inflammatory damage to peripheral nerves is caused by the infective agent and its toxins or by an immunological response triggered by them has long been debated. Much evidence supports an immunological cause for this condition, but it is not clear whether our patient's neuropathy was of this type or a specific feature of legionnaires' disease. Kennedy *et al*³ found neurophysiological evidence for an acute axonal neuropathy in five patients with legionnaires' disease, though clinical manifestations were evident in only a single case. Their findings and the small rise in cerebrospinal fluid protein concentration in our patient offer some support for a specific neuropathy accompanying legionnaires' disease.

We thank Dr A Pringle for permission to report this case.

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Acute polymyopathy during total parenteral nutrition

Total parenteral nutrition is often used as nutritional support¹; deficiency states of various nutrients have been recognised in association with it. We report four cases of an acute myopathy associated with total parenteral nutrition that improved dramatically either when the nutrition was stopped or with intravenous lipid supplementation.

Case reports

Case 1—A 59-year-old woman with coeliac disease and previously treated oesophageal carcinoma was started on total parenteral nutrition. Weight gain was 6 kg in 34 days. A lipid supplement (Intralipid 10%, Vitrium) was initially part of the regimen but was stopped because of thrombophlebitis at infusion sites. The figure shows plasma creatine kinase activities (normal <140 U/l) from the day after the last infusion. Aspartate aminotransferase activity rose transiently on day seven, but no associated increase in bilirubin concentration or alkaline phosphatase activity occurred. Parenteral nutrition was stopped on day 20 because of muscle pain, tenderness, swelling, and tachycardia. By day 21 muscle symptoms had abated and by day 22 creatine kinase activity was normal. The ratio of 5,8,11-eicosatrienoic acid to arachadonic acid in plasma lipids was 1.4 on day 20 (normal <0.4). Seven days after parenteral nutrition was stopped the ratio was 0.3, and after 14 days no 5,8,11-eicosatrienoic acid was detected.

Cases 2 and 3—Two further patients with similar symptoms and responses to total parenteral nutrition were seen; they did not receive lipid supplementation.

Case 4—A 54-year-old woman was started on total parenteral nutrition for small-bowel fistula after gastrectomy for carcinoma of the stomach and gained 7 kg in weight. On day 23 she complained of muscle weakness and plasma creatine kinase activity was raised (figure). By day 29 she had developed muscle pain and tenderness. On day 32 a lipid infusion (Intralipid 10%, 500 ml) was started and total parenteral nutrition continued. Muscle pain immediately lessened and then disappeared. Creatine kinase activity reached normal values by day 36. The ratio of plasma 5,8,11-eicosatrienoic acid to arachadonic acid on day 32 was 1.9. No eicosatrienoic acid was detected seven days after the start of lipid treatment. Parenteral nutrition was continued until day 40 without further complications.

Methods—Total parenteral nutrition was performed by standard techniques and provided up to 16.7 MJ (4000 kcal)/day. Recommended allowances of vitamins and trace elements were administered. Biochemical and haematological tests were performed thrice weekly and concentrations of vitamins A, C, and E, β -carotene, thiamine, and pyridoxine measured weekly. Electromyography confirmed an acute myopathy. Muscle biopsy specimens could not be obtained. The constituent fatty acids in plasma were analysed

Creatine kinase activity during total parenteral nutrition in cases 1 and 4.

by gas-liquid chromatography after extraction and hydrolysis. Deficiency of essential fatty acids was diagnosed when the ratio of 5,8,11-eicosatrienoic acid to arachadonic acid was greater than 0.4.²

Comment

The acute myopathy in these patients was clearly related to parenteral nutrition. Stopping the regimen resulted in clinical and biochemical improvement within hours and complete resolution of the myopathy within days. Hepatomegaly was not detected clinically, and there was no biochemical evidence of hepatic dysfunction.

The cause of the myopathy was not established, though deficiency of essential fatty acids probably played a part. Characteristic chemical evidence for such a deficiency was detected. Though measurable amounts of 5,8,11-eicosatrienoic acid are expected after parenteral nutrition without lipid supplementation, a ratio of eicosatrienoic to arachadonic acid of the magnitude seen here indicates severe deficiency.³ Parenteral nutrition causes high circulating insulin concentrations, which suppress release of endogenous essential fatty acids from adipose tissue stores, resulting in a relative deficiency. Stopping parenteral nutrition thus allows release of these acids. The rapid clinical and biochemical response brought about by adding a lipid supplement to the regimen (case 4) is consistent with this hypothesis. Deficiencies of phosphate, vitamin E, and selenium have been associated with myopathies and muscle damage but were excluded in these cases.

No further cases of acute myopathy occurred after intravenous lipid supplementation was added on a regular basis to the nutritional support protocol. Whether a pure deficiency of essential fatty acids was the direct cause of this syndrome was not established. The lipid supplement may provide another as yet unidentified nutrient or, alternatively, essential fatty acids may reverse the abnormality without correcting the basic defect. Whatever the mechanism, lipid supplementation seems to prevent and reverse the muscular syndrome without the need to stop the nutritional support.

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