long enough for the general practitioner to have referred them to hospital and for them to have been seen in the clinic. As most improvements occurred in patients with a short history of symptoms hospital patients would probably not show such a high rate of improvement.

The results suggest that most sufferers from back pain obtain relief without any specific treatment, and that mobilisation and manipulation may hasten this improvement but make no difference to the long-term prognosis. Moreover, although at the time more patients found the active treatment helpful, in retrospect they failed to distinguish active from placebo physiotherapy. For some patients with back pain prolonged or recurrent courses of mobilisation and manipulation would perhaps provide long-term relief, but an appropriate trial is needed. We were unable to identify any factor other than a short history that might indicate a good prognosis. This is disappointing, as prognostic markers would be helpful not only in managing the individual patient but also in leading to a better understanding of the problem.

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Copies of the tables may be obtained from Professor M I V Jayson, University of Manchester, Rheumatic Diseases Centre, Hope Hospital, Salford M6.

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SHORT REPORTS

Greater auricular nerve in diagnosis of leprosy

In its early stages leprosy is usually diagnosed on clinical grounds,[1] and great emphasis is laid on finding thickened nerves.[2] Indeed, in endemic areas thickened nerves are sometimes accepted as diagnostic of the disease.[3] An exact definition of what constitutes a thickened nerve is therefore of considerable importance. The greater auricular nerve is particularly valuable in this respect because as well as being often affected in leprosy it is said to be rarely palpable normally.[1]

Subjects, methods, and results

Three hundred and thirty-seven 17-year-old Nepali recruits to Britain's Brigade of Gurkhas were examined to find the frequency with which one or both of their greater auricular nerves were either visible or palpable. Their mean (±SD) height was 124±8 cm and weight 64·15±1·7 kg. They were examined with the head laterally rotated and the neck extended 20°. The nerve, when detectable, was then parallel with and 1·5 cm lateral to the external jugular vein. The whole body surface was examined in good light for other stigmata of leprosy, including hypopigmented patches and thickening of the ulnar, lateral popliteal, and other cutaneous nerves.

The greater auricular nerve was visible or palpable on one or both sides in 212 (63%) subjects and ranged in diameter from 2 to 4 mm. In 21, one or both ulnar nerves were considered thickened. None had detectable lateral popliteal nerves, and no palpable nerves were tender. Four had hypopigmented patches, not obviously due to tinea versicolor, but none of these was hypoaesthetic.

Comment

Most studies of the greater auricular nerves in endemic leprosy areas have been concerned with the prevalence of abnormally thickened nerves in leprosy in the absence of other signs, and this has been about 1·5%.[4,5] Despite a widely held opinion that the normal nerve is seldom palpable,[1] I was unable to find a study similar to this one, the purpose of which was mere detection of the nerve, thickened or not. The population studied perhaps favoured detection in that they were young, fit, and slim and had well developed neck muscles (perhaps a result of carrying heavy loads on "dokos," or wicker baskets, supported by a head band). Nevertheless, such a high rate of detection induces a note of caution in assessing the usefulness of this clinical sign. A further problem in distinguishing normal from leprous nerves has been a bulbous swelling, or perhaps kinking, of the nerve commonly present immediately as it emerges from behind the sternomastoid muscle to assume a subcutaneous position.

Leprosy is rare among soldiers of the Brigade of Gurkhas. There has been no new case since 1970. We may reasonably conclude therefore that the greater auricular nerve is often detectable in normal individuals from an endemic area and that its usefulness in the diagnosis of leprosy is thereby diminished.

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Behçet's disease and splenomegaly

Behçet's disease has been defined as recurrent oral and genital ulceration with iridocylitis.[1] It appears to be a systemic disease affecting skin and vascular, locomotor, central nervous, and gastrointestinal systems.[2] Two cases with splenomegaly are described. We did not find a cause for the enlarged spleen and we assumed that it was part of the disease spectrum.

