Medical Memoranda

Alcoholic Myopathy

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The untoward effects of excessive alcohol on the nervous system, heart, and liver are well recognized. That alcohol may cause a variety of clinical syndromes involving skeletal muscle is not so well known.

CASE REPORT

A 46-year-old Irish window cleaner began to complain of sharp pains in the limbs and swelling of the calf muscles in March 1968, in association with gastric pain, anorexia, vomiting and loss of about 2 st. (12.7 kg.) in weight. Since the age of 16 he had been drinking 18 to 20 pints (10 to 11 litres) of beer daily. Four months later his arms and legs had become weak, and his fingers often became cold and blue with occasional swelling of the ankles. By February 1969 he found it difficult to climb stairs and to rise from the horizontal position. He complained of discomfort on swallowing solid food and he had noticed occasional melena. He no longer had abdominal pain and vomiting, and his weight had remained steady. He had not worked for six months and had continued drinking alcohol.

On examination he was a thin, neglected man with rather prominent eyes and a fine tremor of the outstretched hands. There was enlargement of the right breast, in which a smooth, hard, mobile swelling was palpable, but no other stigmata of hepatic disease were found. The heart and blood pressure were normal. The liver was smooth and enlarged by 3 cm. There was obvious muscle wasting and weakness, mainly involving the shoulder and pelvic girdle muscles and the proximal muscles of the limbs. The muscles of the forearm were spared and there appeared to be some pseudo-hypertrophy of the calf muscles with minimal subcutaneous oedema. No neurological abnormality was demonstrable. A provisional diagnosis of myopathy due to an underlying neoplasm or to hyperthyroidism was made.

Investigations showed: haemoglobin 13.5 g./100 ml; blood film normal; W.B.C. 6,800/cu. mm.; E.S.R. 37 mm. in one hour; blood urea 11 mg./100 ml.; serum sodium 134, potassium 4.5, and chloride 95 mEq./L; total bilirubin 1.1 mg./100 ml.; thymol turbidity 0.9 units; alkaline phosphatase 132 K.A. units/100 ml.; alanine and aspartate aminotransferases 30 and 50 i.u./L; lactic dehydrogenase >2,000 units/ml.; total protein 6.4 g./100 ml.; prothrombin time 13 sec.; acid phosphatase 1.8 units/100 ml.; E.L. cells not found; P.B.I. 3.7 mg./100 ml.; radioiodine uptake normal; screening test for urinary porphyrins and myoglobin negative; serum aldolase 52 Sibley-Lehninger units/ml (normal 0–9.6 units/ml.); serum creatinine phosphokinase 3,440 i.u./L (normal 0–80 i.u./L.); serum magnesium 1.85 mg./100 ml.; blood lactate acid basal level 8.9 mg./100 ml., with an abnormally low rise in blood levels after ischaemic exercise; plasma pyruvic acid 0.6 mg./100 ml. Liver biopsy: non-specific round-cell infiltration in the portal tracts, with an accumulation of wear-and-tear pigment in the hepatic cells but no evidence of cirrhosis. Breast biopsy: gynaecomastia but no malignancy. Chest x-ray picture, barium meal, and enema normal; barium swallow: sliding hiatus hernia oesophagitis; spinal x-ray films: degenerative changes, no evidence of metastases. E.C.G.—ischaemic.

Special Investigations (Dr. A. L. Woolf)

Electromyography.—Motor unit action potentials of low to moderate voltage with short duration phases; no slowing of conduction along ulnar or peroneal nerves (44-5 and 41-0 m/sec. respectively)—stimulation and recording with surface electrodes—stimulus of 0.1 m/sec. and 250 V).

Muscle Biopsy.—Haematoxylin and eosin: pale muscle fibres with occasional basophilic fibres, separation of myofibrils, no inflammatory cells, increase in sarcocellular nuclei. Vital staining with methylene blue: distal portions of subterminal nerve fibres stain darkly, swelling and fusion of terminal axonic expansions of motor end plates. Electron microscopy: degeneration of axons and disruption of myelin sheath in intramuscular nerves and in muscle fibres with clumping of organelles, myopathic changes in many muscle fibres with invasion of damaged fibres by phagocytes, mitochondrial swelling, motor end plates (in contrast to vital staining) apparently normal, originally normal expanded terminal motor axon expansions surrounded by membranous profiles and terminal axonic expansions with degenerating processes. Histological studies: large and small vacuoles in many fibres with patchy loss of enzyme activity. In-vitro studies with intracellular electrodes: low mean resting membrane potential of muscle fibres, miniature end plate potentials of normal frequency and amplitude recorded from one fibre.

Progress.—Because of the severity of the myopathy, treatment with prednisolone 60 mg. daily was begun while investigations were proceeding. This had no effect and was gradually reduced and stopped once the diagnosis of alcoholic myopathy had been made and he was advised to cease drinking. Unfortunately he has been unable to abstain completely from alcohol, and though his condition has not deteriorated there has been no improvement in muscle power.

