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# Bell's palsy in pregnancy and the puerperium: a report of five cases

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**Summary**
The incidence of idiopathic facial nerve palsy is higher during pregnancy and the puerperium than in non-pregnant women of childbearing age. An important association also exists between Bell's palsy and hypertensive disorders of pregnancy. We describe three patients with idiopathic facial nerve palsy in pregnancy and two in the puerperium. Two of these were associated with hypertensive disorder of pregnancy. This case report illustrates the fact that Bell's palsy is common in pregnancy and in the peurperium and often associated with hypertensive disorders of pregnancy.

**Keywords:** *Bell's palsy, puerperium, pregnancy, hypertensive, disorders.*

**Résumé**
L'incidence de la paralysie du nerf facial est très élevée pendant la grossesse et en puerperium que les femmes qui ne sont pas enceintes à l'âge adulte. Une association importante existe entre la paralysie de Bell et les désordres d'hypertension en grossesse. Nous décrivons trios patients ayant une paralysie du nerf facial en grossesse et deux en état de puerperium. Deux cas étaient associes avec les désordres d'hypertension en grossesse. Ce rapport illustre le fait que la paralysie de Bell est commun en grossesse et en état de puerperium et souvent associes avec des désordres d'hypertension.

## Introduction

Bell's palsy is one of the most common benign neurological disorders in pregnancy. It is three times more likely to occur in pregnancy and in the puerperium than at other times in women of child bearing age [1,2,3]. This has been attributed to physiologic changes occurring in late pregnancy and in the immediate puerperium and probably, altered susceptibility to herpes simplex viral reactivation during pregnancy [4]. Therefore, pregnancy is regarded as a risk factor for Bell's palsy. An important association also exists between Bell's palsy and hypertensive disorders of pregnancy [1].

The clinical manifestation is the same in pregnancy as in the general population, although, it tends to run a more severe course in the former [5]. Rarely, patients have recurrent Bell's palsy in successive pregnancies [5]. Short course of steroids early in the course of the disease has been used with some evidence of benefit [7].

We present a crop of five cases of Bell's palsy who we saw in quick succession within a few months at the Neurology Unit of the Department of Medicine, University College Hospital, Ibadan.

## Case reports

*Case 1*

A 35-year old multiparous (Gravida 3, Para 3+0, 2 alive) teacher developed sudden deviation of the mouth to the right and difficulty closing the left eye, at the 12th week of gestation. These were preceded by left temporal pain of 2-day duration and associated exaggerated perception of sound. She reported no impairment of taste or lacrimation, vertigo or hearing impairment. Neither were there rashes in the throat or ear. She was not a known diabetes or hypertension patient and her blood pressure at presentation was normal.

Physical examination findings were consistent with left lower motor neuron facial nerve palsy. She was commenced on oral prednisolone about a week after the onset of the illness with Visine eye drop, vitamins B and physiotherapy. The facial weakness improved progressively.

*Case 2*

A 26 year old Gravida 3, Para 1+1 (1 alive) woman developed facial asymmetry with difficulty closing the right eye at the fifth week of gestation. She reported increased lacrimation and dysgeusia on the right but no hyperacusis, vertigo, hearing impairment or diplopia. There was preceding otalgia and malaise.

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She had right lower motor neuron facial nerve palsy with Bell's phenomenon and excessive lacrimation. She was placed on oral prednisolone 60mg every other day for 2 weeks, Visine eye drop and physiotherapy. The facial weakness resolved significantly over a period of five months.

*Case 3*

A 31 year old Gravida 3, Para 2+0 (2 alive) trader developed, a month earlier, right facial weakness with deviation of the mouth to the left, at the 13th week of gestation. She reported no impairment of taste or lacrimation, vertigo or hearing impairment.

She was not a known diabetes or hypertension patient, her blood pressure at presentation however, was 160/130mmHg, there were no clinical or laboratory evidence of long-standing hypertension. She had no pedal edema; urine protein was 1+, serum electrolytes and creatinine, and blood urea were within normal limits. Neurologic examination revealed features in keeping with right lower motor neuron facial nerve palsy (with Bell's phenomenon). She was commenced on oral antihypertensive agents. However, she was not placed on systemic steroid due to late presentation. She improved with physiotherapy.