Case reports

(1) A 29-year-old man presented in November 1976 with a five-year history of oral and scrotal ulcers, intermittent fever, and acne. Previous history included epilepsy diagnosed in 1969 and treated with phenobarbitone and phenytoin. On examination, he had multiple oral and scrotal ulcers, acneiform lesions of the face, and clubbing of fingers and toes. The spleen was palpable 15 cm below the costal margin. The liver was firm and enlarged 10 cm below the costal margin. The haemoglobin was 11·6 g/dl, the red cells showed anisopoikilocytosis, and the white blood count was 5·8 · 10^9/l (5800 mm^3) with a relative lymphocytosis. The ESR varied between 45 and 105 mm Westergren in the first hour. Liver function tests and prothrombin time were normal. Serum protein electrophoresis showed diffuse hyperglobulinaemia. The Paul-Bunnell toxoplasma and brucella tests were normal. Tests for antinuclear factor (ANF) were positive on two occasions and negative on three other occasions. Phenytoin was thought to be responsible for the positives. Tests for anti-DNA antibodies were negative. HLA types were A1, A10, B5, and B12. Growth hormone was normal. Oesophago-

gastroduodenoscopy was normal. Biopsy of rectal tissue was normal and negative for amyloid. Liver and spleen scan confirmed hepatosplenomegaly. Splenoportography showed normal splenic and portal veins with no varices. Liver biopsy showed aggregates of inflammatory cells around portal tracts. Splenomegaly is a feature not previously described in Behçet's disease. The possibility of the patient having a lymphoma led to a diagnostic laparotomy. The spleen weighed 1330 g but showed no specific histological abnormality.

(2) A 17-year-old girl presented in November 1973 with a 10-year history of oral and genital ulcers. She also complained of facial swelling, intermittent fever with temperature up to 40 C, and transient rashes. She had erythema nodosum-like lesions on her legs. On admission she had a tachycardia of 130 beats/min. The spleen was palpable 3 cm below the costal margin. Haemoglobin was 13·7 g/dl, white blood count $7{\cdot}5 \cdot 10^9$/l (7500/mm^3) with relative lymphocytosis, and ESR 20 mm in the first hour. The serum aspartate aminotransferase (serum AST; SGOT) was 70 IU/l. Alkaline phosphatase was normal. Chest *x*-ray examination showed peripheral areas of consolidation in both upper lobes. Paul-Bunnell toxoplasma and brucella tests were normal. The tachycardia, fever, and facial swelling settled within 24 hours of starting treatment with prednisone 40 mg/day. Over the course of the next year she was readmitted on four occasions with facial swelling, fever, tachycardia, and pulmonary infiltrates that always responded to prednisone in high doses. The splenomegaly persisted throughout the illness. On one occasion the spleen was palpable at the umbilicus, but rapidly regressed with prednisone treatment to 3 cm below the costal margin by the next day. Two years later, when aged 19, she was admitted elsewhere with an exacerbation of Behçet's disease, and subsequently died. At necropsy the spleen weighed 560 g: its histology was normal.

Comment

Gastrointestinal manifestations of Behçet's disease are ill-defined. Apparently the small intestine is most often affected.[3] Radiological examination of 85 Japanese patients, 50 of whom had symptoms such as diarrhoea, abdominal pain, and distension, showed dilatation, gas and fluid retention, and segmentation.[3] Lymphangiectasia of the small intestine and ulcerative colitis have been reported. But no gastrointestinal symptoms were recorded in a series of 41 patients from Israel.[2] The gastrointestinal tract was affected in three out of a recent series of 32 patients—one had Crohn's disease, another a resection of the caecum for a non-specific ulcer, and a third recurrent anal ulceration.[4] Two other cases of Behçet's disease and splenomegaly have been reported.[2 5] In both a cause other than Behçet's disease was considered responsible for the splenomegaly—namely, thalassaemia trait[2] and superior vena cava thrombosis.[5]

We thank Professor D O'B Hourihane, Trinity College, Dublin, for the necropsy details in case 2.

