CASE REPORT

Bell's palsy in pregnancy as a prodromal sign of preeclampsia: A report of three cases, pregnancy outcome, and literature review

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Abstract

Bell's palsy is a less common neurological disorder in the general population. Its occurrence during pregnancy can be used as a predictor of adverse obstetric outcomes including preeclampsia and its complications. We report cases of three pregnant women from Botswana who presented with Bell's palsy in the third trimester coexisting with preeclampsia and multiple complications. One of the patient was a case of maternal near-miss with multiple life threating complications including stage 3 acute kidney injury (AKI) and required hemodialysis. The second and third patients developed Bell`s palsy and preeclampsia at term, management of preeclampsia commenced with immediate delivery resulting in good maternal and neonatal outcome. In all the three patients preeclampsia and Bell`s palsy completely resolved post-delivery. Therefore, new onset Bell's palsy in pregnancy may be used as a prodromal sign of preeclampsia. Such patients deserve close follow up for preeclampsia or gestational hypertension for a better obstetric outcome. *(Afr J Reprod Health 2021; 25[3]: 135-141).*

Keywords: Bell's palsy, preeclampsia, gestational hypertension, Botswana

Résumé

La paralysie de Bell est un trouble neurologique moins fréquent dans la population générale. Son apparition pendant la grossesse peut être utilisée comme un prédicteur d'issues obstétricales défavorables, y compris la prééclampsie et ses complications. Nous rapportons les cas de trois femmes enceintes du Botswana qui ont présenté une paralysie de Bell au troisième trimestre coexistant avec une prééclampsie et de multiples complications. L'un des patients était un cas de quasi-accident maternel avec de multiples complications potentiellement mortelles, notamment une insuffisance rénale aiguë (IRA) de stade 3 et une hémodialyse nécessaire. Les deuxième et troisième patientes ont développé une paralysie de Bell et une prééclampsie à terme, la prise en charge de la prééclampsie a commencé avec l'accouchement immédiat, ce qui a donné de bons résultats maternels et néonatals. Chez les trois patientes, la prééclampsie et la paralysie de Bell ont complètement disparu après l'accouchement. Par conséquent, une nouvelle paralysie de Bell pendant la grossesse peut être utilisée comme signe prodromique de prééclampsie. Ces patientes méritent un suivi rapproché de la prééclampsie ou de l'hypertension gestationnelle pour un meilleur résultat obstétrical. *(Afr J Reprod Health 2021; 25[3]: 135-141).*

Mots-clés: Paralysie de bell, preeclampsie, hypertension gestationnelle, Botswana

Introduction

Bell`s palsy is peripheral palsy of the facial nerve resulting in weakness of facial muscles including the forehead without other neurological abnormalities. Mostly it is unilateral but sometimes can be bilateral. Classically the symptom progress over three to seven days and resolves within three to twelve weeks of its onset[1, 2]. Peripheral facial nerve palsy may be primary (idiopathic or Bell's palsy) or secondary. Sir Charles Bell first explained the distribution and function of the facial nerve in 1821 and since then his name has been attached to acute lower motor neuron facial paralysis of unknown etiology[3]. The diagnosis of Bell`s palsy is mainly based on its acute clinical presentation of peripheral facial nerve palsy without other neurological disorder. In case of gradual onset of the facial nerve paralysis over a period of more than two weeks and/or no evidence of recovery within three weeks, other causes of facial nerve palsy should be considered. Such insidious onset of facial nerve palsy with slow recovery may be caused by neoplastic, infectious or autoimmune disorders that

requires further investigations[1]. The prevalence of Bell's palsy among pregnant women is estimated at 45 cases per 100,000 births while for non- pregnant women of the same age group, the calculated incidence is 17 per 100,000 women per year. The majority of cases occur in the third trimester and puerperium[4]. There are few reported cases occurring in the first trimester of pregnancy and some cases may recur in subsequent pregnancies[5-7].