*Case 4*

A 32 year old multiparous woman developed left facial weakness associated with reduced lacrimation on the left and dysgeusia three weeks postpartum. The index pregnancy had been largely uneventful until at estimated gestational age of 37 weeks + 6 days when her blood pressure was found to be 150/90 mmHg and later rose to 180/120 mmHg. Her booking blood pressure at estimated gestational age of 20 weeks + 1 day was 100/60 mmHg. She was managed for pregnancy induced hypertension and was delivered of a live male baby by spontaneous vaginal delivery at 38 weeks + 1 day. She was discharged home on alpha methyldopa 250 mg thrice daily and nifedipine 20 mg once daily. She was on these medications till 3 weeks postpartum when she developed facial weakness. There was no past history of facial weakness. She was not known to have diabetes.

On examination, she had clinical signs in keeping with left lower motor neuron facial nerve palsy with Bell's phenomenon. Her blood pressure was 110/70 mmHg. A three week course of oral prednisolone; 60mg daily for 2 weeks and 30mg daily for another 1 week was commenced together with application of eyelid tape during sleep and physiotherapy. The facial weakness improved slowly.

*Case 5*

A 25 year old Para 2 + 1, 1 alive trader presented with an acute onset left facial weakness occurring a day after an uneventful spontaneous vaginal delivery. There was a preceding history of left ear ache which came on 3 hours after delivery but none of impairment of hearing or balance. There was no past history of such. There was left lower motor neuron facial nerve palsy on examination. She had no other neurological deficit. Ear, nose and throat examination revealed no vesicles in the distribution of the facial nerve. She had no disorder of taste or lacrimation. She was placed on oral prednisolone 20mg twice daily for 5 days, vitamins, eye care to prevent exposure keratopathy, and physiotherapy. Her facioparesis improved slowly.

**Discussion**

Bell's palsy is the most common disease of the facial nerve, accounting for almost three quarters of all acute facial palsies [7]. Reported frequencies range from 11 to 40 per 100,000 [6-8]. Bell's palsy affects people of all ages although it occurs more commonly between the ages of 30 and 45 years [6]. There is no gender predilection [7]. However, there is a three-fold increase in incidence in late pregnancy and puerperium [1,2,4]. Although all the 3 pregnant subjects in this series presented in the first trimester, most studies reported a low incidence in the first trimester [5].

An important association also exists between Bell's palsy and gestational hypertension or preeclampsia, accompanying 22% of Bell's palsy in pregnancy [1]. This is illustrated by the fact that two out of our five cases were associated with hypertensive disorders of pregnancy. Several surveys have also noted a relationship with hypertension and diabetes mellitus [1]. Bell's palsy occurs at all times of the year though some studies have reported seasonal clustering [5,8]. It is almost always unilateral affecting either side approximately equally. Bilateral involvement, recurrence and familial occurrence have been reported both in pregnancy and in the general population [5,8]. Three of our patients had left sided weakness while two had on the right. None had a similar episode in the past.

Bell's palsy is thought to be caused by edema and ischemia resulting in compression of the facial nerve in its course through the bony facial canal [2]. The cause of the edema and ischemia is still a subject of debate. Pregnancy, hypertension, diabetes mellitus and history of recent viral infection, especially herpes simplex, are all identified common risk factors [6,8].

This increased risk in pregnancy has been attributed to fluid retention, hypertension, compromise of the vasa nervorum, infection (particularly with herpes simplex virus (HSV), and an autoimmune process) [3]. Different studies have shown elevated HSV-1 titers in affected patients but most have not isolated viral DNA in biopsy specimen. Cause-and-effect conclusions have also not be drawn from interventional studies [6,8].

Bell's palsy in pregnancy and puerperium, as in the general population, is characterized by sudden onset facial paresis evolving rapidly, reaching maximal deficit within two days [2]. This may be accompanied by short lived, facial or post-auricular pain, otalgia or aural fullness which is typically mild and may precede the palsy [2]. Other features include hyperacusis due to paralysis of the stapedius muscle, decreased production of tears and saliva, and altered taste [2]. Careful neurological and ear, nose and throat examination are necessary in patients with facial paralysis. Finding of other neurological abnormalities warrants further testing such as magnetic resonance imaging of the brain, lumbar puncture, and electromyography (EMG) where appropriate [2,8].

