

RESEARCH ARTICLE

ECLAMPSIA AT 16 WEEKS GESTATION ASSOCIATED WITH PARTIAL MOLAR PREGNANCY

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Manuscript InfoAbstractManuscript HistoryEclampsia is a complication of severe preeclampsia.It's commonly

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*Key words:-*Pre-Eclampsia, Hydatiform Mole, Placental Dysfunction defined as new onset of grand mal seizure activity and/or unexplained coma during pregnancy or postpartum in a woman with signs or symptoms of preeclampsia. It typically occurs during or after the 20th week of gestation or in the postpartum period. [1. 2].Otherwise, hydatidiform mole can be associated with very earlyonset preeclampsia. In both pathologies, various maternal symptoms arise from placental abnormalities. We present a very early case of eclampsia complicating a partial molar pregnancy associated with an exceptional Presssyndrom. Keyword: pre-eclampsia, hydatiform mole, placental dysfunction.

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Introduction:-

Eclampsia is a complication of severe preeclampsia.It's commonly defined as new onset of grand mal seizure activity and/or unexplained coma during pregnancy or postpartum in a woman with signs or symptoms of preeclampsia. It typically occurs during or after the 20th week of gestation or in the postpartum period. [1, 2].Otherwise,hydatidiform mole can be associated with very early-onset preeclampsia .In bothpathologies, various maternal symptoms arise from placental abnormalities.

We present a very early case of eclampsia complicating a partial molar pregnancy associated with an exceptional Presssyndrom.

Case:

A 23-year-old white woman, gravida 1, para 0, with no medical history and no family history of genetic abnormalities .she was admitted to the hospital after a generalized tonic-clonicseizure following an eclamptic fit at home at 16 weeks of pregnancy. Medical examination showed anunconscious patient witha grand mal seizure.Blood pressure was 160/10 mm Hg. Positive proteinuria was found on urine testing. There was no peripheral edema. The eclampsiaseizurestopped under treatment with magnesium sulphate and nicardipine, the blood pressure dropped progressivly to 130/90 mmHg. The ultrasound examination showed a viable fetus with no detectable abnormalities.The following day the patient regained fully consciousness.Biologicalinvestigations showed normal levels of: complete blood profile, electrolytes, urea, creatinine, ASAT/ALAT, coagulation studies .Cerebral computed tomographic scan showed no abnormalities .The b-HCG level was130 000IU/L.24H Proteinuria was at 2, 40g.The MRI showed a white matter signal abnormality under the right parieto-occipital cortex associated with a

Corresponding Author:- Y. Aitbenkaddour Address:- Department of Gynecology Obstetric, University Hospital Mohammed VI, Marrakech. signal abnormality of the right parietal white matter in T1 iso-signal, T2 hypersignal and FLAIR without translation on the diffusion sequence suggesting a PRES atypical unilateral syndrome.



Figure 1 : Atypic PRES syndrom. A-B : Axial FLAIR MRI Images shows a subcortical high signal intensity involving the right occipital lobe. C-D : Diffusion Weighted Images (B1000) : these lesions appear to be hyerintense Indicating restricted water diffusion.

Therapeutic termination of pregnancy has been suggested to thepatient and the induction was preformed after patient consent.

Fœtal description :a female fœtus with no visible malformation. weight:200g.



The Pathological study of the placenta revealed a partial molar pregnancy with defective remodeling of the spiral arteries.



The chorionic villi are bordered by a regular trophoblastic coating. The site of inconstant polar hyperplasia with some aspects of Bulbous dystrophy



A remodeling defect of some spiral arteries with a type of partial retention of the vascular musculature

The patient recovered well post-delivery and was discharged home on oral antihypertensive and LMWH for a few more days. The blood pressure settled over the next few days to 140/90 mm Hg without treatment. She was monitored carefully with bHCG levels which have steadily reduced to normal levels after three weeks.

Discussion:-

The incidence of hydatidiform mole with coexistent fetus is 0.005% to 0.01% of all pregnancies. It should be suspected when cystic placental changes are found in association with fetal malformations on ultrasonography. [7] Very few live pregnancies have been reported except in twin pregnancies with one surviving fetus and co-existing molar changes in the other sac (Marcorelles et al. 2005). Early neonatal deaths due to fetal anaemia or severe growth restriction have been reported. Although 90% of cases of partial mole are associated with triploidy.[8]

In the case presented molar vesicles were not seen on ultrasonography, and the fetus singleton seemed to have an appropriate growth at 16 weeks. The fetal karyotype in our case was not explored.