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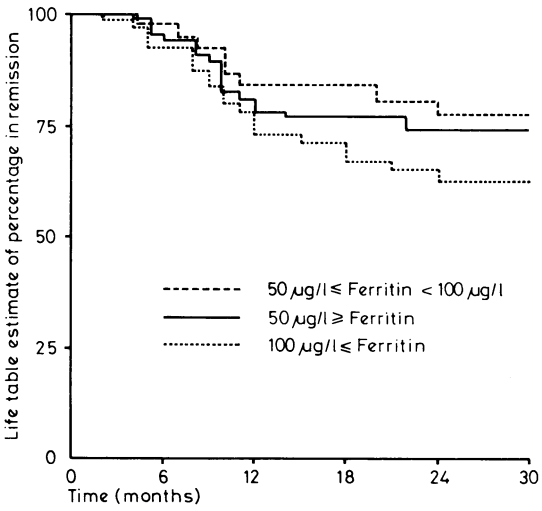
Serum ferritin during unmaintained remission in acute lymphoblastic leukaemia

In children with acute lymphoblastic leukaemia (ALL) serum ferritin concentrations are often raised at presentation before treatment. During chemotherapy much higher values may be reached but they often return to normal in patients who remain in remission after stopping treatment.[1] A raised serum ferritin concentration in ALL might therefore be useful as a guide to prognosis and as a sign of early relapse in patients in remission and off chemotherapy.[1] To test this hypothesis we carried out a prospective study of serum ferritin concentrations in patients with ALL in unmaintained remission.

Patients, methods, and results

Serum samples were obtained after the end of chemotherapy from all patients in the UKALL trials I-III throughout the United Kingdom between October 1974 and January 1977, and subsequent samples were requested at three-monthly intervals until the patients relapsed. A total of 202 patients were studied (116 males and 86 females). Their ages ranged from 2 to 28 years (mean 8·3 years). Serum ferritin concentrations were measured by immunoradiometric assay.[2] Serum iron, total iron binding capacity, and serum aspartate aminotransferase were also measured in each patient. The relationship between length of remission and initial serum ferritin concentration was investigated using the log-rank test.[3]

Out of the 202 patients in the trial, 53 (26%) relapsed during the follow-up period, which varied from 4 to 45 months. In 151 of them, including 42 who relapsed, an initial serum ferritin value was obtained within six months of stopping treatment. There was no significant difference between the distributions of initial values in those who relapsed and those who did not (Kolmogorov-Smirnov test $D = 0{\cdot}126$, $P > 0{\cdot}10$). The duration of remission was not related to the initial ferritin value when patients were divided into two groups using 50 or 100 μg/l as the dividing line ($\chi^2 = 0{\cdot}18$, $P > 0{\cdot}6$ and $\chi^2 = 1{\cdot}88$, $P > 0{\cdot}10$ respectively) (figure). When the 24 patients with serum ferritin concentrations greater than 200 μg/l were compared with the remaining 127 there was still no significant difference in relapse rate ($\chi^2 = 0{\cdot}06$, $P > 0{\cdot}8$). Two or more sequential ferritin measurements were performed in 156 of the 202 patients studied, including 36 who relapsed. There was a significant fall in ferritin concentration between the first and last measurement (sign test, $P < 0{\cdot}001$). In the 36 patients who relapsed the fall in serum ferritin concentration was continued when the two values immediately before relapse were compared (Wilcoxon matched pairs signed rank test, $P \simeq 0{\cdot}035$). The serum ferritin concentration was not related to age, sex, or serum iron or aspartate aminotransferase concentrations.



Life table estimate of percentage of patients in remission for patients with serum ferritin concentrations less than 50 μg/l (——), patients with serum ferritin concentrations between 50 and 100 μg (- - -), and patients with serum ferritin concentrations greater than 100 μg/l (....). Time scale represents months from cessation of chemotherapy.

Comment

In an earlier study[1] we showed that the serum ferritin concentration in children with newly diagnosed ALL was over 10 times higher than normal and that after the completion of successful chemotherapy the concentration fell to normal levels. The low concentrations found in long-term survivors suggested that the serum ferritin might be a useful prognostic indicator and aid in predicting relapse. The results of the present study provide no evidence that the serum ferritin concentration within six months after the beginning of an unmaintained remission in ALL is related to length of remission or that serial measurements of the concentration could be used in predicting relapse. One explanation for these negative findings may be that the ferritin assay using an anti-spleen ferritin antibody is not sensitive