A case of Bell’s palsy in a child of four months


Edinburgh

Although unilateral facial paralysis is a common condition, it is unusual in children especially under the age of two years. The following case admitted to the paediatric ward in Perth Royal Infirmary is therefore thought to be sufficiently unusual to warrant reporting.

Case history

The child was a plump, well-nourished, male infant of four months, weighing 17 lbs 1¼ oz. His mother had noted a right-sided facial paralysis for the first time six hours before he was admitted to the ward. Two weeks previous to the child’s admission, his sister, aged two years, was noted to have had a cold, as had his mother, aged 27 years, one week later. His father, a 34-years-old farm worker, had complained of pain and swelling of the left side of his face five days before the child was admitted, but this had never been very marked, had disappeared within 24 hours and had not caused any paralysis. There was no history of the child having been exposed to draughts or excessive cooling prior to the onset of the paralysis.

Of a healthy family consisting of mother, father and sister, the child was born by spontaneous vertex delivery and weighed 7½ lbs at birth. The subsequent development was uncomplicated and he had thrived well on National Dried Milk. At four months, he was able to sit up with assistance and take a lively interest in his surroundings.

On examination, the child was seen to be fit and alert. There was little evidence of paralysis when the child was at rest, although it was noted that there was a slight droop to the right corner of the mouth, that the right eye appeared wider than the left and the eyebrow higher. When, however, he was encouraged to smile or stimulated to cry, a complete right-sided facial paralysis was evident (see photo). The right ear drum was noted to be red, but there was no lymphadenopathy. The fauces and left ear drum were not injected. No other abnormality was present apart from a soft systolic murmur at the apex.

While in the ward, the child remained well and was in no way disabled by the palsy although the right eye remained open when he slept. There were no feeding difficulties.

Throat and rectal swabs were both negative. A swab from the right ear grew coagulase negative staphylococci. The haemoglobin was 87 per cent and the w.b.c. 9,900 cell/cm. A lumbar puncture was carried out but both the dynamics and the chemistry were normal, as was the cell count. Examination of the urine was satisfactory although on two occasions ‘cleaned up’ specimens were found to contain a significant number of organisms. No pus cells, however, were found and, as the child was afebrile and well, no action was taken on this account.

To counteract the possibility of an infective condition of the middle ear, two eight-day courses of intramuscular penicillin were given. A further 14-day course of oral tetracycline was given but had little effect on the infected ear drum. He was also treated with ephedrine nose drops and ‘otosporin’ ear drops. Physiotherapy was confined to facial massage and no galvanism was employed.

He was discharged from the ward 36 days after admission and followed up as an outpatient. In three months, there was a marked improvement but a right facial paresis was still evident. After six

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months, when aged ten months, he weighed 24 lbs and was well. Apart from a slight droop to the corner of the right eye, no evidence of the facial paralysis remained and his smile was noted to be equal on both sides.

Discussion

Bell's palsy is defined by Kettel as a collective diagnosis for all cases of peripheral facial paralysis in which it is impossible to demonstrate a local lesion. At present it is considered to be caused by arteriolar spasm in the vasae vasorum supplying the nerve which becomes oedematous and is compressed within the facial canal (Hilgar). Miehlke describes this to be the site when there is no eye movement disability, no apparent vestibular disturbance, and no extra temporal lesion evident. Previously, all cases were considered to be caused by a non-suppurative otitis media, whether the drum head was seen to be involved or not and at one time a tuberculous infection of the middle ear had always to be kept in mind. Kettel noted necrosis in the mastoid process cells in 11 out of 50 cases in which he exposed the nerve but found no round-cell infiltration. He concluded that if the ear drum was injected in a case of Bell's palsy this must be part of the general vascular change and not necessarily of infective origin. It is probable in this case that the antibiotics used were unnecessary, especially considering the absence of other manifestations of infection.

Many different forms of therapy have been described. Early use of corticosteroids to relieve the vascular spasm and surgical intervention to relieve the compressed nerve have been advocated. 75–85 per cent of cases, however, make a good recovery with no treatment. Apart from the antibiotic therapy, conservative methods were used in the treatment of this child.

The underlying aetiology in Bell's palsy is still obscure but exposure to a local or general cooling influence is thought to be connected. No unusual cooling, however, was noted in this case.

In a review of the literature on Bell's palsy and facial paralysis, the youngest child reported was two years old, although this does not include the facial paralysis at birth from obstetric manipulation and trauma.

Summary

A report is given of what appeared to be a typical Bell's palsy in a male child four months old. No instance of this condition in so young a child has been reported in the journals. The causation and treatment of Bell's palsy is reviewed.

REFERENCES


A note on primary adult hypothyroidism in a Scottish rural practice

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Two circumstances have aroused the writer's interest in this problem. (1) an apparent absence of accurate information about the extent of the disease in Scotland; (2) the discovery of ten examples of primary adult hypothyroidism in a relatively small rural practice in central Scotland over the period December 1961 to March 1968. The term primary adult hypothyroidism is preferred to myxoedema, which is in fact the final phase of hypothyroidism: it may be that the picture of gross myxoedema imprinted on our minds from student days has retarded our