association between the diseases. This association has been described previously in non-English publications, however.

There are some diseases which could explain both the pulmonary and systemic involvement similar to the one seen in these patients, but these were reasonably ruled out in our patients on the basis of clinical features, special studies, and histological findings.

As immunological phenomena play an important part in polyarteritis nodosa and idiopathic pulmonary fibrosis a common immunopathological link between the two diseases may exist. It is more probable, however, in view of the rarity of the association that the joint occurrence of these two uncommon diseases is simply coincidental. Further reports of new cases might help to determine if this association is real or not.

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References


Letters 877

Autonomic dysfunction in systemic sclerosis: the site of damage

Sir, In considering the site of damage underlying abnormal autonomic function in their patients with systemic sclerosis, Klimiuk and colleagues highlighted the lack of evidence for the presence of somatic neuropathy in the four cases reported by Sonnex and coworkers. As we have emphasised elsewhere there are several reasons why such signs may not have been detected in that study. Firstly, clinical assessment was limited to the legs and to the use of vibration threshold. Secondly, nerve conduction studies were performed in only one case. (Details of these studies were, moreover, not given.) Finally, and perhaps not least, morbid involvement of peripheral nerves in systemic sclerosis may be focal.

After the article by Klimiuk and colleagues had been accepted by the Annals we published a report of the results of cardiovascular autonomic tests in eight unselected cases of systemic sclerosis. The six patients who had abnormal values for one or more of the five tests all had clinical (n=6) or electrophysiological (n=5) evidence of somatic neuropathy also. Of the sites thereby sampled, the sural nerve was involved in every instance, the median motor nerve in two instances, and the median sensory and peroneal nerves in one instance each. Abnormalities were consistent with axonal degeneration. This type of pathological change occurs in angiopathic neuropathy, while arterial damage is a characteristic feature of systemic sclerosis.

Having regard to the above circumstances, we believe it is premature to imply, as Klimiuk and colleagues did, that the neuroanatomical basis of autonomic dysfunction in systemic sclerosis differs essentially from that in other connective tissue diseases because in those conditions a somatic neuropathy is usually demonstrable. The possibility that damage is located more proximally to the effector site is nonetheless an intriguing suggestion and we await with interest the outcome of the authors’ studies. Meanwhile, we wonder whether their next report will also include an assessment of peripheral nerve function.

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References


Does joint cartilage require energy?

SIR, Geborek et al showed that induced increase of synovial fluid pressure decreases synovial blood flow, which, as expected, decreases synovial fluid oxygen tension. In five minutes synovial fluid pressure raised by flexion decreased synovial fluid oxygen tension in 3/10 cases of knee trauma; these three synovial fluids contained few red cells, potential buffers against hypoxia. Even at resting joint angles synovial fluid pressure correlated with synovial fluid hypoxia and, like synovial fluid volume, erythrocytes, and leucocytes also, with synovial fluid lactate (unpublished). Ischaemia cannot induce such changes unless oxygen and glucose are consumed.

We lack data on oxygen and glucose consumption in man, but Bywaters found that each mg of dry weight horse joint cartilage under aerobic conditions consumed 0.01 μl/h oxygen (his Table XIII) and produced 0.171 μl/h of lactate (Table VII), the rates in synovium being 0.8 and 1.7 μl/h respectively. In rabbit leucocytes the rates were 4.15 (oxygen) and 22.5 μl/h (lactate). From the surface areas in one human knee the volume of the innermost vascular synovial intima was calculated to be 0.554 cm³ and that of cartilage 44 cm³. We calculated that in μmol/l (a) this cartilage consumed 3.924 oxygen and produced 67.1 lactate (water 80%), (b) the synovial intima 5.93 oxygen and 12.6 lactate (water 70%), and (c) 10³ leucocytes (water 75%) 0.0419 oxygen and 0.227 lactate. In each hour (d) 10³ erythrocytes consume 0.027 μmol oxygen and produce 0.384 μmol lactate.

In many cases of knee trauma (unpublished) synovial fluid leucocytes and red cells seemed to have consumed more oxygen and glucose than (required by?) the joint cartilage.

The regulation of glycolysis in cartilage differs from that in liver as entry of pyruvate into the citric acid cycle is possibly inhibited by acetyl-CoA derived from fat. In normal cartilage most of the energy may be produced by glycolysis, but if the oxygen consumed is used for oxidation of glucose (lipids?) about a quarter of the energy may depend on oxygen. In acidotic joints proper energy supply might require more oxygen as glycolysis is depressed by high lactate concentrations.

Damage by reperfusion induced oxygen radicals has been stressed and considered. The synovial fluid oxygen tension dropped, however, at a rate (61 to 41 mmHg in two minutes) that might soon have resulted in anoxia and which, at synovial fluid volumes >10 ml, exceeded the calculated consumption by cartilage. We suggest that raised synovial fluid pressure and intra-articular cells deprive cartilage of energy. To avoid ischaemic-metabolic joint damage one should refrain from measures that disturb circulation in capillaries of cartilage in the synovium (removed at early synovectomy) and aim instead to correct changes of physiological mechanisms that, by increasing synovial fluid volume, increase synovial fluid pressure, the simplest being rinsing of joints to decrease the osmotic 'suck' of synovial fluid colloids.

References


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