Gradual onset of facial paralysis, weakness of the contralateral side, facial hyperkinesis, severe pain, headache, recurrent palsy, and involvement of other cranial nerves or history of trauma or infection must raise the suspicion of an alternative cause of facial paralysis and prompt further investigation. Severe pain, vestibulocochlear dysfunction and appearance of vesicles in the distribution of chorda tympani typically accompany herpes zoster infection (Ramsay Hunt syndrome). However, vesiculation may not appear (zoster sine herpete) or may be delayed in up to half of patients [2,8]. Two of our patients (cases 2 and 4) complained of preceding earache but had no other features to suggest herpes zoster infection.

There have been reports of bilateral Bell's palsy both in pregnancy and the puerperium and in the general population. It does not seem to be more common in the former. It is important to note however, that Bell's palsy only accounts for 23% of bilateral facial paralysis and further investigation is necessary as the majority of such patients have Guillain-Barré syndrome, sarcoidosis, Lyme disease, meningitis (neoplastic or infectious), or bilateral neurofibromas (in patients with neurofibromatosis type 2) [9]. Recurrent Bell's palsy in pregnancy and the puerperium has also been reported, presumably as a result of reactivation of a latent herpes virus infection [5,10]. However, pregnancy does not increase the risk of recurrence [5]. Overall, recurrence occurs in 10-15% of patients [5,10]. This may occur on the ipsilateral or contralateral side of the initial palsy. Approximately 30% of patients with recurrent ipsilateral facial palsy were found to have tumours of the seventh cranial nerve or parotid gland [5,10]. Patients with recurrent ipsilateral facial palsy should undergo MRI or high-resolution CT scan to rule out a neoplastic or inflammatory (e.g. multiple sclerosis, sarcoidosis) cause [10].

Incomplete motor and sensory recovery and parasympathetic impairments may all complicate Bell's palsy. These manifest as oral incompetence, epiphora, facial synkinesis and hemifacial spasm; dysgeusia , or ageusia as well as aberrant function of the lacrimal glands, which manifests as crocodile tears and shedding tears while eating. Exposure keratopathy may also occur [2,8].

The diagnosis of Bell's palsy is largely clinical and one of exclusion. Certain clinical features can aid in distinguishing it from other causes of facial paralysis. These include abrupt onset with complete, unilateral facial weakness at 24 to 72 hours, and on the affected side, numbness or pain around the ear, a reduction in taste, and hypersensitivity to sounds [11]. Serum testing for rising antibody titres to herpes virus is not a reliable diagnostic tool. Salivary polymerase chain reaction for herpes simplex virus type 1 is more likely to confirm virus during the replicating phase, but this test remains a research tool. Blink reflex testing showed a diagnostic sensitivity of 81 % and specificity of 94 % compared to the contralateral control side in a study while facial electromyography (EMG) was found generally not useful in routine evaluation of suspected Bell's palsy [12]. Isolated conduction block of the facial nerve to transcranial magnetic stimulation (TMS) early in the course of the disease represents a very sensitive finding in Bell's palsy but lacks specificity [12]. Brain MRI is not routinely indicated but shows gadolinium enhancement, the degree of which correlates with the extent of intratemporal lesions of nerve, especially in the labyrinthine segment of the facial canal [7,13].

The main aims of treatment in the acute phase of Bell's palsy are to speed recovery and to prevent corneal complications. The best available evidence suggests that steroids are probably effective in improving facial functional outcomes but no clear benefit have been demonstrated from antiviral agents or facial nerve decompression [6,7]. In a review of treatment of Bell's palsy using anti-viral agents, the authors concluded that the use of acyclovir alone remained controversial and recommended additional, adequately powered, randomized, placebo-controlled

studies [14]. In a multi-centre, randomized placebo-controlled study, the recovery rate for treatment with valacyclovir and prednisolone therapy was significantly better than that with placebo and prednisolone. The authors concluded that valacyclovir and prednisolone therapy was more effective in treating Bell's palsy, than the conventional prednisolone therapy [15]. However, the results of this study require cautious interpretation because the patients were treated in tertiary centres, and the outcome assessors were aware of the study-group assignments [6].

