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Neurotic angioedema and pregnancy: Report of a case and review of the literature

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ABSTRACT: Hereditary angioedema is a recently identified inherited entity. Schematically, the C1 inhibitor (C1Inh) sees its function decrease with estrogens, an event associated with a semiology made up of edema attacks. Classically, this clinical form is aggravated by synthetic estrogens, and improved by natural estrogens. We report the case of a woman who successfully led a pregnancy, despite the various crises observed throughout the pregnancy with functional repercussions.

KEYWORDS: Sarcoidosis, granulomatous, mastitis.

1 INTRODUCTION

Angioedema is a clinical symptom characterized by sudden localized swelling of the subcutaneous and / or submucosal tissue. It is hypodermic edema [1]. This swelling is the color of the skin, sometimes pinkish but never red. It is not or very little itchy, but can be painful. It is neither inflammatory nor permanent. It disappears without sequelae and can be recurrent. Its prevalence is estimated at 0.05% in the general population [2]. The main causes of recurrent AE are histamines by mast cell activation IgE (allergy) or non-IgE, the latter being related to chronic urticaria [3]. There is a much rarer cause of AE, hereditary, acquired, or drug-induced bradykinic AE (6%) [4]. The diagnosis should not be missed, because these EAs are resistant to treatment of histamine EAs (adrenaline, antihistamines). They respond to a specific treatment which is effective but which has a certain cost. Bradykinic AEs are classified according to whether they are associated with a C1Inhibitor (C1Inh) de fi cit (hereditary or acquired) or with a normal C1Inh ("type III" AEH) or iatrogenic AE secondary to inhibitors of the enzyme converting enzyme. angiotensin (ACEI) [5].

2 OBSERVATION

This was a 36-year-old patient, with no history of hereditary angioedema in the family, G3P2, two living children gave birth vaginally with good psychomotor development followed from birth for hereditary angioedema with C1 inhibitor deficiency, only on corticosteroid therapy and which has successfully brought a pregnancy to term with seizures during the pregnancy mainly affecting the facial area and extremities. The birth was vaginally. The child born with an Apgar at 10 and was taken into pediatric care. Exogenous C1Inh concentrates were not used.

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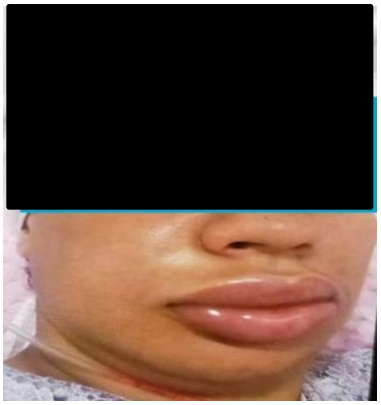

3 DISCUSSIONS

To date, the few published cases and the studies that do exist, generally involve a small number of patients, and provide a variable picture of neurotic angioedema during pregnancy. As expected, because pregnancy and childbirth are stressful situations triggering neurotic angioedema, various authors have reported an increase in seizures during pregnancy and the postpartum period [6], [7], [8]. A potential factor that can influence the rate of seizures is hormonal fluctuation during and after pregnancy, as increased estrogen and hormonal changes can negatively impact the course of neurotic angioedema [9], [10]. We also observed higher seizure rates in the extremities and facial area. The seizures were weaker in the urogenital region. Although the status of neurotic angioedema in the fetus had no effect on the rate of seizures during pregnancy, theory reveals that C1 - INH activity levels were lower in women wearing fetuses with neurotic angioedema than women carrying fetuses without HAE. The possible explanation could be that fetuses with neurotic angioedema may, due to their inherent deficiency, tend to rely on maternal C1 - INH. During pregnancy, the average seizure rate per trimester increased from 3.6 to 5.2 in the second trimester and 7 in the third trimester. Although a similar study previously reported a decrease in attack rates in the second and third trimesters [11].

4 CONCLUSION

Pregnancy in neurotic angioedema requires careful monitoring, both clinical and biological, childbirth can be supervised by simple corticosteroid therapy. This is perfectly tolerated on the obstetrical and neonatal plans.

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