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Case Report

# Bell's Palsy in the Third Trimester of Pregnancy: A Case Report and Review of Management Considerations

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**ABSTRACT**:
Facial palsy is the weakness or paralysis of the muscles of facial expression resulting from dysfunction of the facial nerve (cranial nerve VII), and may involve either upper or lower motor neuron lesions. Bell's palsy, the idiopathic lower motor neuron type, is the most common form and occurs more frequently during pregnancy, particularly in the third trimester. We report a case of a 21-year-old primigravida at term gestation who was admitted for false labour pain secondary to a urinary tract infection (UTI). On the second day of hospitalization, she developed sudden-onset right ear pain followed by right-sided facial weakness. Neurology, ophthalmology and ENT evaluations were sought. She was diagnosed with Grade III right-sided Bell's palsy. Early multidisciplinary assessment enabled prompt diagnosis and initiation of treatment, leading to progressive recovery and favorable maternal and fetal outcomes.

***Keywords: Bell's palsy, pregnancy, facial nerve palsy, third trimester, corticosteroids, valacyclovir.***

## INTRODUCTION:

Bell's palsy is an acute, idiopathic, lower motor neuron paralysis of the VII cranial nerve (facial nerve), resulting in unilateral weakness or complete paralysis of the muscles of facial expression. The global incidence of Bell's palsy ranges from 11 to 40 per 100,000 persons annually[1]. Pregnant women appear to be at a higher risk during the third trimester or postpartum period[2]. The pathophysiology is not well understood in pregnancy. It may be due to fluid retention causing nerve compression, immunosuppression, and possible viral reactivation[3].

It is the most common cause of peripheral facial palsy and typically presents with sudden-onset facial asymmetry, retroauricular pain, hyperacusis, altered taste sensation, and decreased lacrimation[4]. The diagnosis is clinical and based on exclusion of secondary causes such as stroke, neoplasm, trauma, or infectious etiologies like Ramsay Hunt syndrome[5].

Treatment during pregnancy is complex due to the need to balance maternal benefit and fetal safety. Corticosteroids, especially prednisolone, are considered the first-line therapy and are most effective when started within 72 hours of onset of symptoms[6]. The use of antiviral drugs (e.g., acyclovir or valacyclovir) remains controversial, with some trials suggesting limited additional benefit[7]. Supportive therapies such as ocular protection and physiotherapy are essential in preventing complications and promoting functional recovery[8].

While the prognosis is generally favorable, some patients may experience incomplete recovery or residual facial weakness, particularly if treatment is delayed[9]. In pregnant women, prompt diagnosis and a multidisciplinary management approach are crucial for optimizing both maternal and fetal outcomes[10].

## Case Presentation:

A 21-year-old primigravida at term gestation presented to the obstetrics department with complaints of lower abdominal pain, increased frequency of micturition, and burning sensation during urination for the past three days. The patient denied any history of fever, chills, hematuria, vaginal discharge, flank pain, or per vaginal bleeding. There was no prior history of hypertension, diabetes mellitus, seizure disorder, or any neurological or ear ache during the antenatal period. She had not experienced any recent trauma, upper respiratory tract infection, or vesicular eruptions. Her antenatal visits had been regular, and growth and wellbeing of the fetus were appropriate for gestational age.

Upon admission, general physical examination revealed an alert and oriented young woman, afebrile,

with a pulse rate of 80 beats per minute and blood pressure of 124/75 mmHg. She had no pallor, edema, or signs of dehydration. Systemic examination, including cardiovascular, respiratory, and neurological assessments, were unremarkable at the time of admission. Obstetric examination revealed a term gravid uterus with the fetus in cephalic presentation. Uterine tone was relaxed, fetal heart rate was regular, and there were no contractions noted. Per abdominal examination elicited minimal suprapubic tenderness.

