Our findings are in direct contrast with those of Dundee *et al.*[2] A much larger study, of more than 200 subjects, would be required to rule out even a minor antiemetic effect of acupuncture (that is, a 10% difference between the groups). We chose, however, to limit our study to 46 subjects, as our results suggested that acupuncture at the P6 locus, in the way that we used it, is unlikely to be a clinically useful prophylactic for postoperative nausea and vomiting.

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(Accepted 26 August 1987)

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Facial flushing after intra-articular injection of steroid

Several systemic side effects of intra-articular injection of steroid have been described.[1-5] One of these is flushing, an unpleasant subjective sensation of warmth with erythema that affects the face and upper trunk. Whether it depends on sex, age, or disease has not been determined. Although it is generally considered to be a trivial complication[5] and has a reported frequency of less than 1%,[1] spontaneous complaints of flushing after injection of steroid from several patients led us to reassess the frequency and clinical importance of this reaction in a prospective study.

Patients, methods, and results

We studied 130 consecutive patients attending this unit who required intra-articular steroid injection of one knee. Before injection they were specifically questioned about vasomotor instability (perimenopausal flushing, flushing with alcohol, and easy blushing). Each knee was aspirated "to dryness" by a superolateral approach, and 40 mg triamcinolone acetonide was then injected under low resistance. The occurrence of nine possible adverse reactions, including flushing, in the seven days after injection was determined with a questionnaire provided at the time of the injection. Patients were not told that flushing was the primary interest. Between four and six weeks later they were seen again to clarify and confirm their answers. Patients who reported flushing and subsequently needed further intra-articular treatment of the same knee were injected with either triamcinolone acetonide 40 mg or triamcinolone hexacetonide 20 mg. Subsequent adverse reactions were assessed as before. Statistical comparison between the groups was made with the χ^2 test with Yates's continuity correction.

Assessment of reactions was not possible for 26 patients who lost or failed to complete the questionnaire or who gave inconsistent reports and for four others who responded positively to every question. All 30 were excluded from the analysis. The table shows the results for the remaining 100 patients, who had rheumatoid arthritis (36 patients), osteoarthritis (29), or pyrophosphate arthropathy (35). Flushing occurred in 40 and was serious in 15. It occurred at a mean interval of 19 (SEM 1·7; range 2-30) hours after injection and lasted for a mean of 36 (SEM 4; range 6-96) hours. It was associated with being female ($\chi^2=7·9$, $p<0·01$) but not with diagnostic category (rheumatoid arthritis $\chi^2=0·65$, $p>0·45$; osteoarthritis $\chi^2=0·24$, $p>0·45$; pyrophosphate arthropathy $\chi^2=0·41$, $p>0·45$), previous vasomotor instability ($\chi^2=0·19$, $p>0·45$), or age.

Twenty four patients who flushed needed a repeat injection in the same knee. All six who received triamcinolone acetonide reported flushing, whereas only nine of the 18 who received triamcinolone hexacetonide developed the reaction, which was reported to be less severe than before in all cases.

Details of patients studied

	Patients who flushed (n=40)	Patients who did not flush (n=60)	Total (n=100)
Mean (SEM) age in years and range	66 (1·7) 35-82	63 (1·7) 32-88	64 (1·2) 32-88
Ratio of men: women	1:3·4	1:1	1:2·2
No (%) of patients with:			
Rheumatoid arthritis	13 (33)	23 (38)	36 (36)
Osteoarthritis	11 (28)	18 (30)	29 (29)
Pyrophosphate arthropathy	16 (40)	19 (32)	35 (35)
No (%) with previous motor instability	6 (15)*	6 (10)*	12 (12)*

*All women.

Comment

Flushing was a common side effect of intra-articular steroid treatment, occurring in 40% of the documented patients (31% of the initial study group) and being unpleasant in 15% (12% of the initial group). The reaction was significantly more common in women ($p<0·01$) but seemed independent of age, disease category, or prior vasomotor instability. Previous underestimation of the frequency and severity of this response, particularly in women, is difficult to explain. Although in this study the patients were specifically questioned about nine side effects after the injection, the proportion who gave multiple positive responses was low. No other side effect was reported by as many as 40 patients, and the incidence of flushing was still high when all patients in the study were included (44/130; 34%). Every precaution was taken to ensure intra-articular rather than periarticular injection.

Flushing was reproducible within the small group of patients who were given a repeat injection of triamcinolone acetonide. Although not specifically designed to compare the frequency of side effects between different long acting steroids, the study suggested that the propensity to cause flushing may vary between different preparations. Whether the steroid, the side chain, or the vector is responsible for the flushing remains to be determined.

We thank the Arthritis and Rheumatism Council for financial support.

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(Accepted 26 August 1987)

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Tropical spastic paraparesis associated with human T lymphotropic virus type I in an east African naturalised in Sweden

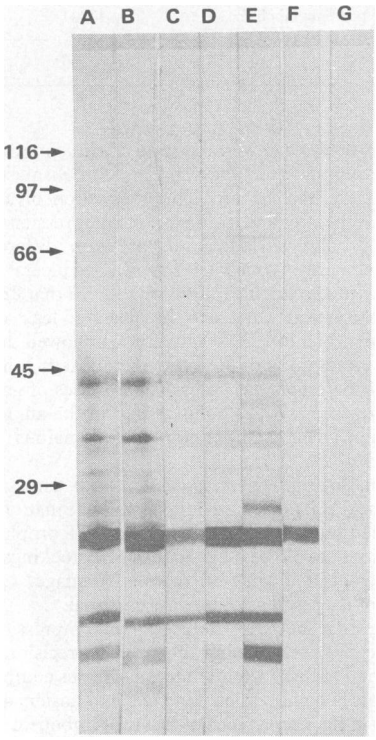
A slowly progressive myelopathy affecting the pyramidal tracts and to a minor extent other systems is known as tropical spastic paraparesis because of a characteristic geographical distribution.[1] Recent evidence suggests an aetiological role for the human T lymphotropic virus type I (HTLV-I) in this condition.[1,2] Though HTLV-I is widespread in Africa, tropical spastic paraparesis associated with this virus has been found in only one African patient, living in the Ivory Coast (west Africa).[3] We report a long record of antibodies to HTLV-I in an east African with tropical spastic paraparesis who had been resident in Sweden for 12 years.

Case report

A 31 year old Ethiopian had settled in Sweden in 1975. From the age of 16 he had had frequency and urgency of micturition and from the age of 21 he had

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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(Accepted 11 August 1987)

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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 chiropodist 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 back 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