Brief Report

Bell's Palsy in Pregnancy and the Puerperium

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The incidence of Bell's palsy is significantly higher during the last trimester of pregnancy and the puerperium. Suggested explanations for this association include fluid retention, hypertension, compromise of the vasa nervorum, infection (particularly with herpes simplex virus), and an autoimmune process. The diagnosis is confirmed by identifying lower motor neurone paralysis and excluding secondary causes for the symptom complex. The majority of cases resolve spontaneously. Recovery may be delayed or incomplete in older patients and those with recurrent episodes or severe initial symptoms. The role of diuretics, steroids, or surgical decompression in treatment of pregnancy-related cases of Bell's palsy has not been well studied.

Key words: Facial paralysis; pregnancy complications; puerperium; diagnosis, differential.
(*J Fam Pract 1993; 36:559-563*)

Bell's palsy is lower motor neurone paralysis of the seventh cranial (facial) nerve of unknown cause. This idiopathic form is the most common type of facial paralysis, accounting for approximately 80% of all facial nerve palsies.[1]

In 1830, Sir Charles Bell, the surgeon for whom the condition was named, suggested that there was an increased incidence of this condition in pregnant women.[2,3] Subsequent studies have estimated that the incidence of Bell's palsy in women who are pregnant or who have recently given birth is approximately three times its incidence in nonpregnant women of the same age group.[3,4] The condition is, however, rare. Estimates of the incidence range from 38 to 45 cases per 100,000 deliveries[3] compared with approximately 17 cases per 100,000 per year for women of childbearing age. This is equivalent to approximately one case per 2600 pregnancies.[2,3]

The risk for Bell's palsy, however, is not equally distributed throughout pregnancy, but is much higher in the third trimester and early puerperium. In the largest series reported, Bell's palsy occurred in 31 of the 42 patients in the third trimester, and 5 in the first 2 weeks postpartum.[6] In a second series of 18 patients, Bell's palsy developed in 12 patients in the third trimester and 6 within the first 2 weeks postpartum.[3] It has been calculated that the incidence of Bell's palsy in women during the final trimester and early puerperium is 118 cases per 100,000 women per year, or approximately six times that of nonpregnant women.[2] Although all of these estimates are imperfect because of the relatively small numbers of cases and problems in defining the comparison populations,[5] Bell's palsy may be encountered by family physicians involved in both prenatal and postpartum care.

As the sudden appearance of neurological signs in women around the time of childbirth can be alarming, awareness of Bell's palsy is important. The following cases illustrate this usually benign condition.

Illustrative Cases

Case 1

A 26-year-old primigravid patient reported to the emergency department at 37 weeks' gestation with symptoms of left-sided facial paralysis, which she first noticed on waking that morning. The patient was very distressed, and hyperventilated during the initial assessment. Once

Submitted, revised, November 24, 1992.

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ISSN 0094-3509

calmed, she gave a history of a mild head cold that had lasted for 2 to 3 days. On the previous evening she had noticed a "pins and needles" sensation on her left cheek, which she attributed to sitting next to an open window during a car trip that lasted about 1 hour.

On examination, she was found to have moderate left-sided facial weakness with drooping of the mouth and difficulty in closing the left eye. She had no abnormality of lacrimation, taste, or salivation but did report numbness on sensory testing of the left side of the face. The remainder of the physical examination was normal for her stage of gestation. There was no additional relevant information from social, family, or personal history.

The patient's family members were very distressed by her symptoms and requested neurological consultation. The neurologist confirmed the diagnosis and concurred with the plan to observe the patient closely in anticipation of spontaneous recovery. Her symptoms had almost completely resolved by the time she gave birth at 39.5 weeks' gestation. She has had no sequelae and no recurrence, even during two subsequent pregnancies.

