenase, succinic dehydrogenase, pyruvate carboxylase, and pyruvate dehydrogenase—are decreased in the liver but not in the brain or muscles.4 Therefore I think that morphological mitochondrial changes should be interpreted cautiously as evidence of primary generalised mitochondrialopathy. Are these changes primary or secondary to carnitine depletion in cases of recurrent Reye’s syndrome?5

Dr Shapira and co-workers comment:

Cases 1 and 3 underwent both liver and muscle biopsies but Case 2 had only a muscle biopsy; morphological abnormalities in the mitochondria were identical in all 3 muscle biopsies and in both liver biopsies. It should be mentioned that liver biopsies were described in recurrent Reye’s syndrome secondary to systemic carnitine deficiency.5

Dr Ozsoylu referred to his vast experience with 947 liver biopsies in children and said that the danger of bleeding should not be exaggerated provided the necessary precautions are taken.9 We agree with him, but in

References


Birthweight, child abuse, and infant death attributed to accidents, poisonings, or violence

Sir,

A recent analysis that links infant deaths that occurred in 1978 to birth records is very welcome.1 One striking feature is the association of infant death attributed to accidents, poisonings, and violence (ICD code E800–999) with birthweight. The incidence of such deaths was 0·31 per 1000 live births. Rates within the birthweight bands <2000, 2001–3000, and ≥3001 g were 1·03, 0·47, and 0·25 per 1000 respectively, a highly significant trend (χ²=35·1, df = 2, P<0·001). If these results are considered in the light of data from Cardiff2 relating to the incidence of non-accidental injury (not necessarily fatal) in children aged 5 years and below, the incidence is 2·7 per 1000 live births in the entire series, and 13·0, 3·8, and

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