The Impairment of Facial Nerve Function during Pregnancy

Diagnostic and Therapeutic Protocol

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Abstract

The physiological state of pregnancy occupies a special position in the general ENT pathology. When impairment of the facial nerve function occurs during pregnancy, it is important that the ENT specialist cooperate with the obstetrician in view of a better appreciation of the therapeutic conduct. There are numerous controversies regarding the treatment of the facial nerve palsy appeared during pregnancy.

Keywords: *facial palsy, cortisone, pregnancy*

Introduction

The facial nerve is a complex mixed nerve, which has several components: a motor one - the most important-, a sensory one - insignificant, and an autonomous (vegetative) one. Its damage can involve one or all of its components. In the case of idiopathic peripheral facial palsy we are probably dealing with a cranial mononeuritis, of possibly viral etiology. Sometimes, other cranial nerves are implicated. The incidence of idiopathic peripheral facial palsy is 20:100000 per year[1].

During pregnancy there may be an isolated impairment: facial neuropathy[2]. The possibility to have an association between pregnancy and facial palsy was first noticed by Sir Charles Bell in 1830, and since then many authors have reported an increasing incidence of such association. This is also known as the Mona Lisa Syndrome. There have been speculations that the famous smile may have been caused by the contraction of the facial muscles, appeared as a result of incomplete recovery after facial palsy. Mona Lisa is said to have been pregnant while posing for Leonardo da Vinci[3]. In 1975, Hilsinger calculated the incidence of Bell's Palsy in pregnant women at 45.1/100,000, as compared to 17.4/100,000 in non-pregnant women of the same age[4]. The hazard ratio for developing facial palsy is 3.3 higher in pregnant women. Facial palsy more frequently appears during the third trimester of pregnancy[5].

There are several theories regarding the increased incidence of facial palsy in pregnancy.

A. The first hypothesis would be that, as a result of an increase in the interstitial fluid volume, an edema may result, leading to a compression of the nerve and ischemia in the Fallopian canal. This is a bony passage in the bone, approximately 3cm. long, through which the facial nerve passes; the facial canal commences at the internal auditory Meatus with the horizontal part which passes at first anterior (medial crus of facial canal) then turns posterior at the geniculum of the facial canal to pass medial to the tympanic cavity (lateral crus of facial canal); finally, it turns downward (descending part of facial canal) to reach the stylomastoid foramen. There are two portions of nerve at the level of the canal: the tympanic portion and the mastoid portion. The nervous implication takes place first as a demyelization, which can subsequently develop into an axonal damage, and, eventually, through Walerian degeneration, the neural mass can be destroyed[1]. This is most commonly a unilateral process, the bilateral being much rarer. It is a mechanism similar to the one believed to be involved in the Carpal tunnel syndrome in pregnancy.

B. The other important hypothesis is the viral cause. During pregnancy, gestational immunosuppression is induced by the increase in cortisone level. This can trigger an activation of the latent common cold sore virus, Herpes Simplex. Some authors, starting from an assumed herpetic etiology, recommend the treatment of idiopathic facial palsy with Acyclovir in the first three days[2]. It has been demonstrated that the incidence of Herpes Simplex infection is higher in pregnant women than in the general population.

The diagnosis is established after examination and investigation of the facial nerve.

There are multiple clinical manifestations.

On the Facial Motor Exam:

1. On inspection - facial asymmetry, disappearance of facial wrinkles and folds, widening of ipsilateral palpebral fissure, oral commissure ptosis, inability to frown, raise the eyebrow, blink, lagophthalmia, inability to blow, smile on the affected area, show teeth, articulate, phonate, whistle, swallow properly, drooping, drooling, twitching, facial hypertonia;

2. On palpation: the absence of cornea reflux.

On the sensory component, we find rash and otodynia, otalgia, otoneuralgia, as well as a destructive action manifested as the anesthesia of the Ramsay Hunt area (i.e. cochlea, the posterior half of the external auditory meatus and the tympanic membrane).

The sensory system malfunction is manifested by hemiageusia (loss of taste on one side of the tongue), hypogeusia - diminished taste, on the lingual presulcal part, and lacrimal secretion malfunction manifested as xerophthalmia (a condition described by pathologic dryness of the conjunctiva and cornea).

Methods of investigating the facial nerve:

- Schirmer's test determines whether the eye produces enough tears to keep it moist;
- Blatt salivary flow test;
- Bornstein's method gastrometry- a sensory exploration of the facial nerve;
- Krarup electrogastrometry- a neurosensory exploration of the facial nerve;
- tonal liminary audiometry-testing the auditory organ, to indicate the degree of hearing impairment;
- impedance metry using the GSI 33 autotympanometer to measure: middle ear pressure, middle ear compliance volume of ear canal, stapedial reflex;
- supraliminary audiometry- the reflex threshold level and the persistence of the reflex response on prolonged stimulation, i.e. the reflex decay test;
- x-rays taken in Schüller's, Chausse's II, Stenvers', Hirtz's projections (to be avoided during pregnancy);
- CATs with administration of a contrast agent (to be avoided during pregnancy);
- MRI;
- Hilger Facial Nerve Stimulator;
- Electrical Impedance Myography[2].

Prognosis and treatment

As for the prognosis and treatment of facial palsy, they are similar in pregnant and non-pregnant women, except for pregnancies in the first trimester[4,6].

The treatment of Bell's palsy in pregnancy is subject to controversy. Systemic corticotherapy is the most commonly chosen treatment option. In addition, there is a secondary therapy of the facial nerve, depending on the type of palsy. The current information on the safety of corticotherapy during pregnancy is conflicting and difficult to interpret.