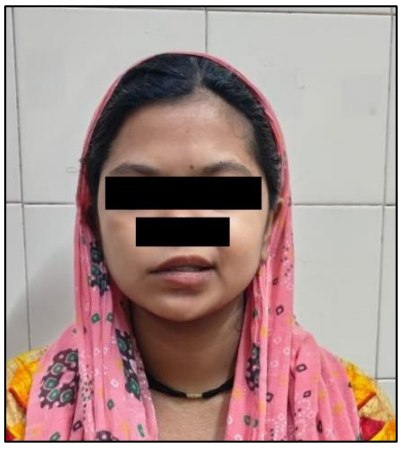
**Routine investigations were sent, which included a complete blood count and urine routine with microscopy.**

| INVESTIGATIONS | REPORT |
|---|---|
| Hemoglobin | 11.6 GM% |
| Total leukocyte count<br>Differential leucocyte count | 15,700/$MM^3$<br>Neutrophils - 75%,<br>Lymphocytes - 20%,<br>Eosinophils - 4%,<br>Monocytes - 1% |
| Platelet count | 2.80 lakh/mm |
| Urine analysis | Protein – trace<br>RBC – 0<br>WBC – 12 per high power field<br>Epithelial cells - 5<br>Bacteria - 243/µL |

These findings were consistent with a clinical diagnosis of urinary tract infection. The patient was managed with a single-dose oral fosfomycin 3 gm, followed by a five-day course of cefixime 200 mg twice daily.

On the second day of hospital stay, the patient developed sudden onset of discomfort in the right ear, followed within few hours by acute right-sided facial weakness. She noticed inability to close her right eye, difficulty while speaking, and drooping of the right angle of mouth. There was no preceding trauma, vesicular rash, or recent infection. She denied any visual disturbances, limb weakness, altered sensorium, or dizziness. The facial weakness was non-progressive from the time of onset and was localized to the right side. Importantly, there was no true earache or discharge, and hearing was subjectively preserved.

On physical examination, the patient exhibited classic signs of right-sided lower motor neuron facial nerve palsy. There was incomplete closure of the right eye (lagophthalmos), flattening of the right nasolabial fold, drooping of the angle of the mouth on the right side, loss of forehead creases on the right, and a deviation of the mouth to the left on attempted smiling. The Bell's phenomenon was clearly observed on the right side. She was unable to puff her cheeks or raise her right eyebrow. Speech was mildly slurred due to facial muscle involvement, but there was no evidence of dysphagia or limb weakness.

A.  B. 

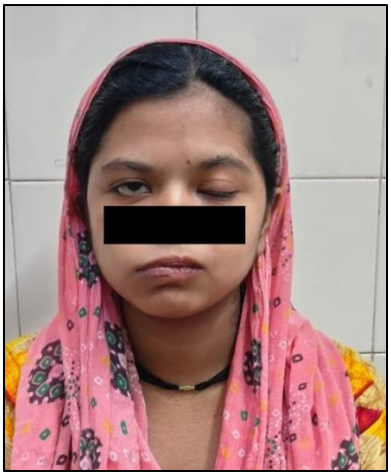
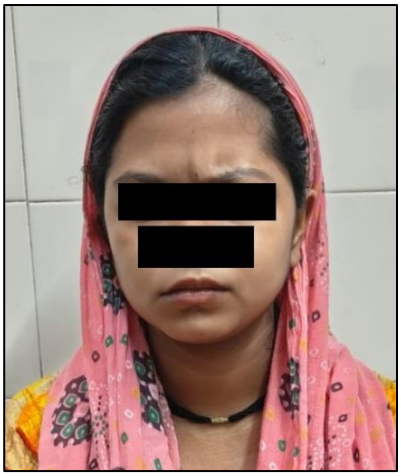
C.  D. 