From studies done by D. Shmorgun *et al*[8] in Canada, Bell's palsy in pregnancy was found to have association with serious obstetric complications such as gestational hypertension and/or preeclampsia in 25% of the cases. It has also been reported that there is a higher incidence of low birth weight and preterm deliveries among these cases. The onset of Bell's palsy in the third trimester was reported to be 71- 80% and 10 -21% during puerperium[8-10].There are also some speculations about possible genetic predisposition as familial occurrence of idiopathic facial nerve palsy has been reported [9]. Bell's palsy can be a prodromal sign of underlying early preeclampsia or coexist with disorders like gestational diabetes mellitus, carpal tunnel syndrome and HELLP syndrome (hemolysis, elevated liver enzyme and low platelet count)[10].

We report three cases of Bell's palsy, all of them developed preeclampsia and one patient developed complications including stage 3 acute kidney injury, HELLP syndrome and fetal demise.

Case presentation

Case 1

A 25 year old Gravida 2 Para 0, abortion 1 African woman from Botswana presented to a primary hospital at 29 weeks of gestation with two days history of right sided facial weakness, failure to close her ipsilateral eye lid and dryness of the same eye. Her blood pressure at presentation was 130/85mmHg with fundal height of 28cm and fetal heart rate of 136 beats per minute with the diagnosis of right sided incomplete facial nerve palsy, she was given oral 6-methyl-prednisolone 60 mg/day for 7days with eye care. She reported that her 30 year old elder sister developed permanent right sided facial weakness at a gestational age of 7 months 3 years ago.

At 30 weeks of gestation, she was referred to a tertiary hospital (Princess Marina tertiary hospital) with a two-hour history of lower abdominal pain, vaginal bleeding and absent fetal movement. She had headache and blurring of vision. Her blood pressure was 172/110mmHg, Pulse rate of 80 beats per minute and her booking weight at 14 weeks was 60kg. Abdominal examination revealed a tense and tender abdomen with a fundal height of 32 cm, cephalic presentation and absent fetal heart tones confirmed by ultrasound. The cervix was 50% effaced and 3cm dilated with intact membranes and minimal bleeding at the time of examination. Urine protein was 3+ on dipstick, and her fasting blood sugar was 10.5 mmol/L (189mg/dl). She was admitted to labor ward with a diagnosis of severe preeclampsia complicated with intrauterine fetal death (IUFD), placental abruption, and Bell's palsy; she also has gestational diabetes mellitus. Labour was induced with amniotomy and oxytocin 10 miliunits per minute the same day and she delivered female fresh still birth weighing 1300gm with no gross congenital anomalies. There was total placental separation. Despite active management of the third stage of labor she lost an estimated 1800ml of blood. Oxytocin infusion was continued at 20 units in 500ml saline and misoprostol 400 microgram per rectum was administered the bleeding subsided. Her blood pressure dropped ranging between 140/80 to 155/95 mmHg, pulse rate 116 beats per minutes. Methyldopa and Hydralazine were used to control blood pressure, Magnesium Sulphate for seizure prophylaxis and regular human insulin initiated. On day three of her admission she developed fever at 38-39^{O}C with leukocytosis of 17.9×10^{3}/ μL. Septic screening was considered and broad spectrum intravenous antibiotics were commenced. Postpartum, her hemoglobin dropped from 9g/dl to 4.9g/dl and she was transfused with 4 units of packed red cells. She was also anuric with serum creatinine 1800U/L, urea 12mmol/L and lactate dehydrogenase 439μmol/L. Serum alanine aminotransferase 130U/L, aspartate aminotransferase 200U/L and bilirubin 15U/L. Peripheral blood smear revealed fragmented red cells with features of hemolysis. Her platelet count was 43 x 10^{3}/ mm^{3}. Based on hemolysis, elevated liver enzymes and low platelet count, HELLP syndrome with stage 3 acute kidney injury was diagnosed. She was therefore admitted to the intensive care unit (ICU) where she received

4 units of Platelet, fresh frozen plasma and packed red cells. Hemodialysis was initiated, she received a total of 6 sessions, and she was also put on erythropoietin. Six days after delivery, the facial palsy started to improve and it had completely resolved by day 8. Three weeks after delivery her laboratory results and urine output had normalized: her hemoglobin was 13g/dl, platelet count 194,000/µL, serum urea 2.2mmol/L, creatinine 38mmol/L, alanine aminotransferase 10U/L, aspartate aminotransferase 19U/L and bilirubin 1.1U/L. On 25th day of her admission she was discharged in good condition and advised to have a weekly outpatient follow-up. On her sixth week post-delivery, facial palsy had completely resolved and blood pressure of 100/60mmHg with normal clinical and biochemical findings and fasting blood sugar was 4.3mmol/L. All the medications including insulin were discontinued. A year later she conceived and delivered at term with uneventful pregnancy outcome.

