and phenytoin causing characteristic ataxia, dysarthria, nystagmus, and drowsiness. It is difficult to compare symptomatic chronic toxicity of these two drugs with a relatively short two to four year follow up. Both drugs seem to have similar effects on biochemical and haematological variables, although phenytoin but not valproate is associated with decreased red cell folate concentrations. We did not undertake psychometric testing in our patients, but other evidence suggests that phenytoin has a greater effect on cognitive function than does valproate.¹⁴ During this study none of the rarer and more serious adverse reactions to valproate occurred. In particular no patient developed evidence of hepatotoxicity related to valproate. The only serious adverse effect occurred in the phenytoin group and consisted of erythroderma and hepatitis in one patient.

There are few studies of the factors predicting the outcome of treatment in previously untreated patients. It is therefore of interest that the most dominant factor in outcome in both this study and that of Shorvon and Reynolds¹⁵ was the history of partial seizures. Shorvon and Reynolds also found that the presence of a neurological, psychiatric, or social disability had an adverse effect on prognosis. We did not find this to be the case, possibly due to the difference in definitions used.

Overall, there seems little evidence of any major difference in efficacy between valproate and phenytoin in adult patients with recent onset of epilepsy, irrespective of the type of seizures that the patients suffered. This study does not disclose any difference in the symptoms of toxicity between the two drugs. Although the five cases of early idiosyncratic reactions which required drug withdrawal were all in the phenytoin group, this was not statistically significant at the 5% level. The unusual pharmacokinetic properties of phenytoin make it a more difficult drug for routine use, necessitating small dosage increments and frequent assays of serum drug concentration. In comparison, the relation between dose and serum concentration for valproate is linear, and regular blood monitoring of monotherapy does not

seem to be critical. In women patients of childbearing age the choice of anticonvulsant drug remains difficult since both drugs appear to be teratogenic.¹⁶

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SHORT REPORTS

Necrotising tongue and skin lesions in temporal arteritis: follow up of a case with a possible iatrogenic factor

Temporal arteritis is a generalised vascular disease characterised by formation of granulomas (giant cell arteritis) which gradually constrict the lumen.¹ Arteries of medium and small size are those mainly affected, the site of predilection being within the cranial vascular territory. Cases with necrotising lesions of the scalp, skin, and tongue have been reported, probably resulting from massive disease of branches of the external carotid artery. We describe these complications in a patient with histologically proved disease² and review published work to see how often they have been reported and under which circumstances they occurred.

Case report

A previously well 69 year old woman was admitted to hospital with a two month history of muscular pain, particularly in the shoulders, neck, and jaw. On examination she appeared fit and did not have headache or visual disturbances. The temporal arteries were slightly tender. Pulsation was normal. Sedimentation rate and plasma fibrinogen concentration were substantially increased. Ophthalmological findings were normal. A biopsy sample was taken from the right temporal artery under local anaesthetic containing a vasoconstrictor. Six hours later she complained of hypoaesthesia of the tongue and interior right side of the jaw and impaired movement of the tongue. A nasogastric tube was inserted because of difficulty with swallowing. She was given large doses of corticosteroids and improved within 24-48 hours.

The histological picture was giant cell arteritis. During the following days she developed anaesthesia of the right lower lip, inferior gingiva, and tongue, which became cyanotic. After nine days most of the tongue was necrotic

together with the right lower lip. Tracheostomy was performed to prevent aspiration of the sloughing tongue. Revision of the tongue was postponed until demarcation had taken place six weeks later. Most of the alveolar process of the mandible was also necrotic. In addition, she developed necrotic lesions on the scalp, which were left to heal spontaneously.

The necrotic area of the right lower lip healed with severe shrinkage of the lip and fixation to the floor of the mouth. The lip was corrected by an Abbé operation and two months later a sulcoplasty was performed with a free graft of split skin. The patient was given a new denture for the lower jaw. Later the prosthetic treatment was completed with a tongue obturator fixed to a palatal plate. By these means she could speak adequately.

Four months later the patient was in excellent health and the tracheostomy was closed. Problems with swallowing still necessitated a feeding tube, but after a year this was removed; she remained, however, able to take only a blended diet. She was followed up regularly as an outpatient for five and a half years and felt well taking a continual low dose of prednisone (5 mg daily).

Comment

Forty nine cases of necrotising lesions of the scalp, facial skin, and tongue have been reported, and in 29 of these there was histological proof of giant cell arteritis. In two of these cases the necrotising lesions within the cranial vascular territory developed shortly after biopsy under local anaesthesia.³⁻⁵

Plainly, necrotising lesions may develop spontaneously in cases of advanced vascular changes of a constrictive-thrombotic nature. It seems reasonable to assume that tissues relying on a critical blood supply may undergo necrosis after an arterial spasm, retrograde in character, with thrombotic occlusion of all collateral and communicating vessels triggered by the vasoconstrictor in a local anaesthetic. Our patient showed symptoms from the cranial vascular territory with pain in the neck and jaw. The diagnosis was plain clinically, and the delay in instituting steroid treatment was probably an important factor in the development of severe necrosis.

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Erythema nodosum and circulating immune complexes in acne fulminans after treatment with isotretinoin

The syndrome of acne fulminans includes severe ulcerating cystic acne, fever, and arthralgia.¹ Other documented features include microscopic haematuria, an increased erythrocyte sedimentation rate and white cell count,¹ anaemia,² myalgia,¹ and, in one case, erythema nodosum.³ Circulating immune complexes have not previously been reported, although their presence is suggested by the findings of arthralgia, microscopic haematuria, and erythema nodosum.