A subsequent double-blind, placebo-controlled, randomized trial, showed that early treatment with prednisolone significantly improved the chances of complete recovery at 3 and 9 months. There was no evidence of a benefit of acyclovir given alone or an additional benefit of acyclovir in combination with prednisolone [6].

Corticosteroids probably do no harm in pregnancy and the puerperium so a short course of prednisolone is prescribed early in the course of the disease [18]. The use of corticosteroids in pregnancy remains controversial, however, available evidence supports the use of prednisolone at any stage of pregnancy. Eighty-eight percent of prednisolone is metabolised before crossing the placenta, so it is preferred to dexamethasone when steroids must be given for more than a few days during pregnancy [16]. Concerns have been expressed that neonatal adrenal hyperplasia or insufficiency may result from maternal corticosteroid administration; however, any adrenal suppression following prenatal exposure usually resolves spontaneously after birth and is rarely clinically important. There is no convincing evidence that systemic corticosteroids increase the incidence of congenital anomalies. When administration is prolonged or repeated, which is rarely necessary in the management of Bell's palsy, intrauterine growth restriction may result [17]. The use of prednisolone in pregnancy is not restricted to the management of Bell's palsy. It is used, often in high doses, in the management of acute exacerbations of asthma and autoimmune diseases, and prophylactic treatment of neonatal respiratory distress syndrome [18]. Prednisolone was used in all the cases presented except in the third due to late presentation. None of the patients reported any symptom attributable to the effects of corticosteroids.

The role of surgical decompression in management remains controversial. A systematic review concluded that the effect of facial nerve decompression in adults and children with Bell's palsy is unknown [19]. The effectiveness of acupuncture and hyperbaric oxygen therapy in treating Bell's palsy has not been established [20,21].

Eye care is an essential and universally acceptable part of the treatment of Bell's palsy; this includes the use of artificial tears and eyeglasses during the day and application of lid tape after applying eye lubricants at night [2]. Gold weight eyelid implants in studies also provided significant reduction in lagophthalmos and significant improvement in corneal coverage [22]. Symptoms or signs of exposure keratopathy warrant urgent referral to an ophthalmologist.

Facial neuromuscular retraining for oral synkinesis, facial exercises with mirror or EMG biofeedback and physiotherapy for residual paresis have been employed with some evidence of benefit [23-25]. Other treatment options with some evidence of effect include methylcobalamin, an active form of vitamin B-12; facial retraining and botulinum toxin for synkinesis and hemifacial spasm [2].

The natural course of Bell's palsy varies from early complete recovery to substantial nerve injury resulting in persistent paralysis and synkinesis. Overall, outcome is good with about 71% recovering full function [7]. There is a strong correlation between the peak severity of the palsy and outcome [2-7]. Pregnancy appears to be a poor prognostic index; in a study, only 52% of the women with Bell's palsy of pregnancy whose facial palsy progressed to complete paralysis within 10 days of onset recovered to a satisfactory level compared to 77% to 88% of comparison patients [2]. Our patients had peak House-Brackmann grade of between III and V [26]. They all improved gradually over time. Other identified poor prognostic indices include other associated conditions like hypertension and diabetes, age over 60 years, lack of recovery by three weeks, severe pain and severe degeneration of the facial nerve shown by electrophysiological testing [2,5].

In conclusion, pregnancy and puerperium are risk factors for Bell's palsy. They are also poor prognostic indices as it is more likely to progress to complete paralysis, and recovery to a satisfactory level tends to be delayed compared to others. These need be taken into consideration in the management and counselling of these patients. However, as most patients, understandably are alarmed, they need reassurance that this is a benign condition and the probability of long term sequelae is low. A prompt and accurate diagnosis with immediate institution of appropriate management based on best current evidence is important in avoiding worsening of the symptoms and improving overall prognosis.

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