good anti-herpes zoster virus antibody response she was thought to be immunocompetent. She was treated with intravenous acyclovir 10 mg/kg every eight hours for 10 days and improved rapidly. The pain from her skin lesions was greatly relieved, and after 48 hours of treatment she no longer had delusions or hallucinations and her ataxia was improving. After one week the skin lesions and ataxia had resolved, but partial abducent, facial, and vestibulocochlear palsies persisted.

Comment

Herpes zoster associated encephalitis was diagnosed in this patient from clinical features and the typical cerebrospinal fluid and electroencephalographic findings. Six months later she remained well, with no evidence of immunosuppression. Her abducent and facial nerve palsies resolved but the deafness in her left ear remained. Serum zoster titres decreased to 1/8.

Intravenous acyclovir has been effective in the treatment of severe cutaneous zoster in immunocompetent[1] and immunocompromised[2] adults. It has also proved effective in treating herpes simplex encephalitis.[3] There has not to our knowledge been a study of the use of acyclovir in herpes zoster associated encephalitis. Six patients with disseminated malignancy or other profound immunosuppression all improved dramatically within 24 hours of starting treatment with acyclovir.[4,5]

Our patient had no evidence of immunosuppression at diagnosis or afterwards and the dramatic clinical improvement was temporally related to the start of treatment with acyclovir.

1 Bean B, Braun C, Balfour HH. Acyclovir therapy for acute herpes zoster. *Lancet* 1982;ii:118-22.
2 Balfour HH, Bean B, Laskin OL, *et al.* Acyclovir halts progression of herpes zoster in immunocompromised patients. *N Engl J Med* 1983;**308**:1448-52.
3 Skoldenberg B, Alestig K, Burman L, *et al.* Acyclovir versus vidarabine in herpes simplex encephalitis. *Lancet* 1984;ii:707-11.
4 Steele RW, Keeney RE, Bradsher RW, *et al.* Treatment of varicella zoster virus meningoencephalitis with acyclovir and demonstration of virus in CSF. *Am J Clin Pathol* 1983;**80**:57-60.
5 Cheesbrough JS, Finch RG, Ward MJ. A case of herpes zoster associated encephalitis with rapid response to acyclovir. *Postgrad Med J* 1985;**61**:145-6.

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Resolution of diabetic cheiroarthropathy

We report on two patients with diabetic sclerosis or cheiroarthropathy in whom both skin and joint changes resolved completely. In one case this improvement seemed to be related to better diabetic control. This is the first time such improvement has been reported.

Case reports

The flexion contractures of the interphalangeal joints of the hands were assessed in the two patients by the method of Grgic *et al*,[1] which we use routinely in evaluating such diabetic patients. The patient's hands are placed palm down on a table top with fingers spread. These fingers are then viewed by the examiner at table level and the contact of the fingers with the table determined. Normally the entire palmar surface of the fingers makes contact. Patients are classified as having stage 1 disease if they are unable to make contact with some portion of one finger and stage 2 disease if unable to make contact with two or more fingers.

Skin thickness was assessed over the fingers as outlined by Siebold[2]: 0=R normal; 1+ = slight but definite thickening with inability to tent the skin between the examiner's thumbs; 2+ = mild to moderate changes; 3+ = severe thickening; 4+ = extreme thickening.

Glycosylated haemoglobin concentration was estimated using electrophoresis on cellulosic matrix (Glyco-Phore, Gelman Sciences Ltd) (normal range up to 8·5%).

Case 1—A 16 year old girl with an 11 year history of insulin dependent diabetes mellitus developed limited movement of all interphalangeal joints in her hands with associated tightening and thickening of overlying skin. She was classified as having stage 2 disease and skin thickness 2+. The symptoms developed after three months of poor diabetic control when she had a persistently raised blood glucose concentration and a haemoglobin A_1 concentration of 11%. With improved diabetic control the hand changes resolved completely over five weeks. She did not have any evidence of microvascular diabetic complications.