Case 2

During a routine postpartum examination 2 weeks after delivery, left-sided facial weakness was noted in a 13-year-old female patient. Neither the patient nor her mother expressed concern about the symptom since a close relative had completely recovered from what they termed "Belling's palsy." The patient reported that the symptom had been present for about 1 week and had greatly improved from the initial manifestation of complete facial paralysis. At the time of examination, the only physical symptom found was mild facial weakness. The patient was asked to return 1 week later. At the time of the follow-up visit, she was completely asymptomatic, and she has had no subsequent episodes of facial weakness.

Etiology and Pathology

The etiology of Bell's palsy is unknown. The clinical symptoms may result from any compromise of the nerve, thus Bell's palsy may be caused by several different pathological processes acting alone or in combination.

It has been presumed that the basic lesion is inflammation and demyelination of the nerve close to or within the bony canal.[2] One group of hypotheses attributes this inflammation to a mechanical process. Pregnant women are thus at increased risk for developing Bell's palsy because of fluid retention, which causes perineural edema.[2,7] If this explanation is correct, Bell's palsy is similar to carpal tunnel syndrome and other nerve compression conditions. Correlation between the time of peak incidence of Bell's palsy and the time the maximum extracellular volume occurs during pregnancy supports this theory.[3]

Increased blood pressure could contribute to increased extracellular fluid volume or cause nerve compromise through other mechanisms such as vascular spasm, microemboli, or thrombosis of the vasa nervorum.[3,8] In the general population, hypertension is associated with Bell's palsy, and in one study, pregnant women who developed Bell's palsy were six times more likely to be preeclamptic than other pregnant patients.[3]

An alternative hypothesis is that Bell's palsy is caused by an infection either directly or by initiating an autoimmune process. Evidence supporting this hypothesis includes the observation of a seasonal increase in Bell's palsy occurring in the last 3 months of the year[10] and reports of clusters of cases.[2] Some cases that were previously diagnosed as Bell's palsy (ie, idiopathic), however, may have been caused by Lyme disease.[9] Most interest in infectious etiology has focused on herpes simplex or similar viruses. Patients are reported to show significantly higher rates of antibodies to this group of viruses,[3,4,7] and herpes simplex virus is commonly either acquired or reactivated[2,3] during pregnancy.

Finally, it has been suggested that Bell's palsy is a *forme fruste* of Guillain-Barré syndrome, since some patients have been shown to have biochemical and electroneuronographic abnormalities of several cranial nerves.[2] This could in turn be linked to infectious or mechanical triggers of the acute neuropathy.

Clinical Features

Since Bell's palsy in pregnancy is relatively rare, clinical information is available only from small series of patients and case reports. The paralysis appears to occur, follow the same clinical course, and have a similar outcome as in nonpregnant patients.[2,7] The onset of symptoms is sudden, and the maximal deficit is reached within hours.[1,11] In retrospect, many patients report cold injury to the face or symptoms of otalgia[1] or upper respiratory tract infection before the onset of paralysis, as in the first case. The significance of these reports is not known.

Paralysis may involve either side of the face. In one series, the left side predominated,[8] and in another, the right side was twice as commonly affected as the left.[3] Bilateral and recurrent cases of Bell's palsy have also occurred during pregnancy.[2,7]

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The clinical signs result from a combination of paralysis of voluntary and involuntary motor function on the affected side and unopposed action of the muscles of the unaffected side. This was vividly described in the original report by Sir Charles Bell: "The immediate effect has been the horrible distortion of the face by the prevalence of the muscles of opposite side . . . and that distortion is unhappily increased when a pleasurable emotion should be reflected in the countenance."[2]

Patients with Bell's palsy usually have a drooping face and mouth and a smooth-appearing forehead. If the face is still, only widening of the palpebral fissure and smooth forehead may be apparent, but the condition quickly becomes obvious with any voluntary or involuntary facial movement. Because the reaction of others is distressing, patients may learn to minimize facial expression during the acute phase of the illness.

