Matters arising

Peripheral neuropathy complicating pancreatitis

We were interested in the observations of Gross et al.1 In 1970, we reported a case of a 30 year old sportsman who presented with acute pancreatitis, which was treated by surgery. Immediately after the operation, the patient developed encephalopathy characterised by a confusional state. Within a few days, a severe sensorimotor polyneuritis had led to quadriplegia. Nerve biopsy demonstrated a very severe axonopathy (reported at the VI Congrès International de Neuropathologie, 1970). The neuropathy disappeared completely over the next few months, followed by complete remission of the encephalopathy.

Although all reported cases had acute pancreatitis, there are some notable differences between our case and that reported by Gross et al. The neuropathy in our patient appeared within a few days of the onset of the pancreatitis, although in other cases, the first signs of neuropathy were only observed some weeks after the pancreatitis.

Our patient was not diabetic, and was not taking metronidazole or receiving parenteral nutrition. This would tend to rule out an aetiology involving significant vitamin deficiency. These conditions of onset also rule out the so-called "critically ill polyneuropathy".2 In addition, it is noteworthy that the acute pancreatitis in our patient was accompanied by involvement of both central (transient encephalopathy) and peripheral nervous systems. We feel that a peripheral neuropathy may, in some circumstances, result from an acute pancreatic lesion. However, further cases will need to be identified before a causal link can be established.

References

Letters

The conduction velocities of peripheral nerve fibres conveying sensations of warmth and cooling

Sir: Evidence that in man the sensations of warmth and cooling are conveyed by different fibres is indirect.3 Fowler et al.4 using reaction times to supramaximal heat and cold stimuli in normal controls recently demonstrated that the two modalities were conducted at different velocities suggesting that cooling was subserved by small myelinated and warming by unmyelinated fibres. We provide objective evidence that dissociated loss of warming and cooling sensation exists in diseases as seen in three patients with peripheral neuropathy associated with the acquired immunodeficiency syndrome.5 Thermal thresholds6 were tested using...
Matters arising

Table  Thermal thresholds and nerve conduction studies in peripheral neuropathy associated with AIDS

<table>
<thead>
<tr>
<th>Patient</th>
<th>Thermal thresholds °C</th>
<th>Nerve conduction studies</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Site</td>
<td>Hot</td>
</tr>
<tr>
<td>1</td>
<td>Wrist</td>
<td>0.1</td>
</tr>
<tr>
<td>2</td>
<td>Ankle</td>
<td>0.8</td>
</tr>
<tr>
<td>3</td>
<td>Wrist</td>
<td>1.5*</td>
</tr>
<tr>
<td>4</td>
<td>Ankle</td>
<td>4.2*</td>
</tr>
<tr>
<td>5</td>
<td>Wrist</td>
<td>0.1</td>
</tr>
<tr>
<td>6</td>
<td>Ankle</td>
<td>4.2*</td>
</tr>
<tr>
<td>Normal</td>
<td>Wrist</td>
<td>&lt;0.4</td>
</tr>
<tr>
<td></td>
<td>Ankle</td>
<td>&lt;2.8</td>
</tr>
</tbody>
</table>

The right side was tested in all 3 patients. Skin reference temperature 34°C. Abbreviations: * = abnormal; SAP, sensory nerve action potential (µV); MCV = maximal motor conduction velocity (ms⁻¹); Med pl = medial plantar nerve; C per = common peroneal nerve.

References


Fowler replies:

When a temperature testing device which can measure thresholds for warming and cooling separately is used in neuropathic patients, the finding of a differential loss affecting one thermal modality more severely than the other is common. In general we have found more marked impairment of perception of warming than cooling in diabetics. In some patients with mild diabetic neuropathy who have erectile dysfunction as the only manifestation of small fibre neuropathy, perception of warming on the sole of the feet may be absent while perception of cooling remains within normal limits.1 Also in diabetics with plantar ulcers occurring in the context of severe generalised neuropathy, we have shown a more severe abnormality in warming perception than cooling on the feet, hands and face.2 Indeed, using our particular testing system,2 such a pattern is almost the rule in diabetics; only in patients with Fabry’s disease have we observed the converse, a more severe abnormality for cooling than warming.

However, some caution should be exercised in the interpretation of such results. In our paper on the extent of small fibre neuropathy in diabetics with plantar ulcers3 we point out that selective damage to unmyelinated fibres may not be the only explanation for relatively high thresholds of warming: that the perception of cooling is a

a commercially available device (Medelec Triple T).

Case 1. A 34 year old homosexual man had noted numbness in his fingers and difficulty in doing up buttons for 6 weeks. He had noted an increased sensitivity to painful stimuli in his legs for 3 months. He had been HIV antibody positive for 3 years, had Pneumocystis carinii pneumonia 17 months before and had received bleomycin (270 mg total) and vincristine (3 mg total) for Kaposi’s sarcoma during the last year. He was on zidovudine, ketoconazole and cotrimoxazole. Neurological examination revealed no motor deficit with intact deep tendon reflexes. Pinprick and light touch were impaired in his fingers and toes and he had hyperpathia and hyperaesthesiae to mid thigh and mid forearm. Proprioception and vibration sense were intact. Thermal thresholds and nerve conduction studies are shown in the table.

Case 2. A 31 year old homosexual man had a 4 week history of numbness and tingling in all his toes and the little finger in his left hand. This had become painful after 2 weeks. He had been HIV antibody positive for 3 years, had Pneumocystis carinii pneumonia 27 months before and CMV pneumonitis diagnosed 2 weeks before. He was on zidovudine and foscarinet. Neurological examination revealed normal power and intact deep tendon reflexes with no deficit of light touch, pinprick, proprioception or vibration sense. The result of thermal thresholds and nerve conduction studies are in the table.

Case 3. A 33 year old homosexual man complained of pain and tingling for 3 months that had begun in his feet and gradually spread to his knees. He had been HIV antibody positive for 18 months, had CMV oesophagitis a year before and was taking acyclovir and ketoconazole. Neurological examination revealed no motor deficit and bilaterally absent ankle reflexes. Light touch and pinprick were absent to the ankle and impaired to the knees and vibration sense was absent to the knees bilaterally. Proprioception was normal. The results of thermal thresholds and nerve conduction studies are seen in the table.

All three patients had electrophysiological evidence of peripheral neuropathy. Cases 1 and 2 had exclusive impairment of either cooling or warming thresholds respectively. Case 3 had a disproportionate abnormality of warming threshold. There was no evidence of local cutaneous lesions, myelopathy or encephalopathy. The aetiology of the peripheral neuropathy in these cases is not clear.

It is of interest to note the association of spontaneous pain with the loss of warmth sensation and hyperpathia with the loss of the sensation of cooling. Although clinicians have long known that hot and cold sensations may be affected in different degrees1 the differential involvement of cooling and heating seen in these patients provides objective evidence that these modalities are conveyed separately. Such a dissociation was mentioned by Jamal et al3 in a variety of neuropathies but not discussed. Whether in our cases the pathology has affected differentially the receptors, the nerve fibres or both is not clear and further studies may provide more information.

We thank Dr B Gazzard for allowing us to study cases 1 and 3. GNF is supported by the MRC.

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