In incomplete palsy, Adour and Hetzler's cortisone modified scheme of treatment is recommended; Prednisone 1mg/kg/day for 5 days; if the palsy remains incomplete, the treatment will be gradually interrupted after 5 days. This therapy will start on the 1st, 3rd, 10th at the latest, day after onset of palsy[1].

If it becomes complete, or it is complete from onset (which rarely happens in pregnancy), treatment in hospital is often indicated. In this case we cannot recommend high dose corticosteroid therapy (Prednisolone from 100mg to 1000 mg/day) over a long period of time, according to Stennert's protocol[1].

Other authors recommend, irrespective of the type of palsy, low dose Prednisolone, 1mg/kg/day, for not more than 4 days, after which the dosage is decreased gradually (the total must not exceed 300mg for the whole duration of treatment). The prolonged treatment with cortisone may inhibit the myelin sheath regeneration, damaged through facial palsy[70].

In the 2nd and 3rd trimesters of pregnancy, minimum doses of glucocorticoids are accepted, with the remark that maternal glycemia should be under observation in the case of long term treatment[8].

Studies on animals pointed to the possible adverse effects on the developing fetus when corticosteroids are administered systemically: orofacial clefts, intrauterine growth retardation, placental insufficiency, intrauterine fetal death. Nevertheless, the information on humans under systemic corticoid treatment is not so worrying[9,10]. A large part of the results of the studies on humans published in the last 25 years have not confirmed the fears generated by the studies on animals[11]. Occasionally, oral corticoids may be necessary in the obstructive nasal polyposis, facial palsy or non-allergic eosinophilic sinusitis which does not respond to topical treatment. Prednisone and Prednisolone seem to present a very low risk for the developing fetus, as only a tiny quantity of drug passes the placental barrier. Their administration is preferred in pregnancy also because the hydric retention they produce is very low, whereas their anti-inflammatory activity is very high (medium-dose glucocorticoids). Ideally, the oral forms should be administered after the first four months of pregnancy, if the patient's clinical condition allows such delay[12].

The secondary therapy of facial palsy

Along with the etiologic treatment of facial palsy, it is always necessary to provide a symptomatic treatment of the eyes, in order to protect the cornea (corneal ulcer hazard), in the case of incomplete lid closure. At night, a watch glass face shaped patch should be applied, at daytime glasses with lateral protection can be worn. Simultaneously; an ophthalmic ointment will be prescribed (for example Bepanthen) for the night, and artificial tears (for example Vidisic eye

gel) for the day. In the case of incomplete recovery of the nerve potential, a supplementary programme of facial exercises is advisable. Consequently, on the one hand, it is possible to restore some residual facial nerve function, and, on the other hand, the nerve regeneration will be accelerated. For permanent palsy with incomplete eye lid closure, auxiliary plastic surgery (Tarsorrhaphy for example)may be resorted to[2]. Physiotherapy (ionizations, faradisations) is forbidden two months after the onset of facial palsy because of the risk of subsequent facial spasm.

For pregnant women, the treatment with steroids mentioned above can be used[4]. It has been noticed that an increased risk of fetal malformations exists. Therefore, monitoring the adrenal function is mandatory[4].

Incomplete facial palsy has a good prognosis. In complete palsy, 50-60% of the patients completely recover without any treatment, and 40% recover partially. With steroid treatment, 80-90% recover completely, even in a short time.

Personal Case Reports between 2007-2010

We will present six clinical cases in which we discovered this association, without establishing any correlation between facial palsy and pregnancy.

1. Four of the cases have been of pregnant women in the last trimester of pregnancy, who presented with facial palsy within 48 hours from onset. After clinical and paraclinical examination, Bell's Palsy was diagnosed. The possible risks for the fetus of a steroid treatment were explained. All patients refused treatment during pregnancy, agreeing to return for reevaluation after delivery. The only treatment was to protect the cornea (with artificial tears), all of them presenting lagophthalmia. Two weeks after birth, on reevaluation, there was complete remission of facial palsy, without any need for secondary therapy.

2. The fifth case was of a 39-year-old woman, in week 25 of her pregnancy, who, on clinical and paraclinical examination, was diagnosed with Bell's Palsy. With the patient's consent, treatment with Prednisone 1mg/kg/day p.o. was given for 4 days, and for the next 3 days the dosage was decreased, all in all the treatment comprising 300 mg of Prednisone. Secondary therapy for eye protection and facial exercises were instituted from the very beginning. After 3 weeks there was complete remission.

3. The last patient was a 28- year- old woman, in week 27 of pregnancy, with a similar presentation to case 5. The cases differed in that there was no complete remission either at the end of the treatment, or during the pregnancy. Postpartum physiotherapy was resorted to, leading to complete remission after 10 sessions.

Discussion

The diagnosis of this condition is established on medical history, and clinical and paraclinical examinations, to exclude facial nerve palsy of a different etiology.

During pregnancy, the most likely etiology of facial palsy is edema, resulting from the increase in the interstitial fluid volume, which can lead to the nerve compression and ischemia in the Fallopian canal.

Facial palsy appeared in the last trimester can remit spontaneously postpartum, without any therapeutic intervention, with reversal of the nerve lesions.

If occurring in the second trimester, after consultation with the obstetrician and consent from the patient, treatment with cortisone p.o. will be instituted

Conclusion

1. Pregnant women are 3.3 times as likely to develop facial palsy as non-pregnant women.

2. The most frequently chosen therapy option is by systemic corticoids.

3. The collaboration between the ENT specialist and the obstetrician is mandatory.

4. The prognosis is similar for pregnant or non-pregnant women. ■

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