suffered from stiffness of the legs, difficulty in walking, and fatigue. Symptoms had progressed slowly from onset.

Examination showed a moderate spastic paraparesis with exaggerated knee and ankle jerks and extensor plantar responses. Slight mimic facial palsy was present on the right. Sensation was normal. Urodynamic study showed an uninhibited bladder and sphincter-detrusor dyssynergia. Contrast myelography, magnetic resonance imaging of the brain and spinal cord, visual evoked responses, blood biochemical values, and blood and bone marrow microscopy showed nothing abnormal. Multiple cerebrospinal fluid studies since 1981 had shown total protein concentrations of 0.29 to 0.55 g/l, 14×10^6 to 46×10^6 mononuclear cells/l (>95% lymphocytes), and oligoclonal bands. IgG index was 1.0-1.7.

Analysis of anti-HTLV-I antibodies by electrophoretic immunoblotting. A: Serum from present patient diluted 1/700. B: Cerebrospinal fluid from present patient diluted 1/70. C: Serum from Japanese patient with adult T cell leukaemia diluted 1/1000. D: STLV-I antibody positive monkey (*Macaca fascicularis*) serum diluted 1/500. E: Second STLV-I antibody positive monkey serum diluted 1/500. F: Mouse monoclonal antibody to HTLV-I p24. G: Human negative control serum. Numbers denote positions of molecular marker proteins.

Anti-HTLV-I IgG activity, assayed by enzyme linked immunosorbent assay (ELISA),⁴ was strongly increased in serum (relative antibody activity 45, titre 1/2000) and cerebrospinal fluid (relative antibody activity 30, titre 1/200). (Relative antibody activities were calculated against a pool of HTLV-I antibody positive macaque serum.) The serum to cerebrospinal fluid antibody ratio was 10 and the antibody index (cerebrospinal fluid/serum antibody titre:cerebrospinal fluid/serum albumin concentration) 33 (normal <2), indicating intrathecal synthesis of anti-HTLV-I IgG. All serum samples were assayed simultaneously for antibodies reactive with an HTLV-I antigen and an uninfected cellular control antigen prepared in an analogous way. We found that the antibody activity could be absorbed out by T cells infected with HTLV-I but not by uninfected T cells. Several serum and cerebrospinal fluid samples obtained since 1975 showed essentially stable anti-HTLV-I values. Immunoblotting against purified HTLV-I antigen showed antibodies directed against the p19, p24, and p36 of HTLV-I. The antibody patterns in serum and cerebrospinal fluid were similar (figure) but distinct from the patterns of a patient with adult T cell leukaemia and two monkeys seropositive for simian T lymphotropic virus type I (STLV-I). Anti-HTLV-I was not found in the patient's wife or two children.

Antibody titres to several other relevant viruses, including human immunodeficiency virus, were normal or negative, as were the results of screening for other infectious diseases. No complement fixing antibodies to human brain or peripheral nerve were found in serum or cerebrospinal fluid. The skin tuberculin reaction was positive. The T4:T8 ratio was 1.4. The patient's lymphocytes had a

normal phytohaemagglutinin response, but the unstimulated thymidine uptake was high (11 600 counts/min; normal <1000), suggesting spontaneous lymphocyte activation.

Comment

Tropical spastic paraparesis associated with HTLV-I was the probable diagnosis in our patient. This has not been reported in an east African before, and in Europe has only recently been found in a few cases, most of them of West Indian origin.⁵

Our patient is of special interest because of the extended serological records and evidence of lymphocyte activation. The pronounced intrathecal synthesis of anti-HTLV-I antibodies suggested the presence of the virus in the central nervous system. The mechanism by which HTLV-I may cause tropical spastic paraparesis in a small portion of the large number of people infected with this virus, however, is unknown.

We thank Dr Torgny Hallberg for the lymphocyte studies.

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Horner's syndrome after manipulation of the neck

Manipulation is a safe and effective means of relieving painful mechanical problems of the spine including the neck. As with all treatments, however, things may go wrong, and neurological damage from manipulation of the cervical spine has been reported before.¹ I describe a case of Horner's syndrome after chiropractic manipulation of the cervical spine in a fit man with no known contraindications.

Case report

A 45 year old chiropractor with a history of intermittent neck pain for several years that had been relieved by manipulation went to a chiropractor complaining of pain at the base of the neck on the left side, which had been present for three weeks. The painful area was manipulated, the chiropractor making a sharp downward thrust with both thumbs placed deeply at the base of the neck on the left posterolaterally while the patient lay on his right side. This movement was repeated once or twice, and the patient felt a "crunch."

The local soreness increased immediately, and while the patient was on his way home a severe headache and a pain "like migraine" developed in the left eye. He felt sick and went to bed. Next morning he still had pain on the left side of the head and around the left eye, and the left eyelid was drooping and the left pupil smaller than the right. During the next few days the left side of his face and forehead did not sweat like the right. The neck pain and headache gradually improved, as did the ptosis, but the constricted pupil and the facial anhidrosis on the left side, with slight ptosis, persisted. No other neurological symptoms were evident, and a full neurological examination performed three weeks after the manipulation showed no abnormalities. Radiographs of the neck, including T1, and of the chest showed nothing unusual.