In the past three years we have seen three patients with erythema nodosum and acne fulminans, which in two cases appeared to have been triggered by treatment with the recently introduced analogue of vitamin A, isotretinoin. Evidence of circulating immune complexes was sought in these two patients, and we report our findings.

Case reports

Case 1—An 18 year old man had had acne for several years which had recently become resistant to conventional treatment. He was treated with isotretinoin 0.5 mg/kg daily, increasing after four weeks to 1 mg/kg daily. Three weeks later he was admitted to hospital with fever, crusted and ulcerating lesions on his chest, and erythema nodosum on his shins. Investigations showed the following abnormalities: erythrocyte sedimentation rate 70 mm in the first hour; haemoglobin concentration 11.9 g/dl; white cell count $11.5 \times 10^9/l$, neutrophils 80%; platelet aggregation test for circulating IgG immune complexes yielded a positive result on two occasions at 1/160 dilution (normal less than 1/20); and faecal occult blood was weakly positive on three consecutive occasions. Results of latex and sheep cell agglutination tests, antinuclear factor titre, and results of urine microscopy were normal or negative. *Propionibacterium acnes* was isolated from acne lesions.

Case 2—A 21 year old Asian man with severe nodulocystic facial acne unresponsive to oral antibiotics was treated with isotretinoin 1 mg/kg daily. His acne became progressively more inflamed over the ensuing three weeks, with the development of arthralgia, lesions similar to those of pyogenic granuloma on the chest,⁴ and erythema nodosum on his shins. The following abnormalities were found: erythrocyte sedimentation rate 50 mm in the first hour; white cell count $14.3 \times 10^9/l$, neutrophils 67%; platelet aggregation test for circulating IgG immune complexes yielded a positive result on two occasions at 1/160 dilution (normal less than 1/20); and serum IgG concentration was 21.7 g/l (normal 8.0-18.0 g/l). Microscopy of urine yielded normal results.

In both cases symptoms responded rapidly to moderate doses of prednisolone (25-30 mg daily). Improvement was accompanied by a fall in the titre of circulating immune complexes to normal values within four weeks. Isotretinoin was continued at 1 mg/kg daily and achieved excellent results in three to four months.

Comment

Acne fulminans is a rare complication of severe acne. The two patients reported on here developed the condition after starting treatment with isotretinoin. Although acne does commonly worsen in the first few weeks of treatment with isotretinoin,⁵ we are not aware that it has previously been implicated as a precipitant of acne fulminans.

Circulating immune complexes are found when erythema nodosum

occurs in other conditions such as sarcoidosis, and their presence has been predicted in acne fulminans.² On the basis of the clinical findings in a patient with acne fulminans and erythema nodosum Williamson *et al* postulated that an Arthus reaction was taking place.³ They were able to show an exaggerated response to an intradermal injection of *P. acnes* suspended in saline, and suggested that this antigen might also play a part in the Arthus reaction. They did not, however, show the presence of circulating immune complexes. Using the platelet aggregation test, a reliable method for detecting soluble IgG complexes, we confirmed that circulating immune complexes may be found in acne fulminans.

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Is a local anaesthetic necessary when using fine gauge spinal needles?

Over the past 100 years since Bier introduced spinal anaesthesia in man needles for this procedure have improved in design, becoming finer and sharper. The use of fine gauge needles lessens the risk of headache after lumbar puncture.¹ The most commonly used sizes are 22 gauge, 23 gauge, and 25 gauge. The use of 25 gauge needles reduces the incidence of headache after lumbar puncture to less than 2%.² The 25 gauge needle is flexible, however, and the use of an introducer is recommended.

Drummond and Scott have shown that needles may be more accurately directed in the midline if the bevels of the needles face caudally rather than laterally.³ This method has been followed by one of us (GRP), who finds it unnecessary to use an introducer in routine clinical practice with 25 gauge spinal needles.

To make insertion of a spinal needle as painless as possible it is almost universal practice to infiltrate the skin with a small volume of local anaesthetic using a 25 gauge intravenous needle. This is painful owing to the stinging sensation of the local anaesthetic. It seems unnecessary to use infiltration anaesthesia if a lumbar puncture is performed with a 25 gauge needle alone. As discrimination of needle size in the lumbar region is said to be poor, it may be unnecessary to use infiltration anaesthesia for sizes other than 25 gauge.

We have tried to compare the pain caused by the insertion through the skin of spinal needles in common use to see if they can be discriminated and to see if infiltration anaesthesia is necessary with the use of fine needles.

Subjects, methods, and results

Twelve anaesthetists from this department, of all grades and both sexes, volunteered for the study. The 10 cm visual analogue scale⁴ was explained to them. The conventional limits of the scale are no pain at all and the worst pain you could possibly imagine. The scale was modified so that the upper limit of pain was the pain caused by the insertion of an 18 gauge spinal needle (Becton Dickinson, España, SA) through the skin without local infiltration anaesthesia. This avoided crowding at the lower end of the scale.

An area 15×15 cm around the spinous processes of L3 and L4 was cleaned using a chlorhexidine spray (Dispray, Stuart Pharmaceutical) and dried with a gauze swab. A previous study of 10 volunteers had shown sensitivity over the spinous processes to be no different from that of skin more laterally. An 18 gauge spinal needle was introduced through the skin to give the upper limit of the visual analogue scale. The same investigator then inserted