MASSIVE VULVAR EDEMA IN A WOMAN WITH SEVERE PREECLAMPSIA. A case report and review of literature.

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ABSTRACT

We report a case of massive vulvar edema in a 20 years old primigravida woman with severe preeclampsia at 32 weeks gestation. Other causes of vulvar edema were excluded. The vulvar edema appeared as the blood pressure increased, and cesarean section was performed for increasing preeclampsia and fetal distress. In the post partum period, the vulvar edema resolved progressively. By the fourteenth day post cesarean section, the vulvar edema had completely regressed and the blood pressure was within the normal range. The aim of this report is to alert clinicians that vulvar edema complicating preeclampsia could be a poor prognostic sign.

KEY WORDS: Vulvar - Edema - Pre-eclampsia.

I- INTRODUCTION

Vulvar edema complicating preeclampsia is unusual [1-3]. Preeclampsia is a multisystemic disorder of pregnancy. It complicates 3% to 5% of pregnancies [4]. It is a major cause of maternal morbidity and mortality worldwide. The cardinal clinical features of the condition are hypertension and proteinuria occurring after 20 weeks gestation in women who were not previously known to be hypertensive [4]. Other signs and symptoms include edema and headache. In severe cases, the condition is associated with seizures ( eclampsia), liver and kidney dysfunction, clotting abnormalities, adult respiratory distress syndrome and fetal growth restriction [4, 5]. We report a case of massive vulvar edema in a 20 years old primigravida with severe preeclampsia at 32 gestational weeks.

II- CASE REPORT

The authors report the case of 20-year-old primigravida, admitted in the obstetric and gynecology unit of Yaoundé General Hospital for severe preeclampsia at 32 gestational weeks. Her prenatal care was done regularly in a private clinic and the routine prenatal tests (hemoglobin level, syphilis, rubella, toxoplasmosis, bacteriuria, glucosuria and proteinuria)
were normal. She was of blood group A rhesus positive, hemoglobin electrophoresis AA, and her HIV serology was negative.

On admission, physical examination revealed, a good general state, blood pressure of 170/112mmHg, weight of 74.5 kg, height of 167cm and body temperature at 37°C. On obstetric examination, the fetus was in cephalic presentation, the fundal height was 31 cm and the fetal heart rate was normal. Pelvic examination including inspection of the vulva was normal. Examination of the legs revealed bilateral moderate pitting pedal edema up to the ankle.

Urine analysis showed positive proteinuria at 2+. The 24 hours urine collection showed significant proteinuria at 3.4g /24 hours. Other laboratory tests were within the normal range thus: serum fasting glucose; hepatic tests; hemoglobin level: 12.2g/dl; haematocrit: 30.8%; uricemia: 45g/l; creatinemia: 0.5g/l; uremia: 12mg/l; serum albumin: 3.1g/dl. Ultrasonography showed a normally evolving fetus with an estimated fetal weight of 2026g at 33 gestational weeks. The biophysical profile was normal.

The diagnosis of severe preeclampsia was made. Conservative treatment was initiated. This included bed rest, antihypertensive treatment with methyldopa and atenolol, and magnesium sulphate was administered for 24hours to prevent convulsions. Steroids were also administrated to accelerated fetal lung maturity, and a 12 hourly non stress test was instituted.

On day 4 hospitalisation, we noted the rapid appearance of a massive vulvar edema (Figure 1) and the blood pressure values remained elevated. In addition, there was a progressive increase of the pedal edema beyond the ankles, associated with bilateral palmar edema. The doses of the blood pressure drugs were increased.

On day 10 hospitalisation, at 34 weeks gestation, fetal evaluation revealed an abnormal biophysical profile. The blood pressure was raised at 180/114mmhg, and there was persistence of the massive vulvar edema. The patient had an unfavourable cervix and was stressed. After counseling the patient and her parents, a cesarean section was opted for. It resulted in the delivery of a male baby of weight 2100g, APGAR score of 7 at the first minute and 9 at the fifth minute. During cesarean section, marked ascites (800ml) was noted.

During post partum period, we advised the vulvar immersion in an antiseptic solution (Gynecological Betadine®). In the next seven days the blood pressure and vulvar edema gradually regressed. When the patient was seen on the fourteenth day of post partum, the vulvar oedema had completely resolved (Figure 2) and normal blood pressure was noted.

III- DISCUSSION

Clinical presentations of patients with preeclampsia vary greatly. This is partly related to the severity of affected vascular beds and organ systems involved [4].

To our knowledge, there are very few reports of vulvar edema associated with preeclampsia (Table I) [1,2,3,6]. A suggested mechanism for the development of vulvar edema in pregnancy is that rennin angiotensin activation causes fluid retention, which results in edema, especially in the legs and feet. The vulva can be affected by the pressure-volume disturbances since it has a thin epithelium and loose connective tissue [1-4]. We postulate that vulvar edema and ascites in preeclamptic patients develop from the common mechanism of hypoalbuminemia and/or increased capillary permeability, observed in preeclampsia[5]. In our case, we assumed that, the presence of ascites and vulvar edema correlated with the above mechanism.

We hypothesize that the clinical course of vulvar edema in preeclampsia patients might correlate with the development of ascites, and should therefore be considered a poor prognostic sign. In such cases, preeclampsia worsens with the appearance of vulvar edema.
Table I- Cases of vulvar edema in preeclampsia

<table>
<thead>
<tr>
<th>References</th>
<th>Year</th>
<th>Age (years)</th>
<th>Gestational age (weeks)</th>
<th>Related pathology</th>
<th>Treatment modality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morris et al(^{b})</td>
<td>1990</td>
<td>24</td>
<td>20</td>
<td>Preeclampsia, hydrocephalus</td>
<td>Resolved after termination of pregnancy</td>
</tr>
<tr>
<td>Bracero et al(^{l})</td>
<td>1991</td>
<td>-</td>
<td>-</td>
<td>Preeclampsia</td>
<td>Mechanical drainage</td>
</tr>
<tr>
<td>Daponte et al(^{3})</td>
<td>2007</td>
<td>17</td>
<td>33</td>
<td>Preeclampsia</td>
<td>Resolved after Cesarean section</td>
</tr>
<tr>
<td>Törkyılmaz et al(^{b})</td>
<td>2008</td>
<td>19</td>
<td>34</td>
<td>Preeclampsia</td>
<td>Resolved after termination of pregnancy</td>
</tr>
</tbody>
</table>

Others causes of vulvar edema include infections, neoplastic mass, congenital anomalies, trauma, inflammatory diseases (Crohn disease, vulvovaginitis, contact dermatitis), venous thrombosis, nephritic syndrome, congestive heart failure, tocolytic therapy [7-11]. All of these were excluded in this case.

Treatment of vulvar edema is necessary, since it may be alarming to the patient, painful, uncomfortable and may cause occlusion of the vaginal outlet. Treatment should be dictated by the specific cause of edema [7, 11]. Symptomatic treatment includes bed rest, trendelenburg positioning, and application of ice bags, hypertonic saline bags and local antibiotics [8, 12]. Heparin therapy may be considered since one of the reasons may be venous thrombosis [7]. Some authors suggest mechanical drainage as an alternative, in case of non response to non invasive methods [7, 13]. If delivery becomes urgent, cesarean section may be necessary. In our case, delivery was by cesarean section and the vulvar edema resolved gradually in the post partum.

This report aims at alerting clinicians that vulvar edema complicating preeclampsia could be a poor prognostic sign necessitating comprehensive review of the patient’s management.

REFERENCES