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Recurrent lower motor neurone facial paralysis in four successive pregnancies

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Abstract
Recurrent lower motor neurone facial paralysis in successive pregnancies is a rare phenomenon of which there are few case reports in the literature. There have been two previous reports of recurrent Bell's palsy in two and three successive pregnancies respectively, but it is felt that these should have more appropriately been reported as recurrent lower motor neurone facial paralysis in pregnancy as, by definition, any facial paralysis that recurs should not be classified as Bell's palsy even though Bell himself commented on the association between facial paralysis and pregnancy. We report the first case of recurrent lower motor neurone facial paralysis in four successive pregnancies and review the literature.

Key words: Facial paralysis; Pregnancy

Case report

First pregnancy

A 19-year-old Caucasian primigravida presented in the antenatal clinic at eight weeks gestation with a previous history of grand mal epilepsy between the ages of 11 and 16 years. Anticonvulsant medication was stopped at the age of 16 years and she had suffered no recurrence. Combined oral contraceptive pills had been used up until December 1986. General clinical examination was unremarkable. Investigations revealed she was Rubella immune, Pku normal, Khan test negative and blood group O Rhesus positive.

The pregnancy was uneventful until she was admitted at 29 weeks gestation with a history of sudden onset of loss of taste, deafness on the left side followed by hyperaesthesia of the face 48 hours later. She had been treated with prednisolone and amoxycillin by her GP.

Clinical examination revealed a left-sided lower motor neurone facial nerve palsy. There was absence of any other neurological deficit and other clinical findings were normal. Prednisolone therapy was discontinued and the pregnancy continued uneventfully although the facial palsy persisted.

Following spontaneous labour and normal delivery of a live female infant weighing 2830 gm at 40 weeks gestation, the palsy resolved spontaneously and completely three days postpartum.

Second pregnancy

The patient was 20-years-old and had suffered no recurrences since the last episode. Contraception was not practiced and she became pregnant two months after her last delivery. She was admitted with similar symptoms as in her first pregnancy, at 12 weeks gestation. Examination revealed lower motor neurone lesion of the right facial nerve. The other cranial nerves were intact, corneal reflexes were maintained and the optic discs were normal. Chest and skull X-rays did not reveal any abnormality, random blood sugar was 4.0 mmol/l and the erythrocyte sedimentation rate (ESR) was 22 mm per hour (Westergren). She was managed conservatively but the palsy worsened as the pregnancy progressed and she required application of tapes to keep the right eye closed when sleeping.

Following spontaneous labour and normal vaginal delivery of a live male infant weighing 3340 gm at 39 weeks gestation, the palsy completely resolved two days postpartum.

Third pregnancy

Combined oral contraceptive pills were used until July 1989 and there was no recurrence of the facial palsy in between pregnancies. The antenatal period was uneventful until she developed right-sided lower motor neurone facial paralysis at the 29th week of pregnancy. Clinical examination did not reveal any other abnormality. Her random blood sugar was 4.5 mmol/l. She was managed conservatively and was admitted at the 36th week of pregnancy with a history of cough with production of sputum, nausea, vomiting and generalized body aches. She was febrile and following clinical examination, a diagnosis of acute upper respiratory tract infection was made and treatment commenced with ampicillin.

Foetal distress developed and she was delivered of a live male infant by emergency lower segment Caesarian section. On the second post-operative day, she developed herpetic lesions on the lips, nose and nostrils which were treated with Zovirax cream. By the sixth post-operative day, the facial palsy had completely resolved and the herpetic lesions were almost completely healed.

Fourth pregnancy

There had been no recurrence following her last episode. The combined oral contraceptive pill was used until pregnancy was confirmed in October 1991. She was booked for antenatal care at 16 weeks and random blood sugar was 4.4 mmol/l. Lower motor neurone paralysis developed with hyperacusis and altered taste on the left side a few days later and was managed conservatively throughout pregnancy as it had always resolved spontaneously following delivery.

She had an uneventful labour with spontaneous vaginal delivery of a live female infant weighing 3790 gm at 39 weeks gestation. The facial palsy slowly resolved over a few weeks and when laparoscopic sterilization was performed five months postpartum, there was no evidence of a residual lesion.