Hydatidiform mole and preeclampsia are two disorders unique to pregnancy. Placenta dysfunction is a common disorder in both pathologies. Therehave been very few studies on the molecular mechanisms that link hydatidiform moles with preeclampsia(PE) ,much regarding these mechanisms remains unknown. Many recent studies have demonstrated that placental dysfunction underlies the development of PE due to hypoxia elicited by defective invasion of the spiral arteries (9). Anti-angiogenic factors produced by trophoblast cells enter the maternal blood and induce PE symptoms. High levels of solublefms-like tyrosine kinase 1 (sFlt-1), an antagonistof vascular endothelial growth factor and placental growthfactor, have been found in women with PE,[10[11][12]. Enhanced expression of sFlt-1 has also been reported in the blood and placenta of patients with hydatidiform moles. These findings suggested that sFlt-1 may be involved in the underlying pathophysiological mechanism of PE subsequent to hydatidiform mole .The placental dysfunction plays a central role in the development of early-onset PE. Therefore, investigation of the pathological mechanisms associated with the development of maternal PE symptoms in hydatidiform mole may lead to the further clarification of the pathophysiology of placental abnormalities related to PE[13.14].

Although early preeclampsia in the second trimester is a frequent association with both partial and complete molar pregnancy, eclampsia is rare. We found only 58 cases of eclampsia in association with molar pregnancy reported in the literature since 1866. In only 10 of those was a fetus discovered concurrently. They noted that neurologic manifestations were present in a large majority of patients before the first seizure and that most patients experienced multiple seizures.[10]

Conclusion:-

Development of preeclampsia/eclampsia prior to 20 weeks of gestation should prompt a clinical evaluation to exclude the possibility of an underlying hydatidiform molar pregnancy.

References:-

[1] Mattar F, Sibai BM. Eclampsia. VIII. Risk factors for maternal morbidity. Am J Obstet Gynecol. 2000 Feb. 182(2):307-12.

[2] Douglas KA, Redman CW. Eclampsia in the United Kingdom. BMJ. 1994 Nov 26. 309(6966):1395-400.

[3] Ochiai D, Nakamura K, Sakurai T, et al. Atypical severe preeclampsia superimposed on chronic hypertension without molar change at 19 weeks of gestation :

A case report. Arch GynecolObstet 2012;286:1329-30.

[4] Sibai BM. Diagnosis, differential diagnosis and management of eclampsia. ObstetGynecol 2005;105:402-10.

[5]. Rahimpanah F, Smoleniec J. Partial mole, triploidy and proteinuric hypertension: two case reports. AustNZJObstetGynaecol 2000; 40:215-8.

[6] Sherer DM, Dalloul M, Stimphil R, et al. Acute onset of severe hemolysis, elevated liver enzymes, and low platelet count syndrome in a patient with partial hydatidiform mole at 17 weeks gestation. Am J Perinatol 2006;23: 163-6.

[7] Graham JM, Rawnsley EF, Simmons GM, et al. Triploidy: pregnancy complications and clinical findings in seven cases. Prenat Diagn 1989;9:409-19.

[8] Marcorelles P, Audrezet MP, Le Bris MJ et al. 2005. Diagnosis and outcome of complete hydatidiform mole coexisting with a live twin fetus. European Journal of Obstetrics, Gynecology and Reproductive Biology 118:21 – 27.

[10] Newmann RB, Eddy GL. Association of eclampsia and hydatidiform mole: case report and review of the literature. ObstetGynecolSurv 1988;43:185-90.

[9] Chaiworapongsa, T., Chaemsaithong, P., Yeo, L. & Romero, R. Pre-eclampsia part 1: current understanding of its pathophysiology.

Nature reviews. Nephrology 10, 466–480, https://doi.org/10.1038/nrneph.2014.102 (2014).4.Steegers, E. A., von Dadelszen, P., Duvekot, J. J. & Pijnenborg, R. Pre-eclampsia. Lancet 376, 631–644

[10]Levine, R. J. et al. Circulating angiogenic factors and the risk of preeclampsia. Te New England journal of medicine 350, 672–683,

Koga, K. et al. Elevated serum soluble fms-like tyrosine kinase 1 (sFlt1) level in women with hydatidiformmole.FertilSteril 94,

[11]305-308, https://doi.org/10.1016/j.fertnstert.2009.02.015 (2010).

Kanter, D. et al. Angiogenic dysfunction in molar pregnancy. American journal of obstetrics and gynecology 202, 184 e181–185,

https://doi.org/10.1016/j.ajog.2009.09.005 (2010)

[12]Kanter D, Lindheimer MD, Wang E, et al. Angiogenic dysfunction in molar pregnancy.Am J ObstetGynecol 2010;202:184.e1-5.

[13]Jennifer Uzan1Marie Carbonnel et al Pre-eclampsia: pathophysiology, diagnosis,

and management Vascular Health and Risk Management 85.46.84.158 on 30-Jul-2018

[14]Kaoru Kawasaki1 Eiji Kondoh1Live-born diploid fetus complicated with partial molar

pregnancy presenting with pre-eclampsia, maternal anemia, and seemingly huge placenta: A rare case of confined placental mosaicism and literature review 2016 Japan Society of Obstetrics and Gynecology; 69: 149–152.