Letters

Muscle hypoxia in polymyositis

Sir: The pathogenesis of polymyositis is unknown, but there is circumstantial evidence that pathological changes in the intramuscular blood vessels may play a role. Ultrastructural abnormalities have been found in the endothelium of the microvessels, and capillary basement laminae have been reported to be thickened. Capillaries are lost in polymyositis, particularly in the dermatomyositis subtype.1

These changes, possibly triggered by immune complex deposits in the vessel walls,2 may be responsible for the reported decline in muscle blood flow in polymyositis,3 but it is not known whether they are able to render the muscle ischaemic enough to contribute to the necrotising damage in the muscle tissue.

We measured tissue gas tensions in five adults (four women, one man, age 42–67 years) with 2–7 years history of definite polymyositis as diagnosed by clinical, neuropsychological and muscle biopsy examinations. One of them had skin symptoms suggesting dermatomyositis. All of these patients were ambulatory, and were at a chronic stage of disease with only minimal clinical symptoms (one further patient—with a low resting PO2—is not included in the results, as her exercise test had to be interrupted because of fatigue). Five healthy volunteers (two women, three men, age 42–68 years) served as controls. The measurements were carried out by means of an implanted Silastic tonometer.4,5 Under local anaesthesia, a 16 cm long silicone elastomer tube with an external diameter of 1.4 mm and an internal diameter of 1.0 mm was inserted into the medial belly of the gastrocnemius muscle. Care was taken to insert the tonometer at the same depth in the patients and controls. The recordings were made four days after implantation. The tube was slowly perfused with hypoxic saline, and the PO2 and PCO2 of the efflux were continuously monitored both at rest and during a heel lifting exercise.5

As shown in the figure, the calf muscle PO2 levels were consistently lower in the polymyositis patients than in the controls. The differences are statistically significant both at rest and during exercise (p = 0.004, Mann-Whitney U test). The lowest values were found in the dermatomyositis patient. PCO2, on the other hand, behaved similarly in the two groups. The response of PO2 to exercise in the polymyositis patients was different from that in patients with occlusive arterial disease: in the latter condition the PO2 is close to normal at rest but decreases during exercise.5 It seems, therefore, that in polymyositis the larger vessels are patent and they are able to respond to the needs of autoregulation. The finding of low average PO2 in polymyositis patients both at rest and during exercise suggests that the capillaries are unable to maintain normal tissue oxygenation. Thus, the behaviour of PO2 in polymyositis is consistent with the hypothesis that circulation is hampered at the level of small peripheral vessels.

It may be asked whether the disability and inactivity of the polymyositis patients causes diminished blood flow and drop of temperature in the muscle tissue with resultant unspecific changes in tissue gas tensions. The patients, however, were ambulatory and had only minimal weakness at the time of the study. Furthermore, low tissue temperature would enhance the solubility of O2 in the extracellular fluid, and higher (not lower) PO2 would be found in measurements carried out at the standard temperature of 37°C.

The finding of marked relative muscle hypoxia in idiopathic inflammatory myopathy suggests that microvascular obstruction plays a role in its pathogenesis. The result may also have therapeutic implications.

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References


Accepted 1 April 1986

Dilated tonic pupils in neurosyphilis

Sir: In patients with central nervous system syphilis, pupillary abnormalities are a relatively common finding, and the most important early ocular sign.1 The following case is presented as an example of a previously noted2 but not widely recognised association between bilaterally dilated tonic pupils, fixed to light and accommodation, and neurosyphilis.

A 48-year-old male presented with a 4 to 5 year history of mild photophobia. There was no history of diabetes, hypertension, trauma, headaches, fever, chills, nausea, vomiting, seizures, weakness, pain, or use of medications or illicit drugs. He claimed no alcohol use for at least four months but admitted to previous heavy ethanol abuse. He stated that he had occasional diplopia and intermittent urinary incontinence. At age 26 years, the patient presented with a penile chancre and positive serum VDRL, and was treated with a series of intra-
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J Neurol Neurosurg Psychiatry 1986 49: 1455
doi: 10.1136/jnnp.49.12.1455