Comment

Alcohol may cause either an acute or a chronic myopathy. The acute syndrome (Hed et al., 1962; Perkoff et al., 1967) is characterized by painful localised or generalized muscular cramps and sometimes by weakness and tenderness of muscles with subcutaneous oedema. Raised levels of muscle enzymes with a diminished ability to increase blood lactic acid levels in response to ischaemic exercise are common findings. Myoglobinuria, hyperkalaemia, renal failure, and sudden death may occur. Recovery after abstinence from alcohol is the rule.

In the chronic syndrome (Ekbom et al., 1964; Perkoff et al., 1967) muscular weakness and sometimes tenderness of the proximal muscles occur. The neurological complications of alcoholism and cirrhosis are commonly associated. Biochemical abnormalities are similar to those in the acute syndrome, but are less striking and the electromyograph is myopathic. Recovery occurs in about 50% of cases if alcohol is stopped.

The features and relationships of the acute and chronic syndromes were investigated in detail by Perkoff and his colleagues (1967). They also showed that alcoholic patients, in whom no muscular symptoms were present but from whom a history of muscle cramps was frequently obtained, often had biochemical findings similar to those found in symptomatic subjects.

The clinical and biochemical features in the present patient are more characteristic of the chronic syndrome, whereas the muscle biopsy would suggest that the case falls into the acute rather than the chronic group according to the criteria of Klinkerfuss et al. (1967). It would appear that there is considerable overlap in the various syndromes. The conduction velocities of the ulnar and peroneal nerves were normal, but as alcoholic neuropathy is an axonal degeneration the conduction velocities may not be significantly abnormal, and, though the intramuscular nerves appeared normal in light microscopy, electron microscopy showed axonal degeneration. A diagnosis of alcoholic myopathy may be overlooked, especially in the acute group, when the symptoms may be attributed to an alcoholic neuropathy. If oedema and swelling of the calf muscles are present the clinical picture may closely resemble bilateral deep vein thrombosis. If myoglobinuria occurs the condition may be diagnosed as idiopathic myoglobin-
Recurrent Virus Meningitis

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Aseptic meningitis is a common clinical problem, but one patient having two separate attacks with different viruses is exceptional enough to be of interest. Gray, Moffatt, and Sangster (1969) described such an event in a patient who, having recovered from mumps meningitis in 1961, developed E.C.H.O. 30 meningitis in 1966. A review of the literature and correspondence with colleagues of wide experience have not revealed any further virologically proved examples of such double attacks. The following case history therefore seems worthy of report.

CASE REPORT

A 6-year-old boy, an only child, was admitted to Taunton Isolation Hospital on 26 July 1968 with a 24-hour history of headache, drowsiness, and some vomiting. The clinical picture was one of meningeal irritation; his cerebrospinal fluid (C.S.F.) contained 76 cells/cu.mm., mainly lymphocytes, but was otherwise normal. He made a rapid and complete recovery, being well enough to return home within a week. An E.C.H.O. virus type 6 was isolated from his faeces.

Ten months later he was readmitted, again having been unwell for about 24 hours, with headache and neck stiffness. On this occasion he was more severely ill and remained febrile for almost a week. The diagnosis of tuberculous meningitis had now to be entertained, since the C.S.F. contained 120 cells/cu.mm. (rising later to 550), 60% lymphocytes, a protein of 75 mg./100 ml., and a sugar of 36 mg./100 ml.; no organisms were seen on the film. His Mantoux 1/100, however, was negative, his chest x-ray picture was clear, and no choroidal tubercles were seen on ophthalmoscopy. He remained alert and rational, and his electroencephalogram was normal; chemotherapy was therefore withheld. Equivocal fullness of one parotid was overlooked; but during the second week his mumps complement fixation tests were reported as V/1/10,240 and S/1/160; his serum amylase was 210 units/100 ml. (upper limit of normal 150). His mother then recalled contact with a child incubating mumps 19 days before admission. He was now recovering steadily, and soon returned home, resuming school attendance before the end of term. When reviewed in August 1969 he was physically fit, but was reported to be bad-tempered and easily upset. His mumps complement fixation tests had fallen to V/1/120 and S/1/40; the serum amylase was 85 units %; mumps virus had been grown from a throat swab, but not from the faeces or C.S.F. His serum proteins were normal, including electrophoresis of the immunoglobulins.

COMMENT

Recurrent bacterial meningitis is a well-recognized clinical problem usually associated with structural or immunological anomalies. More than one attack of proved virus meningitis, however, seems a curious coincidence—presumably without such clinicopathological implications. Nevertheless, a challenging diagnostic and therapeutic predicament may be encountered in the individual patient. Careful separate assessment of each clinical episode is absolutely essential. The risk that a half-treated suppurative condition may masquerade as a "virus" meningitis must always be recalled (Brown, 1967), and tuberculous meningitis must be kept in mind, though fortunately this is now a clinical rarity (Anderson, 1964; Christie, 1969). The temptation to give ill-judged antibacterial therapy may be strong in a second meningitic illness, particularly if virological investigation of the first attack was incomplete and there is therefore past as well as present diagnostic uncertainty. The above case report illustrates the value of full virus studies (combined selectively with E.E.G. and other aids to exclude a space-occupying lesion) in clinical management.

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REFERENCES