**Figure A-D Right sided bells palsy A. inability to wrinkle brow, B. drooping mouth / asymmetric smile , C. lagophthalmos , D. inability to frown**

A multidisciplinary evaluation was promptly initiated. Neurology consultation confirmed the diagnosis of right-sided idiopathic facial nerve palsy (Bell's palsy), with severity graded as House-Brackmann Grade IV. On neurologic examination, the patient had a Glasgow Coma Scale (GCS) score of E4V5M6. Cranial nerve examination showed isolated involvement of the right facial nerve. Extraocular movements were full and symmetrical, pupils were bilaterally equal and reactive to light, and there were no signs of raised intracranial pressure. Motor examination revealed normal tone and full strength (power 5/5) in all four limbs, with intact reflexes (deep tendon reflexes 2+ bilaterally) and a downward plantar response. No cerebellar signs or sensory deficits were noted. Based on these findings, a diagnosis of isolated right-sided lower motor neuron facial nerve palsy in late pregnancy was made, with no evidence of central nervous system involvement.

Pharmacologic management was initiated promptly. The patient was started on valacyclovir 1 gram three times daily for 7 days. Prednisolone (Omnacortil) was prescribed at a dose of 20 mg, 2 tablets once daily for 10 days, followed by a tapering schedule of 20 mg 1.5 tablets once daily for 2 days, and then 20 mg once daily for 2 days.

Opinion was obtained from ENT specialist, which revealed bilateral impacted cerumen but there was no evidence of otitis media, mastoiditis, or other structural ear abnormalities. ENT confirmed the diagnosis of Bell's palsy, House-Brackmann Grade IV, and prescribed ear drops(Paradichlorobenzene, Benzocaine, Chlorbutol, and Turpentine oil ear drops) three times daily for 5 days. Facial physiotherapy was initiated early, focusing on muscle stimulation and neuromuscular retraining.

Ophthalmology consultation was sought due to the inability to close the right eye and risk of exposure keratopathy. The ophthalmic assessment confirmed lagophthalmos of the right eye secondary to facial nerve palsy. The corneal surface appeared clear with no signs of ulceration or dryness. The patient was advised artificial tear drops(Carboxymethylcellulose (CMC) eye drops) for daytime use, eye ointment at night, and taping of the right eyelid during sleep to prevent corneal injury.

Over the next few days, the patient showed gradual improvement in facial muscle tone and movement. Eye closure improved, drooling reduced, and speech articulation became clearer. There was no deterioration in her neurological status, and she remained hemodynamically stable throughout the course of treatment. The urinary symptoms resolved completely by day five of antibiotic therapy. Daily fetal monitoring with cardiotocography and biophysical profile scoring remained reassuring throughout the hospital stay.

The patient was discharged in stable condition with instructions to continue prescribed medications and physiotherapy on an outpatient basis. Follow-up appointments were scheduled with neurology, ENT, ophthalmology, and obstetrics teams. At two-week postpartum review, the patient showed significant improvement in facial symmetry and motor function, with grading of her facial palsy improving to House-Brackmann Grade II, and near-complete resolution of symptoms.

## DISCUSSION:

Bell's palsy is the most common cause of acute peripheral facial nerve paralysis, accounting for 60–75% of cases in the general population, with an incidence of 11–40 per 100,000 annually[1]. During pregnancy, particularly in the third trimester and early postpartum period, the incidence increases nearly threefold[2]. This heightened risk has been attributed to physiological changes such as fluid retention, hormonal shifts, immunosuppression, and microvascular compromise, which may predispose the facial nerve to edema and ischemia within the fallopian canal[3].

Although the precise pathophysiology remains unclear, proposed mechanisms include reactivation of latent

herpes simplex virus type 1, autoimmune-mediated demyelination, and vascular inflammation[4]. Bell's palsy has also been associated with hypertensive disorders of pregnancy, including preeclampsia, though our patient remained normotensive with no systemic involvement[5].

Many researchers have conducted studies to understand the increased incidence and altered disease dynamics of Bell's palsy during pregnancy. Kim et al. conducted a nationwide population-based cohort study in Korea, analyzing over 2.4 million pregnancy-related health records. Their findings demonstrated that the risk of Bell's palsy was significantly elevated during the third trimester and early postpartum period, with the incidence nearly tripling compared to non-pregnant women[2]. They postulated that pregnancy-induced changes in vascular tone, immune modulation, and hormonal shifts may contribute to peripheral nerve susceptibility.

