chronic arthritis. The virus divides preferentially in rapidly dividing cells particularly erythroid precursors, but by the time the rash appears, viremia is no longer detectable. This suggests that the neuralgic amyotrophy seen in this patient is due to an idiosyncratic immune response.

We recommend serological testing for parvovirus IgM in cases of neuralgic amyotrophy to ascertain the relative causal contribution to this painful and disabling condition.

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References


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Failure to detect plasma neuropeptide release during trigeminal thermocoagulation

Sir: Calcitonin gene related peptide (CGRP) is a novel 37 amino acid residue peptide which is widely distributed in the human nervous system and has potent pharmacological effects. CGRP-immunoreactive neurons are found in spinal and trigeminal ganglia as well as in their terminations in the spinal cord and brainstem. The peptide is released from rat trigeminal ganglion neurons in tissue culture; it causes vasodilatation of human pial arteries in vitro and when injected into human skin results in a flare-type reaction. Recently CGRP has been detected in plasma. It was therefore of interest to see whether CGRP was released during trigeminal ganglion thermocoagulation which was being carried out for intractable trigeminal neuralgia.

Vasoactive intestinal polypeptide levels were also measured. This peptide is contained in parasympathetic nerve fibres and it has been suggested that trigeminal ganglion stimulation results in the indirect activation of facial nerve parasympathetic fibres. Plasma samples (10 ml) were taken before the onset of thermocoagulation, towards the end of 3 minutes of thermocoagulation and 20 minutes after the end of the procedure. This series of samples was taken from five patients. Each plasma specimen contained 5000 units of the protease inhibitor Trasyloil and was cooled in ice prior to radioimmunoassay as previously described. No CGRP was detected in any of the samples assayed. VIP levels were all within the normal plasma range and showed no rise during the procedure (less than 5 pmol/l).

During trigeminal thermocoagulation some patients develop ipsilateral facial flushing which can persist for up to 30 minutes after the procedure. This is thought to be due to the antidromic release of neurotransmitter substances. A similar release of a peptide transmitter, such as substance P, from trigeminal fibres innervating the major cerebral arteries has been proposed as the cause of headache in migraine.

The failure to detect the release of CGRP or VIP into the plasma during trigeminal thermocoagulation may have various explanations. Even if these peptides are released at sensory and parasympathetic terminals respectively, it is possible that they are not released into the circulation in significant amounts. Alternatively plasma degredation may be very rapid. It is known that VIP has a plasma half life of under one minute. However this is unlikely to explain the failure to detect CGRP as this peptide has a plasma half life of 7 minutes.

If neuropeptides really do have a physiological role in man and, in particular, are involved in the mediation of certain pain states, the demonstration of their release in vivo is of great importance. It may be that cerebrospinal fluid or jugular venous blood samples would be more likely to show changes although to obtain such specimens would be harder to justify ethically.

References

Letters


Accepted 10 October 1986

Paroxysmal presentation of motor neuron disease

Sir: A 40 year old lady complained of brief attacks of a tight feeling in her throat, slurring of speech and difficulty in swallowing. No precipitating factors were known. Several such episodes were seen during a twenty minute consultation, each lasting for 15 to 20 seconds. In an attack she became severely dysarthric, indeed almost unintelligible. Her 12 month old child had recently been causing her considerable alarm by having breath holding attacks. There were no abnormalities on either general medical or neurological examination.

A definite diagnosis was not made and it was considered that the attacks might be “functional” in nature. On review 2 months later she had developed wasting of the small muscles of the hands, slight wasting of the tongue and had definite, mild, permanent dysarthria. Over the next few months she developed bulbar palsy and upper and lower motor neuron lesions in all four limbs. The now obvious diagnosis of motor neuron disease was made and electromyographic studies revealed denervation in all four limbs. She deteriorated steadily and died within eighteen months at home. Permission for necropsy was not granted.

The author has not seen this presentation of motor neuron disease previously. Awareness of the possibility would have prevented the consideration of functional disorder which held sway for a brief period of two months before the fatal nature of the disorder became apparent. The patient was never aware of the suspicion of a functional diagnosis. Once more the danger of the diagnosis of hysteria is highlighted. Absence of a diagnosis should never be equated with absence of all possible physical disease.

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