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Discussion

The association between pregnancy and facial palsy was first suggested by Sir Charles Bell (Bell, 1830). The frequency of Bell's palsy in pregnancy and the puerperium ranges between 41 per 100 000 deliveries (McGregor *et al.*, 1987; Falco and Eriksson, 1989) and 45.1 per 100 000 births (Hilsinger *et al.*, 1975) which is much higher than 17.4 per 100 000 per year for all women of child-bearing age. Hilsinger *et al.* (1975) calculated that pregnant women were 3.3 times more likely to suffer from this condition than their age-matched colleagues.

Different authors have quoted the rates of recurrent Bell's palsy in the general population as 10 per cent (Mamoli *et al.*, 1977) 15.2 per cent (Boddie, 1972) and 0.5 to 10.4 per cent (Yanagihara *et al.*, 1984). El-Ebiary (1971), studying 580 cases of facial paralysis, quoted the recurrence rate to be twice as high in females as in males. However, recurrent 'idiopathic' facial palsy in successive pregnancies appears to be very rare and to date, there has been only one report each of recurrent Bell's palsy in two (Deshpande, 1990) and three successive pregnancies (McGregor *et al.*, 1987). It is however currently felt that these should have been more appropriately recorded as recurrent lower motor neurone facial paralysis in pregnancy as by definition, any facial paralysis that recurs should not be classified as Bell's palsy, more so in this case where recurrences only occurred in pregnancy, even though Bell himself commented on the association between facial paralysis and pregnancy.

The aetiology of Bell's palsy in pregnancy, a mononeuropathy of lower motor neurone type of sudden onset, remains controversial. Various theories have been propagated albeit without proof and these have included: fluid retention, reactivation of herpes simplex virus infection within the VIIth nerve ganglia, gestational immunosuppression, unknown changes induced by oestrogen and or progesterone, vascular thrombosis or spasm (Robinson and Pou, 1972; Hilsinger *et al.*, 1975; Nobori *et al.*, 1981).

While the relationship between idiopathic Bell's palsy and high blood pressure in the general population is established, there is no statistical evidence in support of such a relationship in pregnant women (Abraham-Impijn *et al.*, 1982). Also diabetic neuropathy does not appear to be a causative factor in pregnant women when compared to the general population (Korczyn, 1971).

Adour *et al.* (1975), reported that 100 per cent of their patients with Bell's palsy had antibodies to Hérpes simplex virus. It is of interest that this patient developed herpetic lesions in her third puerperium, an occurrence which tended to lend credence to the aetiological theory of reactivation of herpes simplex infection within the VIIth nerve ganglia due to immunosuppression by increased cortisol levels of pregnancy (Hulka and Mohr, 1969; Jenkins, 1977; Gleicher and Siegel, 1980). It is also known that pregnant women demonstrate an increased susceptibility to infection by herpes simplex virus and perhaps other viruses (Anderson *et al.*, 1974). Why she did not develop herpetic lesions in the first two and last pregnancies remains a matter for conjecture.

Treatment remains supportive as neither surgical decompression nor steroid administration has been consistently demonstrated to be effective (Adour, 1975; Wolf *et al.*, 1977; Huizing *et al.*, 1981). However, some workers have shown a good response to treatment with corticosteroids especially when used within 24 hours of onset of the condition and the dose tapered off over about a week. Although the use of high doses of corticosteroids in pregnancy is fraught with the potential risk of development of adrenal suppression, cleft lip and palate in the foetus, Hilsinger *et al.* (1975) did not experience foetal complications in 31 children of mothers with idiopathic facial paralysis treated with 40–60 mg of prednisolone daily.

There are no published figures for the prognosis of recurrent Bell's palsy in pregnancy but as in our patient, the prognosis is generally good, if the initial paralysis is partial, and fair if it is complete (Adour, 1975) and does not appear to be different from that of the nonpregnant female population.

On a lighter note, the two episodes of right-sided lower motor neurone facial paralysis were associated with the births of male infants while the two left-sided episodes were associated with births of female infants. Laparoscopic sterilization in this patient precludes us from ascertaining the relationship between the affected side in lower motor neurone facial paralysis in pregnancy and the sex of the baby.

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