Comment

I have found only one other report of Horner's syndrome after manipulation of the neck, in a patient who had an occlusion of the right vertebral

artery, which resulted in multiple neurological symptoms on the right side in addition to Horner's syndrome.² The lesion was therefore in the central or brain stem portion of the cervical sympathetic supply, which invariably produces other neurological abnormalities as well as Horner's syndrome, such as hemihyperhidrosis, which my patient did not have. The absence of other neurological symptoms and signs, apart from transient headache and slight nausea, in my patient suggested that only the sympathetic nervous system was affected.

During manipulation the forceful thrusts at the base of the neck on the left may have caused a traction or avulsion injury to the white ramus communicans between the first thoracic nerve and the first thoracic or inferior cervical sympathetic ganglion, where it runs under and in front of the head of the first rib; the injury would have been due to forward movement of the left first thoracic vertebral transverse process. Thus the injury was probably in the preganglionic fibres. Accidental or iatrogenic trauma is the commonest cause of preganglionic Horner's syndrome,³ and traction injury to the preganglionic fibres, though mild and sustained rather than forceful and quick, was thought to have caused transient Horner's syndrome during an operation.⁴ Transient Horner's syndrome may result from ischaemia of the postganglionic fibres surrounding the carotid arterial supply.⁵

The fact that mishaps may occur should not result in use of manipulation being discouraged; nor does this report necessarily suggest that chiropractic manipulation is more hazardous than medical techniques, although the particular manoeuvre described here may be more liable to problems than some others. This is the first report of isolated Horner's syndrome occurring as a complication of spinal manipulation.

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Hazards of compression treatment of the leg: an estimate from Scottish surgeons

Compression has been used for centuries to manage varicose veins, the post-thrombotic syndrome, and leg ulcers, and also in modern times for preventing deep vein thrombosis. Recent improvements in compression bandages and hosiery allow pressures approaching 60 mm Hg to be sustained,^{1,2} with consequent narrowing of the margin between benefit and hazard.

We had observed several cases in which the injudicious use of compression in a limb with occult arterial disease had apparently led to severe skin necrosis and, in a few instances, to amputation. We therefore performed a survey to see if our colleagues had had similar experiences and to discover the extent of the problem.

Methods and results

All (154) consultants in general surgery in Scotland were asked in a questionnaire about the number of cases of ulcers or necrosis that they had seen in the past five years which were specifically induced or aggravated by compression bandages, elastic stockings, or anti-thromboembolism stockings. They were also asked whether any of these needed subsequent reconstruction of the arterial supply or amputation of the leg.

All 154 surgeons replied. Forty nine (32%) reported at least one case of damage induced by compression and 32 more than one. Analysis of the positive replies is shown in the table. The damage had necessitated reconstruction of the arteries in seven cases² and amputation in 12.

Comment

Like most effective treatments compression has certain dangers. This survey showed that about one third of surgeons in Scotland recognised damage of the leg that was caused by compression. This is probably a considerable underestimate for several reasons; the survey was retrospective

and depended on recollection, damage of this kind is often not recognised, and, most importantly, 80% of patients with leg ulcers are managed entirely in the community.³

Compression damage: positive responses

	No (%) of surgeons	No of cases
Ulcers/necrosis due to:		
Compression bandages	29 (19)	73
Elastic stockings	16 (10)	36
Stockings used to prevent embolism	17 (11)	38
Total	62 (40)	147

As many cases of damage were associated with compression bandages as with graduated support stockings and antiembolism stockings together. Several factors may be relevant. The application of a bandage is more dependent on the operator and the degree of compression more variable than with a stocking. Clinical indications are also different; in particular compression bandaging is widely used to treat leg ulcers. In a detailed study of 600 patients with chronic ulceration we showed that 22% had measurable impairment of the arterial supply to the ulcerated leg,³ and another survey showed such impairment in 31%.⁴ Our studies showed that the palpation of distal pulses in ulcerated and often oedematous legs is not always reliable. We recommend therefore that unless distal pulses of good volume can be felt, Doppler pressures should be measured in the ankle before treatment with compression. The compression of oedematous legs seems to be particularly hazardous.

Antiembolism stockings were associated with damage in 38 cases. We suggest that if the distal pulses are not palpable the manufacturer's warnings should be heeded and an alternative method of prophylaxis considered. Similar precautions should be taken with elastic stockings, which the survey showed to be associated with 36 cases of damage. Correct fitting and application of both types of stockings are essential.

We conclude that necrosis induced by compression is common in Scotland. While fully recognising the lack of precision in this study, we think that it is vital to draw attention to this serious complication because of the improvements in compression bandages and hosiery and the widespread use of stockings in the prophylaxis of thromboembolism.

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Corrections

A simple test to diagnose iritis

We regret that an editorial error occurred in this short report by Mr E Mark Talbot (3 October, p 812). In the figure the two lower photographs should be above the two upper photographs.

Fatal toxicity of antidepressant drugs in overdose

We regret that an editorial error occurred in this paper by Mr Simon Cassidy and Dr John Henry (24 October, p 1021). In table II, in the column headed No of deaths per million prescriptions (95% confidence intervals), the figure for lofepramine should be 0.0 and not 3.7.