STUDII DE CAZ

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Neurological disorder in pregnancy

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ABSTRACT

Bell facial paralysis association with pregnancy pathology is quite rare in practice, but reported to the cases outside gestation it is 2 times more frequent, which was why we decided to present this case.

In the presented case, we note that the neurological pathology was revealed in a patient, aged 34 years and located in 34 weeks gestation with pregnancy-induced hypertension which was presented accusing facial asymmetry insidiously installed.

The treatment was agreed between the obstetrician and the neurologist. Birth was conducted in normal dynamic parameters, evolving naturally with a good fetal and maternal prognosis. Further development of the peripheral facial paralysis was slowly favorable, with a partial remission 3 weeks later. A year and a half after this episode that took place during the gestation, the neurological and clinical examination revealed the persistence of a discrete facial asymmetries.

Keywords: idiopathic facial Bell paralysis, pregnancy-induced hypertension, pregnancy, eutocic labour

INTRODUCTION

An uncommon pathology in pregnancy is represented by the cranial nerves one. If headache, back pain, seizures are commonly met, impairment of the seventh cranial nerve, the facial nerve, is less known and controversial. In 1830, Sir Charles Bell described the first association between the idiopathic peripheral facial paralysis called Bell's paralysis and the pregnancy (1). Since then until now, various studies and theories have attempted to explain the connection between the neurological pathology and the pregnancy, given that the incidence of the

Bell pathology is estimated at 45 cases/100,000 pregnancies, much higher than in non-pregnant 17 cases/100,000 for women at gestational age (2). Most of the cases occur in the third quarter of pregnancy or 2 weeks before the delivery or post-partum.

The facial nerve is both a sensitive – motor nerve and a somato- vegetative one (3), with the origin of the motor fibers in the bridge and it innervates all the mimic muscles insuring a sensory-sensory function from the conca auricular, external auditory canal, the external face of the eardrum, part of the retro auricular skin and the taste sensitivity in 2/3 anterior of the

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tongue. The cortico-nuclear fibers damage causes the central type facial paralysis, while the lower nuclear and infranuclear injuries determine the peripheral facial paralysis.

CASE PRESENTATION

We present the case of a 34-year-old pregnant woman, 34 week of gestation, which was admitted in the neurology department for facial asymmetry insidiously installed 3-4 hours prior to the presentation to the medical office. We take into consideration that in the woman's history it was a birth 8 years ago, an eutocic birth at term, with a healthy child, the woman being a patient without chronic illnesses. The anamnesis shows a normotensive patient antepartum, having the blood pressure over 150/90 mmHg during the pregnancy and with antepartum overweight.

The objective neurological examination revealed a facial paralysis of peripheral type with asymmetry of the face: lagophthalmia - slot eyelid enlarged in the sick side, epiphora – leaking tears through the inner corner of the eyelid on the cheek, the Negro sign - the eye of the affected side seems moved up, the folds of the hemiforehead on the paralysed side deleted, nasal groove of the paralyzed side is deleted, the corner of the mouth of the sick side being lowered, with missing blinking the sick side, the sign Charles Bell - when closing the eyes, the eyeball of the sick side diverts up and in or up and outside, no impairment of taste or sensibility, without earache, so that it was established the diagnoses of idiopathic peripheral facial Bell paralysis or "a frigore". The positive diagnosis of peripheral facial paralysis was a purely anamnesis and clinical one, the laboratory investigations and imaging explorations aiming to find the cause - MRI skull, audiogram, CBC, ESR, lumbar puncture. It was established the Dexamethasone treatment 1f im/day, 5 days, and fat-soluble vitamins B group, by mutual agreement between the neurologist and the obstetrician.

After 3 weeks the pregnant woman gave birth to a healthy baby vaginally delivered, without intra- or postpartum incidents or complications. Both labor, parturition and after birth period were within normal parameters. The fetal prognosis was good; the intrauterine fetal condition was not influenced by the coexistent neurological disorders.

The further development of the peripheral facial paralysis was slowly favorable, with a par-

tial remission 3 weeks later. EMG performed 10 days after the acute onset should have highlighted data about the evolution and prognosis (4) voluntary multistage potentials, favorable neurological maternal prognostic or fibrillation in case of unfavorable evolution, but this investigation could not be performed taking into account the state of the 36 weeks pregnant. A year and a half after this episode spent during the gestation, the clinical examination revealed the persistence of a discrete facial asymmetry and the skull imaging examination MRI without pathological changes.