The paralysis affects both the upper and lower face. Weakness of the orbicularis oculi muscle leads to difficulty in closing the eye and exaggeration of Bell's phenomenon, the normal upward movement of the eye with lid closure.[1] Early in the clinical course, the patient may complain of pain in the ear or cheek, tinnitus, fever, and either decreased hearing or increased sensitivity to loud noise (hyperacusis).[1,3,11] Other symptoms that may develop include dryness of the eye, drooling, numbness of the face or tongue or both, and alterations in taste on the anterior two thirds of the tongue.[1–3,11] The symptom complex in individual cases reflects the location of abnormality along the course of the nerve.

Diagnosis and Differential Diagnosis

By definition, Bell's palsy is idiopathic and is therefore diagnosed by first excluding all other possible causes of facial nerve paralysis. Although rare in pregnant women, many of these conditions (Table) are very serious and require urgent treatment as opposed to the usually conservative management of Bell's palsy.

The diagnosis of Bell's palsy rests on two issues: verification that the lesion is lower motor neuron, and elimination of any secondary cause for the paralysis. The involvement of both upper and lower parts of the face distinguishes lower motor neurone paralysis from supranuclear lesions.[1] The classic test is to ask the patient to attempt to wrinkle the forehead. In Bell's palsy and other lower motor neurone lesions, this cannot be achieved. Careful history-taking, physical examination, and use of appropriate laboratory tests are indicated to rule out the conditions listed in the Table. A secondary cause of facial paralysis should be suspected when the paralysis is bilateral.

Differential Diagnosis of Bell's Palsy

Infection
Otitis media
Mastoiditis
Other structure directly involving or compromising lower motor neurone of facial nerve
Herpes zoster of geniculate ganglion (Ramsay Hunt syndrome)
Other herpetic infection of facial nerve
Lyme disease
Trauma
Soft tissue injury
Facial or skull fracture
Tumor
Choleostoma
Parotid neoplasm
Any neoplasm impinging on lower motor neurone of facial nerve
Other
Sarcoidosis
Diabetes
Guillain-Barré syndrome
Bleeding disorder
Leukemia
Vascular lesions

Management

Conservative management is usually appropriate in cases of Bell's palsy that occur during pregnancy.[4] This involves patient education, observation, and emotional support. Patient reaction to the symptoms varies greatly, as illustrated by the two cases described. Some patients require considerable explanation and physician support before accepting that conservative management is appropriate. If weakness of the eye muscles makes corneal drying a possibility, methylcellulose drops and an eye patch to protect the cornea from injury may be necessary. Massage of the affected muscles may be useful[11] and electromyographic (EMG) stimulation has been used in severe, prolonged paralysis.[1]

Diuretics have been used empirically in cases of Bell's palsy, but this treatment has not been evaluated and is not recommended.[2]

In nonpregnant patients with Bell's palsy, steroid treatment is reported to improve the rate of full recovery if given early in the course of the condition. Treatment with prednisone, 60 mg daily for 5 days and tapered over the next 5 to 7 days, has been recommended in adult patients.[1,11] There are several reports of successfully treating pregnant patients who have Bell's palsy with oral steroids,[4,6] but no objective, controlled studies have been done.[2] The theoretical risks to the infant of developing cleft lip or palate as a result of the mother taking steroids during early pregnancy and of adrenal suppression if

steroids are taken in late pregnancy have not been observed in practice.[4,7] In spite of this, most authors recommend caution in prescribing steroids to pregnant women. The role of steroids appears to be limited to those third-trimester or puerperal patients with severe paralysis and risk of poor outcome. Surgical decompression of the nerve has not been widely reported in pregnant patients with Bell's palsy.

Prognosis

The prognosis for spontaneous recovery from Bell's palsy is good, with two thirds of patients fully recovered within 2 weeks of onset of symptoms.[2] The vast majority of patients are reported to make a full recovery within 6 months.[2,3] Full recovery is reported to be more likely in younger patients.[1] Incomplete recovery is reported to be more likely in recurrent cases, older patients, and those presenting with severe initial paralysis and symptoms of hyperacusis and diminished taste.[1] In cases in which symptoms persist 2 weeks after onset, EMG testing for nerve degeneration can provide prognostic information.

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