International Journal of Academic Health and Medical Research (IJAHMR)
ISSN: 2643-9824
Vol. 8 Issue 2 February - 2024, Pages: 86-90

Mirror syndrome, a rare clinical entity: about a case

YACOUBI KHEBIZA S [1], FDILI A FZ [2] , Jayi S[3], Chaara H[4] , Melhouf MY A[5]

Department of Obstetrics Gynecology II CHU Hassan II of Fez.

Abstract : Known since 1892 and very rarely present in diagnoses, Mirror Syndrome is a maternal pathology designating a syndrome of fetal hydrops complicated by more or less generalized maternal edema. It is a rare clinical entity. We report a case of a patient admitted to our structure for the management of generalized edema and a review of the literature to challenge clinicians on the existence of this syndrome which could be undertested for the benefit of easy diagnosis of the pre-eclampsia due to the association of hypertension, proteinuria and maternal edema with which it shares a clinical similarity

Keywords: Mirror syndrome, Fetal anasarca, Maternal edema, pre-eclampsia

1- INTRODUCTION

le Syndrome miroir est une pathologie maternelle désignant l´association d´une anasarque fœtale immune ou non immune, et d´un œdème maternel généralisé pouvant se compliquer d´une pré-éclampsie. C'est une entité clinique rare. Nous en rapportons un cas diagnostiqué dans notre formation avec une revue de la littérature afin de mettre en avant les particularités diagnostic et thérapeutique de cette entité clinique grave. [1,2].

2- CLINICAL CASE

This is a 36-year-old patient, 3rd operation, 2nd parent, with no notion of consanguineous marriage, no notable pathological history, admitted on the day of our call for uterine contractions in a so-called 8-month pregnancy with a doubly scarred uterus. Pregnancy not followed by an apparently normal course, in particular without neurosensory signs of arterial hypertension with a negative infectious history.

Furthermore, the patient reported excessive weight gain with significant edema affecting the face, upper limbs and lower limbs reaching the vulvar level which occurred suddenly 1 week before her consultation.

1- The general admission examination:

Found a conscious parturient, slightly dyspenic, hypertensive with a arterial tension of 16/10 cmhg on two occasions without neurosensory signs, and a positive urine dipstick with 2 crosses with generalized edema (puffy face, edema of the lower limbs reaching the vulvar level and hand infiltration), weight 110 vs 70 kg before pregnancy.

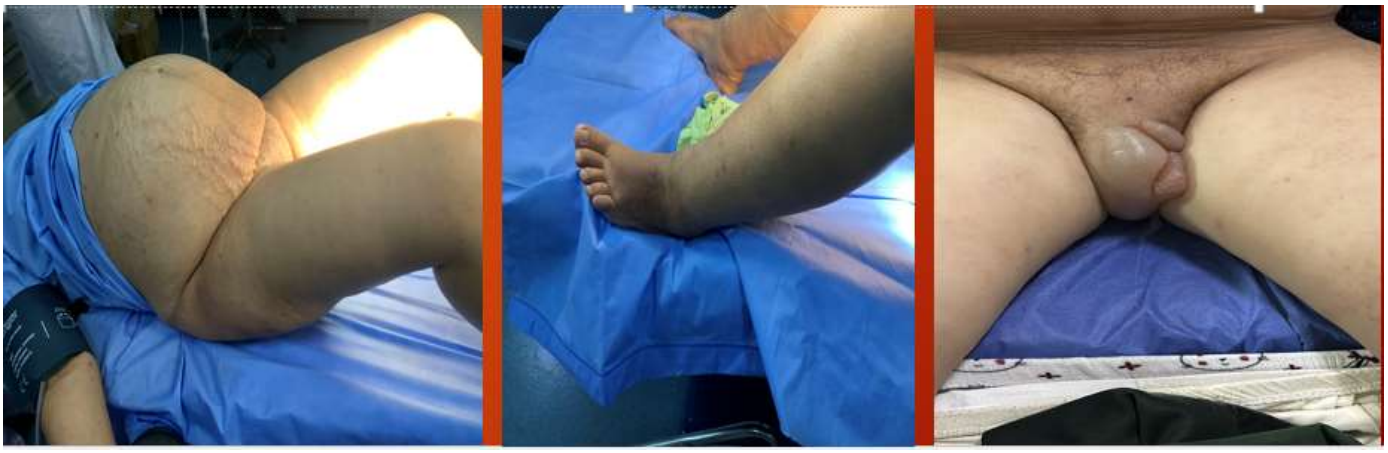

Figure 1 : Generalized edema of the lower limbs, vulvar and abdominal

2-On the ultrasound

The emergency ultrasound performed showed Non-progressive single-fetal pregnancy, negative cardiac activity, thickened fundal placenta with fetal anasarca made of very abundant ascites and bilateral pleural effusion without pericardial effusion or prefrontal edema associated with very abundant polyhydramnios (Greater cistern 18cm), figure 2