A narrative review by Muñoz-Aguilera et al. focused specifically on Bell's palsy in the context of pregnancy. They proposed that elevated estrogen and progesterone levels during late gestation lead to fluid retention and interstitial edema, which may compress the facial nerve within the narrow bony fallopian canal[3]. Additionally, the review discussed how pregnancy-related immune suppression may enable viral reactivation, particularly of herpes simplex virus type 1, which has been implicated in the etiology of idiopathic facial nerve paralysis[4].

Grewal et al., in a focused review on Bell's palsy in pregnancy, emphasized the importance of early diagnosis and intervention. The authors noted that although the overall prognosis is good, pregnant patients may have delayed or incomplete recovery compared to non-pregnant counterparts, particularly when associated with gestational hypertension, diabetes, or preeclampsia[6]. They advocated for a multidisciplinary approach to optimize outcomes and mitigate risks related to both maternal and fetal health.

Lastly, Kim et al. (2018) also demonstrated a statistically significant correlation between Bell's palsy and hypertensive disorders of pregnancy, especially preeclampsia, further supporting the hypothesis that microvascular ischemia and endothelial dysfunction play a role in facial nerve compromise during gestation[7].

Diagnosis is clinical, characterized by acute unilateral facial weakness involving both upper and lower facial muscles. Lower motor neuron palsy, as seen in Bell's palsy, is differentiated from central lesions by involvement of the forehead and the presence of a positive Bell's phenomenon[8]. In our case, these features were clearly present, and alternative etiologies—including Ramsay Hunt syndrome, otitis media, and central causes—were excluded through appropriate clinical evaluation[9].

Prompt initiation of corticosteroid therapy within 72 hours of symptom onset remains the standard of care and significantly improves outcomes[10]. Prednisolone is preferred in pregnancy due to minimal placental transfer[11]. Antiviral agents, such as valacyclovir, are commonly used in moderate to severe cases, though their benefit remains debated; both are considered safe in late pregnancy[12]. Our patient received early corticosteroids and antivirals with favorable response.

Supportive care, including ocular protection and facial physiotherapy, is essential in preventing complications such as exposure keratopathy and synkinesis[13]. Ophthalmologic input is crucial in cases with lagophthalmos. Most patients recover within 3–6 months, and although some reports suggest slightly reduced recovery rates in pregnancy, early intervention and multidisciplinary care—as provided in this case—are associated with excellent outcomes[14].

In addition to pharmacologic and supportive therapies, acupuncture has been investigated as a complementary modality in the management of Bell's palsy. Several clinical studies and systematic reviews suggest that acupuncture may promote nerve regeneration, reduce inflammation, and improve functional outcomes when initiated early in the course of disease. The mechanism is thought to involve enhanced local microcirculation, neuromodulation, and stimulation of neurotrophic factors, which together support facial nerve recovery. Acupuncture is considered safe in pregnancy when administered by trained practitioners, and some randomized controlled trials have demonstrated improved facial muscle function and reduced recovery time when combined with standard corticosteroid therapy[15]. However, evidence remains heterogeneous, and more high-quality, placebo-controlled trials are needed to establish its efficacy conclusively. Nonetheless, in selected cases—especially where corticosteroid therapy is delayed, contraindicated, or insufficient—acupuncture may serve as a valuable adjunct in a multidisciplinary treatment plan.

While Bell's palsy does not directly affect fetal health, close antenatal monitoring is advisable given its potential association with hypertensive disorders[7]. This case highlights the importance of timely recognition and coordinated management in achieving favorable maternal and neonatal outcomes.

## CONCLUSION:

Bell's palsy, though infrequent, exhibits a higher incidence during the third trimester of pregnancy. Prompt clinical recognition and initiation of corticosteroid therapy within the therapeutic window are essential for maximizing neurological recovery. A multidisciplinary approach is integral to ensuring comprehensive care, minimizing maternal morbidity, and safeguarding fetal outcomes. When managed appropriately and without delay, the prognosis for both mother and fetus is generally excellent.

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