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Case Report

Preeclampsia in Mirror: Ballantyne Syndrome Complicated by Eclampsia: A Case Report

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Abstract

We wish to recall the existence of Ballantyne syndrome, which was first described in association with feto-placental hydrops whose etiology was Rhesus alloimmunization. We present a case of mirrored preeclampsia, possibly related to non-immune fetal hydrops, with elements of fetal malformation and complicated by eclampsia. Following this case report, a review of the literature will confirm that other non-immunological etiologies have been reported.

Introduction

Ballantyne syndrome, also known as mirror syndrome or pseudo preeclampsia, is a rare but serious medical condition. It is characterized by maternal edema due to hydrops fetalis, generalized maternal edema and placentomegaly [1]. It is usually accompanied by severe preeclampsia. This clinical entity, first described by John Ballantyne in 1892, was initially associated with fetomaternal alloimmunization in the Rhesus system [2].

However, over the decades, understanding of Ballantyne syndrome has broadened to encompass a variety of etiologies, including non-immunological medical conditions such as fetal cardiac arrhythmia, Ebstein's disease, fetal tumors, parvovirus B19 viral infections, and others. Despite these advances, the pathogenesis of this syndrome remains idiopathic [3].

We present a case of mirror image preeclampsia, possibly related to non-immune fetal hydrops, with elements of fetal malformation and complicated by eclampsia [3]. This clinical observation highlights the importance of recognizing Ballantyne syndrome in a variety of contexts, including those unrelated to Rhesus alloimmunization, in order to optimize the management of patients and their babies [1]. We will also discuss the current state of knowledge about this condition and its impact on clinical management, including specific preconception considerations [3].

Case Presentation

A is, 19 years old, IGIP, with no particular pathological history, blood group O rhesus positive, admitted to our hospital for severe preeclampsia in a monofetal evolutive pregnancy of 33 weeks+1 day. On clinical examination, the patient presented with facial puffiness, bucketing edema of the lower limbs, elevated blood pressure of 190 mmHg/100 mmHg in both arms, a protein-positive urine dipstick (x3), with no nitrites or leukocytes, and neurological signs of severe headache, tinnitus, visual fog and epigastric bars, with no metrorrhagia. She reported a rapid weight gain of 12 kg in 2 months, with generalized edema. Obstetrical examination revealed well-perceived fetal sounds with positive uterine contraction; on TV, the cervix was long, firm and posterior, with a tense lower segment. Obstetrical ultrasonography revealed a progressive monofetal pregnancy in hydrops with subcutaneous edema, peritoneal and pleural effusion, and hydramnios. Abdominal ultrasonography, performed because of the presence of epigastric bars, was unremarkable. The diagnosis of Ballantyne syndrome was made in view of the presence of the complete picture. A biological work-up was requested, revealing the absence of Hellp syndrome, and toxoplasmosis, HIV and syphilis serologies were negative; the patient was immune to rubella. Her blood pressure during pregnancy was normal. She showed a rapid weight gain of 12 kg in 2 months, with generalized oedema. Given the severity of the symptoms and blood pressure, and the non-reassuring fetal condition, a caesarean section was performed, resulting in the extraction of a premature baby weighing 1300 g with an Apgar score of 5/10, presenting abdominal distension and a malformative syndrome, and hospitalized in the neonatal intensive care unit. The admission work-up showed hemoglobin at 10 g/dL, hematocrit at 27%, platelet count at 90,000 elements/mm^3, prothrombin rate at 100%, normal liver function, renal function and uremia. Placenta exploration was performed, showing placental hypertrophy, which was referred for anatomopathological examination. However, the post-operative course was marked by persistent high blood pressure, leading to an eclamptic crisis despite the administration of magnesium sulfate, treated with diazepam 10 mg IVD. As a result, the patient was transferred to intensive care for further management. She was discharged at 10 days postpartum with a cardiology consultation, serological tests (parvovirus B19 and cytomegalovirus), and blood pressure monitoring. The patient's monitoring was her particularity.

Discussion

Ballantyne syndrome, a rare entity in clinical practice, is often unrecognized and underdiagnosed. Only twenty cases have been reported in the last 46 years, underscoring its rarity. This syndrome complicates around 50% of cases of hydrops fetalis, which represents around one in every 6,000 pregnancies. It usually occurs in the late second or early third trimester, which is the time of diagnosis in our case. Clinically, Ballantyne syndrome presents similarities to preeclampsia, including the presence of maternal "mirror" edema,

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fetal hydrops, and sometimes elevated blood pressure accompanied by neurosensory signs such as headache, phosphene and tinnitus, as observed in our patient [4-6].

Biologically, hemodilution is an almost constant feature of this syndrome, unlike preeclampsia, where hemoconcentration is common. Proteinuria is also generally slightly increased. Other biological abnormalities may occur, such as non-haemolytic dilution anemia, hyperkalemia, hyponatremia, disturbances in renal function, hyperuricemia, or increased transaminases. However, platelet levels generally remain stable [5-7]. Although there are pathophysiological similarities with preeclampsia, the placental mechanisms appear to be different. Studies have shown an increase in anti angiogenic factors, such as soluble vascular endothelial growth factor receptor.

(sVEGF R-1) and soluble Fms-like tyrosine kinase (sFlt-1), in Ballantyne syndrome, suggesting a similar pathophysiology to preeclampsia. However, differences remain, including different plasma concentrations of endothelial growth factor receptors (sVEGFR-1) in patients with Ballantyne syndrome compared with preeclampsia [8,9].

Fetal prognosis in Ballantyne syndrome is often guarded, with a high risk of in utero death due to the severity of hydrops fetalis. However, what makes this syndrome particularly complex is its potential morbid impact on the mother, which can lead to maternal death due to acute pulmonary edema, renal failure or seizures. Management depends on a number of factors, including maternal condition, gestational age, identified etiology (if available), and in utero management options. When a curable cause is identified, specific in utero treatment is recommended, which can improve fetal prognosis and avoid extreme prematurity. However, in the absence of an identified curable cause and in the presence of threats to maternal life, medical termination of pregnancy should be considered too rapidly normalize maternal clinico-biological parameters. Although spontaneous resolutions have been described, particularly in cases of parvovirus B19 infection, the prognosis remains uncertain, and careful monitoring is necessary for maternofetal salvage [10-13].

Conclusion

Mirror syndrome is an exceptional pathological entity whose pathophysiological mechanism remains poorly elucidated. The diagnosis should be made when maternal preeclampsia is associated with hydrops fetalis. Early management is essential, given the severity of the maternal-fetal prognosis. Specific treatment in utero is highly desirable, as it allows regression of maternal-fetal clinical and biological abnormalities. Knowledge of this entity is useful, as it can avoid untimely termination of pregnancy by treating the etiology more specifically. Preconception counselling is also different from classic pre-eclampsia.

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