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Case Report

Acute facial weakness with severe preeclampsia – a diagnostic dilemma: a case report

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ABSTRACT

The 14 % of maternal mortality is attributed to preeclampsia. The neurological red flags in pregnancy and postpartum are often missed. Acute facial weakness in severe preeclampsia presents a diagnostic challenge because differential diagnosis e.g., stroke, intracranial hemorrhage, posterior reversible encephalopathy syndrome (PRES) and cerebral venous sinus thrombosis (CVST) may lead to maternal mortality. However acute unilateral lower motor neuron facial paralysis in pregnancy, particularly the third trimester and early puerperium, is associated with Bell's palsy. A 23-year-old G2P0L0A1 woman at 40 weeks + 2 days with antenatally diagnosed preeclampsia presented in spontaneous labour. During labour, she had high BP ≥160/100 mmHg despite oral antihypertensive therapy, requiring intravenous labetalol. She developed foetal distress and emergency lower-segment caesarean section was done. Postoperatively, Magnesium Sulphate was administered according to the Pritchard regimen, with antihypertensive therapy antenatally and intensive care monitoring. On postoperative day 4, she developed acute left-sided facial weakness. Examination showed left forehead involvement, incomplete left eye closure, deviation of the angle of the mouth to the right, and slurring of speech, consistent with a lower motor neuron facial palsy. Urgent neurological evaluation was undertaken. MRI brain showed no acute infarction, intracranial hemorrhage, space-occupying lesion or other significant abnormality. Neurology and otorhinolaryngology evaluation supported Bell's palsy. She received prednisolone, acyclovir, ocular lubrication and eye protection, and facial physiotherapy. Facial weakness gradually improved and she was discharged on postoperative day 7. Acute facial weakness in severe preeclampsia should be treated as a neurological emergency. Recognition of a peripheral facial palsy in severe pre-eclampsia should prompt evaluation for serious central neurological causes. Once upper motor neuron causes have been excluded, Bell's palsy should be considered and treated promptly.

Keywords: Bell's palsy, Severe preeclampsia, Puerperium, Facial nerve palsy, Pregnancy

INTRODUCTION

The 10% of all pregnancies are complicated by preeclampsia and related hypertensive disorders of pregnancy. Neurological complications are known and neurologists often become involved with preeclampsia management.[1] Neurological complications account for 30-70% of all deaths in preeclampsia, mainly due to cerebral edema and intracerebral haemorrhage.[2,3] Bell's palsy is an acute idiopathic lower motor neuron paralysis of the facial nerve accounting for the majority of cases of unilateral facial weakness.[4,5] The incidence is increased during pregnancy, particularly in the third trimester and early puerperium various pathophysiological mechanism have been proposed e.g., hormonal changes, extracellular fluid retention, immune modulation, viral reactivation, and microvascular ischemia.[4-6]

Several studies have reported an association between Bell's palsy and hypertensive disorders of pregnancy, especially severe preeclampsia.[4-6] However, neurological manifestations in women with severe preeclampsia frequently raise suspicion for serious neurological complications including posterior reversible encephalopathy syndrome (PRES) or cerebral venous sinus thrombosis.[1] Consequently, Bell's palsy may not be immediately recognized, resulting in delays in appropriate management.[5-7]

We report a case of acute facial paralysis (Bell's palsy) developing during the early puerperium in a woman with severe preeclampsia, emphasizing the importance of prompt neurological evaluation, exclusion of central nervous system pathology and multidisciplinary management.

CASE REPORT

A 23-year-old G2P0L0A1 unbooked woman at 40 weeks +2 days presented with labour pains. She was a known case of preeclampsia diagnosed antenatally and taking tab labetalol. On admission, she was conscious and oriented, with blood pressure 134/86 mmHg. Fetal assessment showed a singleton cephalic pregnancy with normal cardiotocography (CTG) tracing.

During labour, she developed severe pre-eclampsia developed despite oral antihypertensive therapy, with blood pressure high BP ≥160/100 mmHg. Intravenous labetalol was administered. Fetal heart rate abnormalities consistent with fetal distress subsequently developed and emergency lower-segment caesarean section was performed. Investigation done as showed in Table 1.

Table 1: Summary of investigations.