DISCUSSIONS

The Bell's paralysis is a medical condition its diagnosis being established only by the exclusion of the other ethiopatogenic possibilities. Locating the lower part of the aqueduct, the Bell's paralysis does not present sensory, taste, tear, salivation or hyperacusis disturbances, consisting only in motor deficiency of the hemifacial level, the pathology being associated or not with retroauricular pain. The peripheral facial paralyses are determined mostly by the pathological processes localized in the last third of the Fallope aqueduct and can be of infectious nature: influenza viruses, herpes simplex, herpes zoster, bacterial acute or chronic infections, traumatic, tumoral, due to systemic diseases: hypertension, diabetes, leukemia, Hodgkin lymphoma, deficient states (3).

The treatment of the peripheral facial paralysis may be a medical one (4-6) with steroids, virostatics, vitamins of group B, botulinum toxin (7) by surgery (5) – muscle transpositions with temporal, masseter muscles, which are not connected to the facial nerve or the facial nerve decompression, fiziokinetically (8-10): by external massage, electrotherapy. The drug treatment is contested by some authors, but it can be efficient especially if inserted within the first 72 hours, with or without the fiziokinetical treatment, the surgery is reserved for those cases that present important sequelae. Despite the correct and quickly established treatment there are 10% of the cases that remain with a slight facial asymmetry, while 5% remain with important sequelae: muscle dysfunction, dyskinesia (11,12). At our patient, we considered it appropriate the corticosteroid combination with the vitamin therapy, given the state of gestation, which proved to be beneficial to both the mother and the fetus.

The pregnant women predisposition for peripheral facial paralysis were attributed to the higher extracellular fluid, to the inflammation and to the immunosuppression characteristic for the pregnancy (13). Some authors suggest the Bell paralysis association with preeclampsia (14), more frequent in the third quarter of pregnancy, this one presenting subcutaneous tissue and nervous system edema (15), probably due to the neurocompresive effect (16). Another explanation may be the hypercoagulable state associated with preeclampsia, giving way to thrombosis in vasa nervorum, determining ischemia and nerve paralysis (17).

At our pregnant with more than 150/90 mmHg hypertension, preeclampsia can be considered a contributing factor in the appearance of the facial paralysis Bell, taking into account that this is accompanied by edema in the interstitial spaces and by the presence of vasoactive substances that amplify the immunological conflict between the vascular endothelium and the platelets. A hormone based hypothesis was suggested linked to the effects of the estrogen and progesterone during the pregnancy (18), given that on the one hand, throughout the pregnancy the mother's body is hormonally "flooded" and on the other hand, the incidence of Bell paralysis in pregnant women is more than 2 times higher than in no pregnant women. Although the etiology of the Bell paralysis is unknown, being probably a multifactorial one (4), these mechanisms or others that are to be discovered will improve the recovery and the prognosis of Bell paralysis in pregnancy.

CONCLUSIONS

In terms of etiopathogeny in our case, we can consider preeclapsia as a contributing factor in the onset of the Bell paralysis taking into consideration that this one is accompanied by edema in the interstitial spaces, the presence of vasoactive substances that amplifies the immunological conflict between the vascular endothelium and platelets. The appearance of Bell facial paralysis has a low incidence from the obstetrical point of view, but in the world literature it is reported with a 2-fold greater frequency compared with the no pregnant patients. The positive diagnosis was supported by the anamnesis, the clinical general examination, the obstetrical and neurological one being confirmed by the laboratory investigations and by the imaging examination.

The intrauterine fetal condition and the labor progress were not affected or influenced by the coexistent neurological disorders. The treatment established jointly by the neurologist and the obstetrician, was beneficial for both mother and fetus.

The fetal-maternal prognosis was good after birth by natural way, the neurological pathological phenomena remission were recorded with a partial remission 3 weeks later. A year and a half after this acute episode spent during the gestation, the clinical examination revealed the persistence of a discrete facial asymmetry and the skull imaging examination MRI was without pathological changes.

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