Case 2

A 19 year old African primigravida woman was admitted to antenatal ward at a tertiary hospital with a diagnosis of severe preeclampsia and incomplete Bell`s palsy at gestational age of 37 weeks. Two weeks prior to her admission, she was diagnosed as a case of gestational hypertension and she was on expectant management at outpatient clinic with planned induction of labor at 37 weeks. A day prior to her admission, she reported severe headache and right upper quadrant pain. She also reported right sided facial numbness and deviation of the face to the left. She has no other surgical or medical disorder. Her blood pressure after admission ranged between 160/85 to 165/90 mmHg, pulse 98 beats per minute (bpm), respiratory rate 20 breaths per minute and temperature of 37^{o}C. Uterine size was 36 centimeter cephalic presentation and fetal heart rate was 128 beats per minute. Ophthalmic evaluation revealed incomplete closure of the right eye with normal visual acuity. There was remarkable deviation of the mouth to the left side. Her complete blood count, blood chemistries and urinalysis were within the normal ranges. With the diagnosis of severe preeclampsia and incomplete Bell`s palsy at term. Induction of labor was initiated. She was put on Methyldopa for control of blood pressure and Magnesium sulphate for seizure prophylaxis.

Table 1: Time at onset of clinical complications and remissions in the three cases

	Case 1	**Case 2**	**Case 3**
Gestational age at Onset of Bell`s palsy in weeks	29	37	39
Gestational age at onset of Preeclampsia or HELLP syndrome in weeks	30	37	39
Time of complete remission of Bell`s palsy in weeks after treatment with steroid	1	2	2

Neurologist`s review endorsed the diagnosis of Bell`s palsy. She was also given Methyl prednisolone 60mg per day for 7 days including eye care and physiotherapy. She was delivered by emergency cesarean section following a failed induction of labor with an outcome of a male neonate weighing 2405 gram with Apgar score of 9, 9 and 10 in the first fifth and tenth minutes respectively. On day 3 postpartum the patient was discharged with controlled blood pressure. Two weeks post-delivery, the facial weakness had completely resolved. Her blood pressure was normalized without antihypertensive medications.

Case 3

A 40 years old African Gravida 3 Para 2 woman at gestational age of 39 weeks was referred from a local clinic after she presented with right sided facial weakness. She had difficulty closing her right eye. She noted obvious deviation of the mouth to the left side. After three days, she developed headache that was not relieved by analgesics but no blurring of vision or epigastric pain. The fetal movements were normal. She denied any other medical or surgical disorders. Her blood pressure ranged between 130/90 to 150/100 mmHg, pulse rate 65 beats/ minute and respiratory rate 22 breaths/ minute. Remarkable left sided facial deviation was observed and she was unable to close the right eye. Symphysial fundal height was 38cm, with a cephalic presentation and fetal heart rate of 142 beats per minutes. Her leucocyte count was 6,000/µL, haemoglobin 12gm/dL and urine protein was negative. Renal and liver function tests were normal. She was admitted to hospital with a diagnosis of severe preeclampsia and incomplete Bell's palsy at term. Evaluation by a Neurologist confirmed the diagnosis of Bell`s palsy. She was commenced on magnesium sulphate for seizure prophylaxis and on methyl prednisolone 60mg per

day, Neurotone, multivitamins, facial physiotherapy and eye protection using eye patch. Induction of labor was initiated and she delivered a live female neonate, weighing 3000grams with an Apgar score of 8, 9 and 10 in the first, fifth and tenth minute respectively. The third stage of labor was actively managed and was uneventful. On her second follow-up 2 weeks after delivery, the facial palsy had completely resolved and she was asymptomatic.