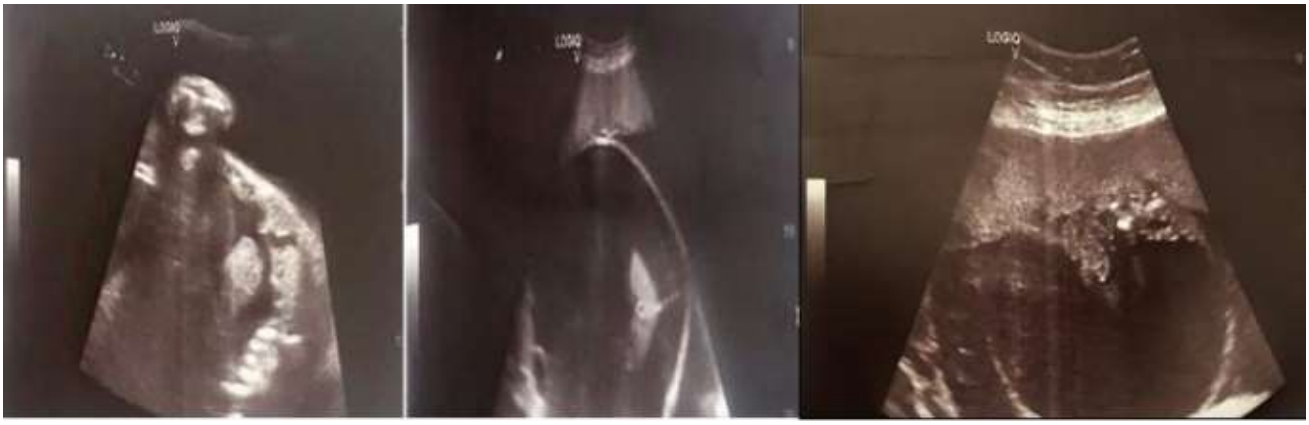

Figure2: pleural effusion, fetal ascites

3- In terms of biological assessment:

A preeclampsia assessment was carried out Complete blood count (normal hemoglobin and platelet levels),
Liver assessment showed cytolysis at three times normal
Normal kidney function,
AB+ blood grouping, negative irregular agglutinin testing

Faced with this observation, the decision was to cesarize the patient given her obstetric history (double scarred uterus)
During the cesarean section we aspirated 3l of amniotic fluid, laborious extraction of a freshly infiltrated stillborn female, weight 4800 g, the rest of the cesarean section was unremarkable,
the patient was hospitalized following confinement with strict monitoring of blood pressure.

Hospitalization was marked by the normalization of blood pressure under monotherapy and the regression of edema in 48 hours with a weight of 90 kg at discharge.
The 24-hour proteinuria recovered came back positive at 2g/24h.

International Journal of Academic Health and Medical Research (IJAHMR)
ISSN: 2643-9824
Vol. 8 Issue 2 February - 2024, Pages: 86-90

Figure 3 : stillborn infiltrated

4- DISCUSSION

Ballantyne syndrome was first described in 1892 by JOHN W Ballantyne; it combines fetal anasarca complicated by more or less generalized maternal edema accompanied by albuminuria and sometimes anemia.
In 1947 Potter described in several patients symptoms similar to those of preeclampsia, but in the context of an immunological fetal pathology,
Several cases are then reported under several names: mirror syndrome, maternal syndrome and feto-placental anasarca, maternal rhesus alloimmunization syndrome, triple edema, pseudo-toxemia, etc.
Rhesus alloimmunization has long been considered the common cause of mirror syndrome, but since then, other etiologies have been discovered: sacrococcygeal teratomas, Gallienus vein aneurysm, placenatary chorioangioma, ebstein's disease . [3,4].

a- definition

Mirror syndrome is the expression of the extreme severity of feto placental damage,
This is a picture suggestive of pre-eclampsia linked to an etiology different from the usual defect of trophoblastic invasion,
The starting point appears to be fluid retention syndrome, which appears to be fluid retention syndrome that occurs regardless of the etiology of hydrops fetoplacenta.
The presence of hemodulition is an important criterion in the occurrence of this symptom contrasting with the usual hemo-concentration of pre-eclampsia, . [5,6].

b- Pathophysiology:

Feto-placental anasarca whatever the etiology + placental thickening → placental ischemia with production of anti-angiogenic factors → vascular dysfunction → pre-eclampsia