Case 2—A 58 year old man (interestingly, the father of the patient in case 1) with a 12 year history of insulin dependent diabetes mellitus developed a clinical picture similar to that of his daughter. He was classified as having stage 2 disease and skin thickness 2+. The symptoms occurred after five months of erratic diabetic control, although the haemoglobin A_1 concentration was only 9·7%. With improved diabetic control the hand changes resolved completely over five months. He did not have any microvascular complications.

Comment

Flexion contractures of interphalangeal joints are well described in insulin dependent diabetics, various series suggesting an incidence of 20-30%.[1-3] In some these contractures are associated with skin that is waxy or has a sclerodermatous appearance, and the terms diabetic sclerosis and diabetic cheiroarthropathy have been used to describe this.

Interest in the pathogenesis of this condition is heightened by suggestions that those who develop joint contractures are at an increased risk of developing microvascular diabetic complications.[3] Joint contractures, and more recently skin thickness, in these patients have been correlated with the duration of diabetes. Workers have suggested that these clinical changes reflect abnormal non-enzymatic glycosylation of collagen, and it has been shown experimentally in rats that these changes in liver, kidney, and haemoglobin are reversible. Such reversibility was not shown to occur in collagen.[4] This contrasts with the findings in our patients, in whom apparently typical skin and joint changes resolved completely. Resolution of joint contractures has not previously been described, although in a small study skin thickness measured by ultrasonography decreased with improved diabetic control.[5]

Any theory that is advanced to explain diabetic cheiroarthropathy must take account of the potential reversibility of these changes. Observations need to be performed on several occasions in affected patients to evaluate the clinical course more fully.

1 Grgic A, Rosenbloom AL, Webber FT, Giordano B, Malone GI, Shuster JJ. Joint contracture—common manifestations of childhood diabetes mellitus. *J Pediatr* 1976;**88**:584-8.
2 Siebold JR. Digital sclerosis in children with insulin dependent diabetes mellitus. *Arthritis Rheum* 1982;**25**:1357-61.
3 Rosenbloom AL, Silverstein JH, Lezotte DC, Richardson K, McCallum M. Limited joint mobility in childhood diabetes mellitus indicates increased risk of microvascular disease. *N Engl J Med* 1981;**305**:191-4.
4 McLennan S, Yue DY, Marsh M, *et al.* The prevention and reversibility of tissue non-enzymatic glycosylation in diabetes. *Diabetic Medicine* 1986;**3**:141-6.
5 Lieberman LS, Rosenbloom AL, Riley WJ, Silverstein JH. Reduced skin thickness with pump administration of insulin. *N Engl J Med* 1980;**303**:940-1.

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Alveolitis and haemolytic anaemia induced by azapropazone

Increasing numbers of drugs have been identified as causing pulmonary disease.[1] Non-steroidal anti-inflammatory drugs may seriously depress haemopoiesis, and immune haemolytic anaemia has also been reported.[2,3] We report three cases of both reversible pulmonary infiltration and haemolytic anaemia in patients who had been taking azapropazone for nine to 18 months.

Case reports

Case 1—A 60 year old woman with an 18 month history of rheumatoid arthritis was treated initially with azapropazone and indomethacin, but both drugs were stopped when a rash developed. After a short course of prednisolone azapropazone was restarted. Eight months later she was admitted to hospital with haemolytic anaemia, breathlessness, and widespread pulmonary crackles. Haemoglobin concentration was 98 g/l and reticulocyte count 22%. A direct Coombs test yielded a positive result. A chest *x* ray film showed bilateral pulmonary infiltrates. Azapropazone was stopped, and she recovered fully after treatment with corticosteroids. Fifteen months later azapropazone was restarted, but after three weeks she again became breathless with fresh pulmonary infiltration. She responded well to corticosteroid treatment after azapropazone was stopped, and one month later there were no abnormal clinical or radiological signs.

Case 2—A 70 year old woman presented with a three week history of breathlessness. She had received azapropazone 600 mg twice daily for nine months because of a painful osteoarthritic knee. She was breathless at rest, with