Discussion

In our first case she was diagnosed with Bell`s palsy at primary hospital a week prior to her presentation at tertiary hospital. On admission to tertiary hospital she was diagnosed to have preeclampsia with severe features. She developed complications including placental abruption, IUFD, stage 3 acute kidney injury, HELLP syndrome and sepsis. Based on WHO criteria she was a case of maternal near miss[11]. Similar presentation was reported by other researchers [12, 13]. Our case had a successful pregnancy outcome a year after her admission for Bell`s palsy and complicated preeclampsia and HELLP syndrome. Similar case has been reported by Pourrat [13]. Katz *et.al*[7] has also reported Preeclampsia with severe feature after onset of Bell`s palsy. However, the poor perinatal outcome of our case is not consistent with their reports.

Case 2 was initially diagnosed with gestational hypertension later on developed some features of severe preeclampsia and Bell`s palsy simultaneously. She delivered by cesarean section a neonate with good Apgar score but low birth weight. Shmorgun *et.al* has reported similar finding with 22% incidence of preeclampsia in a patient with Bell`s palsy compared to 7.3% in the general pregnant population. He also reported higher incidence of cesarean delivery (43%) and low birth weight similar to the complications seen in our patient[8].

In case 3 the patient presented with advanced pregnancy, she developed Bell`s palsy subsequently she was diagnosed with preeclampsia with severe features three days after onset of Bell`s palsy. The perinatal outcome was good, that can be explained by the late onset of Bell`s palsy and preeclampsia. This is consistent with a similar report by Ragupathy and Emovon[14]. Another study also reported that there is no significant association between bell`s palsy and poor perennial outcome[7].

All the three cases in this report has been diagnosed as Bell`s palsy in pregnancy based on their clinical presentation. Although Bell's palsy is a relatively less frequent disorder in the general population, it tends to have strong association with pregnancy. It has higher incidence in patient with preeclampsia or gestational hypertension during antepartum period and puerperium including the uncommon variants of simultaneous bilateral Bell's palsy[2]. Literature from North America, Europe and Middle East cite Bell's palsy as a possible predictor of preeclampsia based on the higher co-occurrence of the disorders during pregnancy and having a hypothetical common pathogenesis[8-10,15].In our first and third cases preeclampsia preceded by Bell` Palsy while the second case initially developed gestational hypertension followed by severe preeclampsia with Bell`s Palsy. Obstetricians usually consider bell`s palsy in pregnancy like the usual idiopathic bell's palsy and may not consider the next possible complications such as preeclampsia. Clinicians should take bell`s palsy as a possible prodromal sign of preeclampsia and other obstetric complications[12].

The facial nerve has a unique course covering a long distance in the bony canal. The labyrinthine segment of the facial nerve is both the shortest and thinnest portion of the facial nerve with in the canal with non-anastomosing arterial arcades. Any disorder like edema of the facial nerve and/or ischemia which compromises the already narrow canal leading to strangulation and facial nerve palsy[16]. Tissue edema especially accumulation of fluid in the extravascular space is common during pregnancy which might explain the possible impingement of facial nerve in the facial nerve fallopian canal; similar pathophysiologic process has been explained in a patient with carpal tunnel syndrome in pregnancy as fluid retention has been used to explain median or ulnar nerve impingement[17,18]. Majority of Bell's palsy occurs in the third trimester of pregnancy that is due to common pathogenesis for both preeclampsia and Bell's palsy which is tissue edema[8,14].The incidence of preeclampsia in pregnant women with Bell's palsy is reported at 22% which is five times higher than the incidence of pre-eclampsia among women without Bell's palsy. Incidence of gestational hypertension was also noted to be similarly higher[8,10]. There are some reports of Bell's palsy among first degree relatives which might raise the question of genetic predisposition. Our first case

reported such a familial occurrence of possible Bell's palsy. The possibility of genetic predisposition requires further investigation[4,9]. The effect of metabolic disorders on Bell's palsy is controversial as some of the findings failed to show any difference in a case of Bell's palsy and general population. Others reported some medical disorders such as diabetes mellitus, chronic hypertension and obesity to have a strong association with Bell's palsy. Such disorders may cause atherosclerosis of vasa nervorum leading to ischemia and neuropathy. In the presence of hypertension micro emboli or thrombosis and vasospasm can lead to facial nerve compression[7,19]. Our first patient had gestational diabetes mellitus, average body weight and no previous history of chronic medical conditions. Therefore, she had no any preexisting metabolic disorder that increases the risk of atherosclerosis that leads to bell`s palsy and severe preeclampsia. The other two cases were also free of other metabolic disorders.