Investigations	Findings
Preoperative investigations	
Hemoglobin	11.0 g/dl
Platelet count	119 × 10^3/µl
Serum creatinine	1.23 mg/dl
Serum uric acid	5.57 mg/dl
Total bilirubin	1.02 mg/dl
Lactate dehydrogenase (LDH)	338.16 U/l
Total serum protein	6.5 g/dl
Postoperative investigations	
Hemoglobin	Decreased to 9.8 g/dl
Total leukocyte count	Increased to 29.05×10^3/µl, subsequently decreasing to 24.18×10^3/µl
Platelet count	Increased to 353×10^3/µl
Serum creatinine	Increased to 1.83 mg/dl, subsequently improving to 1.39 mg/dl
Total bilirubin	Increased to 1.76 mg/dl, subsequently decreasing to 1.01 mg/dl
Serum magnesium	3.9 mg/dl
Evaluation of acute facial weakness (postoperative day 4)	
MRI brain	No acute infarction, intracranial haemorrhage, space-occupying lesion, or other significant intracranial abnormality
Two-dimensional echocardiography	Normal
Thyroid function tests	Within normal limits
Neurological evaluation	Findings consistent with left lower motor neuron facial palsy
Otorhinolaryngology evaluation	Multidisciplinary assessment supported the diagnosis of Bell's palsy

On postoperative day 4, the patient developed acute left-sided facial weakness. Her blood pressure was 138/104 mmHg and she remained conscious and oriented. Examination revealed absent left forehead wrinkling, incomplete closure of the left eye, deviation of the angle of the mouth to the right and slurring of speech. These findings were consistent with a left lower motor neuron facial palsy. Given the recent severe preeclampsia and new neurological deficit, urgent evaluation was undertaken for central neurological pathology. Brain MRI showed no acute infarction, intracranial haemorrhage, space-occupying lesion or other significant intracranial abnormality. Two-dimensional echocardiography was reported as normal, and thyroid function tests were within

normal limits. Neurology and otorhinolaryngology consultations were obtained. The multidisciplinary joint supported a diagnosis of Bell's palsy with pre-eclampsia.

Specific treatment comprised of prednisolone 60 mg/day followed by tapering, acyclovir 400 mg five times daily for 7 days, facial physiotherapy and exercises, and ocular lubrication and protection because of incomplete eye closure. The patient remained neurologically stable, and gradual improvement in facial weakness was documented. She received intensive care monitoring for approximately 5 days, was subsequently transferred to the ward, and was discharged in a clinically stable condition on postoperative day 7. The clinical course is summarized in Figure 1.

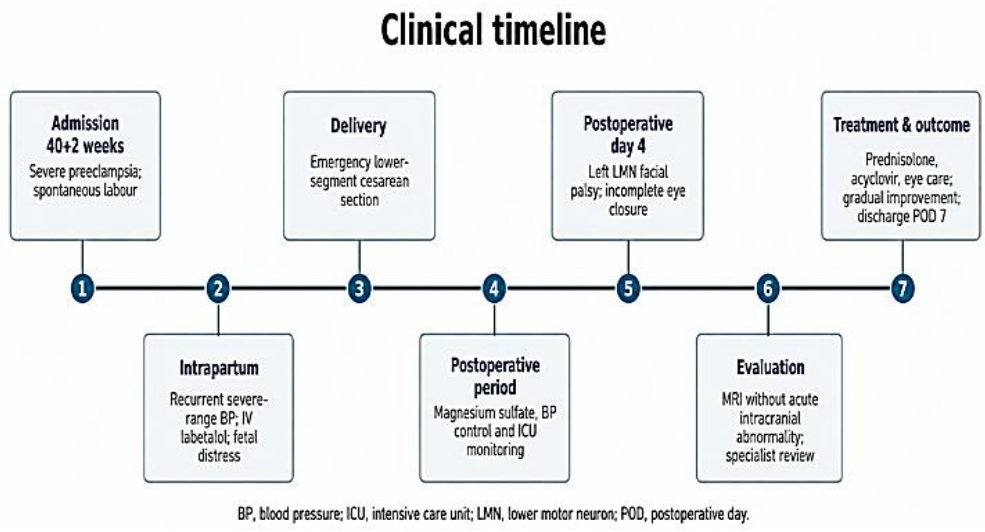


Figure 2: Clinical timeline illustrating sequence of events from admission till treatment and discharge in our case.

DISCUSSION

Neurological complications are among the most serious and life-threatening features of preeclampsia. In fact, 30% to 70% of preeclampsia-related maternal deaths are directly caused by acute central nervous system (CNS) complications. Preeclampsia essentially causes a form of hypertensive encephalopathy characterized by endothelial dysfunction, severe vasospasms, and fluid leakage across the blood-brain barrier.[1] A large portion of severe neurological complications manifest in the postpartum period (up to 44% of eclampsia cases occur after delivery).