In this symptom unlike the usual pre-eclampsia:

- The placental lesions are late (secondary to fetal damage)
- There is arterial vasodilation
- Kidney biopsy is normal

. [6,7].

c- Clinical Diagnosis

Mirrored clinical picture with double hydrops:

1- in the fetus:

-rhesus alloimmunization
-sacrococcygeal teratoma, placental choriangioma Alpha
– thalassemia
- CMV or parvovirus B19 infection
ebstein's disease, supraventricular tachycardia

2- in the mother:

-Toxemia: moderate hypertension, proteinuria, edema
-The progression to eclampsia is unusual
-Generalized pruritus

The points in common:

-association of fetal hydrops and preeclampsia
-maternal manifestations less severe than those of the fetus
-disappearance of maternal symptoms when fetal manifestations disappear

d- Paraclinical diagnosis:

Biology:

Hemodilution
Anemia with drop in hematocrit
High level of HCG in relation to syncytiotrophoblast activity
Albuminuria
Hyperuricemia
In severe cases, hepatic cytolysis

Radiology:

Feto-placental anasarca on ultrasound
Normal uterine Dopplers. [8,9].

e- Differential diagnostics

The main differential diagnosis is **pre-eclampsia**, but in this case the placental involvement is primary,
There is a production of toxic factors for endothelial and renal cells, which results in severe endothelial lesions with nephropathy, and more severe hypertension.
On the biological level we note a hemo-concentration . [10,11].

f- Treatment :

Since the origin of mirror syndrome is fetal, the treatment will be based mainly on the management of the fetal condition:

- In case of alloimmunization: intrauterine transfusion
- Parvovirus B19: intrauterine transfusion
- Teratoma: extraction
- Supraventricular tachycardia: antiarrhythmic (flecaine, digitalis) . [12].

8- Conclusion:

- Mirror syndrome is often underdiagnosed by most clinicians.
- Any fetal anasarca associated with a maternal edematous syndrome should raise this diagnosis given that the fetal prognosis is reserved, and that the maternal consequences can be serious linked to the complications of secondary preeclampsia.
- Treatment depends on fetal care

Bibliography:

[1]. Ballantyne JW. The disease and deformities of the fetus. Edinburgh: Oliver and Boyd, 1892.
[2]. Delaby B, Lanta-Delmas S, Gondry J. Syndrome de Ballantyne résolutif après interruption sélective chez une grossesse gémellaire. J Gyn Obst Biol Reprod 2008;37:88-92.
[3]. Pirhonen JP, Hartgill TW. Spontaneous reversal of mirror syndrome in a twin pregnancy after a single fetal death. Eur J Obstet Gynecol Reprod Biol. 2004;116:106—7.
[4]. S. Proust, H-J. Philippe, A. Paumier, et al. Une pré-éclampsie en miroir : le syndrome de Ballantyne À propos de deux cas. J Gynecol Obstet Biol Reprod. 2006;35:270-274.
[5]O.Pourrat, V. Gouab, M. Maréchaud et al. Syndrome en miroir avec prééclampsie précoce sévère secondaire à un neuroblastome
surrénalien. La Revue de Médecine Interne. 2008;29:151.
[6]. Braun T, Brauer M, Fuchs I and al. Mirror Syndrome: A Systemic Review of Fetal Associated Conditions, Maternal Presentation
and Perinatal outcome. Fetal Diagn Ther. 2010;27:191-203.
[7]. Takahashi H, Matsubara S, Kuwata T and al. Maternal manifestation of Ballantyne‘s syndrome occurring concomitantly with the development of fetal congenital mesoblastic nephroma.J Obstet Gynaecol Res. 2014;40:1114-7.
[8]. O. Touhami, F. Chantraine, K. Delbecque et al. le cas clinique du mois le syndrome miroir : à propos d‘un cas. Rev med liège. 2013;68:440-3.
[9]. Bixel K, Silasi M, Zelop CM, and al. Placental Origins of Angiogenic Dysfunction in Mirror Syndrome. Hypertens Pregnancy. 2012;31:211-7.
[10]. Midgley DY, Harding K. The mirror syndrome. Eur J Obstet Gynecol Reprod Biol 2000;88:201-2.
[11]. Kontomanolis EN, Lambropoulou M, Tsagias N and al.The riddle of Ballantyne‘s syndrome in the aspect of hydrops fetalis. The
Journal of Maternal-Fetal & Neonatal Medicine. 2014;27:1172-3.
[12]. Banner S, Crossan D. Mirror Syndrome in Pregnancy: Two Patients, One Disease. Journal of Obstetric. Gynecologic & Neonatal
Nursing. 2013;42: 97-8.