Our first case was complicated with HELLP syndrome, acute kidney injury and IUFD due to placental abruption and severe preeclampsia. She required ICU admission and haemodialysis. Bell's palsy was diagnosed just a week prior to her admission. In a case reported by Shapiro *et al*, Bell`s palsy might present with partial HELLP syndrome with minimal complication to the pregnancy outcome. While some of the cases might present as a postpartum complication[10,20], Pregnant women with this disorder should be closely followed up and investigated for preeclampsia or gestational hypertension for better obstetric outcomes.

Shmorgun *et al*[8] explored adverse pregnancy outcomes in patients with Bell's palsy including increased incidence of cesarean delivery rate, preterm birth, and low birth weight. But other recent studies done by Katz *et al*[7] revealed no significant association between Bell's palsy and adverse perinatal outcomes. Such study showed a strong association of Bell's palsy with severe preeclampsia. Our first patient presented with intrauterine fetal death a week after onset of Bell's palsy as a result of complications of severe preeclampsia with placental abruption[7,8]. The second and the third cases had a relatively good perinatal outcome with the exception of low birth weight in the case of the second patient which may be attributed to late onset of preeclampsia, Bell`s Palsy and earlier interventions. This finding is similar to cases reported by Ragupathy and Emovon who have reported good perinatal outcome in cases with onset of Bell`s palsy and preeclampsia at term[14].

The value of steroids and antivirals in the treatment of Bell's palsy has been controversial. According to a study by Peitersen E from Denmark, the normal course of Bell's palsy is; 85% will have complete recovery within three weeks while the rest can persist up to 5 months[21]. Even though such studies reported that the use of steroid does not make any difference on the treatment outcome, a Cochrane review of randomized control trials revealed the use of corticosteroid is beneficial in a patient with Bell's palsy[22]. Other studies showed the rate of complete recovery with the use of prednisolone to be significantly superior to not use of prednisolone or use of acyclovir alone or acyclovir with prednisolone in the treatment of Bell`s palsy[23,24]. Meta-analysis do not support the routine addition of antivirals to steroids in the treatment of Bell's palsy as there isn't any benefit over steroid alone, hence antiviral was not used in the treatment of our patients[25]. Therefore, steroids are the current evidence based means in the management of Bell's palsy. Corticosteroids reduce swelling and inflammation of the facial nerve with facilitation of remyelination[26]. Our patients also received prednisolone which might explain the fast recovery of facial palsy. In a patient with Bell`s palsy prednisolone treatment should be initiated within 72 hours of onset of the symptoms and eye care is recommended in patients with incomplete closure of the eye[27]. A Cochrane review failed to generate a strong evidence supporting the benefit of physiotherapy for Bell`s palsy, all our patients received physiotherapy. This is based on some studies supporting physical therapy for improving facial function and most guidelines still recommend it's use [28]. There is no strong evidence in use of surgical decompression of facial nerve impingement (surgical intervention) in management of Bell`s palsy[29].

We have observed three cases of Bell`s palsy in pregnant women over 5 years in a tertiary hospital with average delivery rate of 6000 per year. Obstetricians generally see a case of Bell's palsy less frequently once every seven years from some reports[14]. The diagnosis of Bell`s palsy in pregnancy should raise a high index of suspicion and prompt vigilant surveillance for subsequent development of serous obstetric complications like

preeclampsia and /or HELLP syndrome. Obstetricians should be aware of such uncommon presentation in pregnant woman and use it as an indicator of such complications and initiate appropriate investigations with individualized management plan[14]

Conclusion

Any pregnant woman with facial nerve palsy should be evaluated meticulously for any associated maternal and fetal condition. Obstetricians should commence thorough investigations for the possible pregnancy complications like preeclampsia or HELLP syndrome with its complications so as to prevent adverse maternal and perinatal outcomes.

Consent for publication

Written consent was obtained from all the patients participated in this case series.

Competing interests

All the authors declare that they have no any competing interest.

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Authors' contributions

TMB prepared the draft and DH involved in the draft preparation and reviewed the manuscript. TMB, NM and JS involved in the management of the cases and reviewed the manuscript. All authors reviewed the draft manuscript and approved the final version of the manuscript.

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