Acute facial paralysis (most commonly Bell's palsy) during pregnancy is strongly linked to preeclampsia, occurring roughly 2 to 4 times more frequently in pregnant women especially during the third trimester or the early postpartum period.[4,6]

Perineural oedema: Fluid retention and generalized swelling (oedema) common in preeclampsia can lead to fluid build-up around the facial nerve as it travels through its tight bony canal, causing compression and palsy.[4,6]

Vascular changes: The hypercoagulable state and blood vessel inflammation associated with preeclampsia can reduce blood flow to the facial nerve (vasa nervorum).[4]

Warning sign: Acute facial weakness can occasionally precede the formal diagnosis of severe preeclampsia or hidden HELLP syndrome (haemolysis, elevated liver enzymes, low platelet count).[4,6]

Bell's palsy is an acute idiopathic lower motor neuron facial nerve paralysis, with pregnancy recognized as a risk factor, particularly during the third trimester and early puerperium. Incidence of Bell's palsy in pregnancy is 38-45 per 100,000 deliveries.[8]

Approximately 15% of acute facial paralysis cases in pregnancy may not be due to Bell's palsy.[9,10] New neurological symptoms in a woman with severe preeclampsia should not be presumed to represent a benign peripheral neuropathy. PRES, ischemic stroke, intracranial hemorrhage and CVST can cause focal neurological deficits and require urgent recognition. Hence, it was first priority to determine if this facial weakness was associated with central neurological causes.

The examination provided important anatomical localization. However, in upper motor neurone lesions (UMN) e.g., cerebrovascular accident like stroke, upper third of face is spared. But in lower motor lesions (LMN) e.g. Bell's palsy there is weakness or paralysis of the entire face on affected side. Involvement of the ipsilateral forehead and inability to close the eye indicated a lower motor neuron pattern rather than the typical upper motor neuron pattern of a supranuclear lesion. However, this finding alone did not justify immediate attribution to Bell's palsy given the high-risk clinical context. MRI was therefore performed and showed no acute intracranial

abnormality. Multidisciplinary assessment subsequently supported Bell's palsy.

Bell's palsy is primarily a clinical diagnosis in a typical isolated peripheral facial palsy, and neuroimaging is not routinely required in uncomplicated presentations. In this case, however, imaging was justified by the acute neurological deficit occurring in the setting of severe preeclampsia. This approach helped avoid overlooking of serious central causes. Though mostly Bell's palsy in pregnancy is benign but other complications may be associated in some cases. Gestational hypertension or preeclampsia is associated in 30% cases, foetal complications in 6.74%, HELLP syndrome rarely.[11] Bilateral Bell's palsy is seen in only 0.2-3% cases of all Bell's palsy. In pregnancy it may be first clinical sign followed by severe pre-eclampsia or HELLP syndrome and is associated with slower recovery.[12]

The postpartum timing is also clinically relevant as 68.8 % cases occur in 3rd trimester and rest in puerperium.[11] Neurological complications of hypertensive disorders may present or persist after delivery, and the puerperium should not be considered free of risk. In such patients, careful neurological examination and a low threshold for appropriate imaging are important when new focal deficits develop. Conversely, once dangerous central pathology has been reasonably excluded, recognizing Bell's palsy prevents unnecessary delay in supportive and definitive treatment.

Early corticosteroid treatment is the main pharmacological intervention for Bell's palsy. Antiviral therapy may be used as an adjunct in selected patients. Incomplete eyelid closure requires ocular lubrication and protection from corneal injury. The patient received corticosteroids, acyclovir, eye protection and facial physiotherapy, with gradual clinical improvement.

A limitation is the absence of formal House–Brackman grading and long-term neurological follow-up in the available records. Although gradual improvement was documented during hospitalization, the precise extent and duration of complete recovery cannot be established.

CONCLUSION

Acute facial weakness in women with severe preeclampsia should be taken as neurological emergency. In 15 % case, central neurological causes may be present. Hence Bell's palsy diagnosis may be made only after thorough evaluation has excluded central neurological causes. Early multidisciplinary assessment, appropriate neuroimaging, timely corticosteroid therapy, and meticulous ocular protection are essential components of management and contribute to favourable neurological outcomes.

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