Journal Homepage: - www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/12080
DOI URL: http://dx.doi.org/10.21474/IJAR01/12080

ISSN NO. 2320-5407

RESEARCH ARTICLE

OVARIAN HYPERSTIMULATION SYNDROMEAS A COMPLICATION OF MOLAR PREGNANCY: CASE REPORT

E. Lemrabott, B. Mohamed Mahmoud, K. Mohammed Saoud, N. Mamouni, S. Errarhay, C. Bouchikhi and A. Banani

Service de Gynécologie Obstétrique « I »CHU Hassan II, Fès.Marco Universite Sidi Mohamed Ben Abdellah Fes.

Manuscript Info

Manuscript History
Received: 20 September 2020
Final Accepted: 24 October 2020
Published: November 2020

Abstract

The authors present an atypical case of complete hydatidiform mole.Woman, 27years old, who went to the emergency room for abdominal pain associated with 10 week pregnancy. Echography revealed the presence of hyper stimulated ovaries leading to diagnosisof spontaneous Ovarian Hyperstimulation Syndrome (OHSS). Blood sample revealed high levels of hCG (300000 U/L), which associated with the ultrasound finding of placenta with vesicular areas, led to the suspicion of CHM,Dilatation and evacuation (D&E) were performed at 11 weeks of gestation. The signs and symptoms of OHSS were the severest on day 4 after D&E, when hCGhad already decreased We must be aware that OHSS can occur duringmolar pregnancy, and can be exacerbated after D&E.

Copy Right, IJAR, 2020,. All rights reserved.

Introduction:-

Ovary hyperstimulation syndrome(OHSS) is enlargement of the ovaries associated with fluid shifts secondary to ovulation induction therapy with clomiphene citrate or hCG.[1]. in its mild form, it is a common complication, seen in 5% to 10% of patients undergoing ovulation induction; the moderate form is reported in 2% to 4% of patients undergoing ovulation induction, and the severe form in 0.1% to 0.5%.2

In this syndrome, endothelial permeability increases, sometimes causing lung edema or severe hemoconcentration, with the latter sometimes resulting in a thromboembolic tendency, causing brain infarction [2]. Thus, OHSS can

Sometimes become life-threatening.

Although rare, OHSS can also occur even during naturally conceived pregnancy, especially when the level of hCG is high [3, 4]. OHSS has been reported to be associated with multifetal pregnancy and pregnancy complicated with hypothyroidism, with the former exhibiting high levels of hCG, and the latter having high levels of thyroid stimulating hormone acting similarly to hCG [4]. Molar pregnancy is another example [5]. We present a patient with molar pregnancy in whom OHSS occurred, and it worsened after pregnancy termination. We conducted a literature review to characterize this condition.

Case report:

A 27-year-old 2 parous naturally conceived woman was admitted with a diagnosis of a hydatidiform mole and ovarian masses at the 10th week of gestation. On admission, her blood pressure was 105/64 mmHg and pulse was 70 bpm, with white blood cells of 9,600/µL, hematocrit of 38.4 %, albumin of 3.3 g/dL and hCG of 300,000 IU/L.

Corresponding Author:-E.LEMRABOTT
ADDRESS:- Service de gynécologie obstétrique « I »CHU Hassan II, Fès. marco universite sidi mohamed ben abdellah fes.

Ultrasoundrevealed a “snowstorm” appearance strongly suggestive ofcystic swelling of the chorionic villi and proliferation of the trophoblastic epithelium (Fig. 1). Ultrasound also revealed bilateral ovarian cystic masses characteristicof OHSS (Fig. 2). After diagnosing this condition as hydatidiform mole (partial mole or complete mole) with OHSS, we performed dilatation and evacuation (D&E) at the 10th week of gestation, which yielded hydropic villi. Histological examination confirmed a diagnosis of complete mole.After D&E, when hCG had already decreased (9,830 IU/L, day 8 after D&E), the signs and symptoms of OHSS worsened.The ovaries were enlarged, (2) pleural effusion and ascites aggravated, and (3) she gained weight. All these were the severest on day 4 after the initial D&E. At 17 days after the first D&E, all three manifestations disappeared and she was discharged. At 1 month after D&E, the ovaries became normal in size without OHSS signs and/or symptoms. The serum hCG level decreased,Dropping below the detection level (under cut-off) 20 weeks after the initial Dilatation and evacuation.

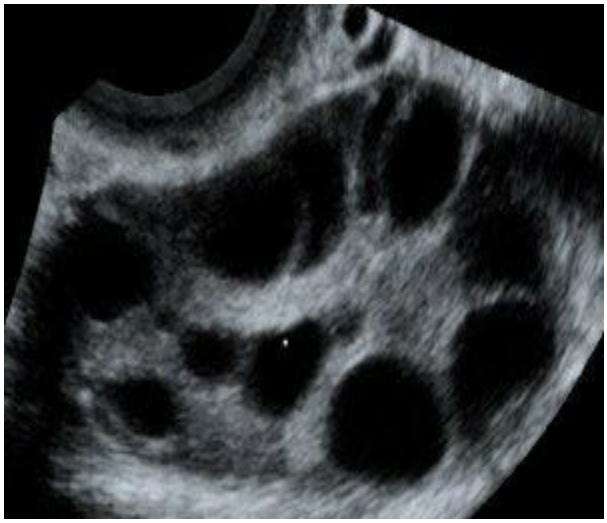

Fig.1:- Ovary with hyper stimulation

Fig.2:-completehydatidiform mole appearance

Discussion:-

OHSS can occur during molar pregnancy without ovulation induction. Since this syndrome is usually accompanied by a high level of hCG, it usually occurs at ovulation induction with hCG, and it is generally considered to be an adverse event of infertility treatment. Besides hCG, vascular endothelial growth factor (VEGF) has also been considered an important factor associated with this syndrome [6, 7]. In short, hCG, in some cases, hyperstimulates ovarian follicles. They secrete some angiogenetic factors, whichincrease the endothelial permeability [4]; of those factors, VEGF has attracted wide attention [6–7].

OHSS accompanying molar pregnancy are similar to those of OHSS accompanying infertility treatment. The initial manifestation of OHSS occurred at a median of the 12th week of gestation (range 7–16),

which may be later compared with OHSS caused by ovulation induction. Excluding this, manifestations and/or features characteristic of OHSS accompanying molar pregnancy may not be present. Any type of molar pregnancy, i.e., complete, partial, or invasive, can accompany OHSS.

In our patient, OHSS was secondary to molar pregnancy and markedly elevated hCG levels. Hydatidiform mole or molar pregnancy is a cystic swelling of the chorionic villi and proliferation of the trophoblastic epithelium. Elevated circulating hCG is thought to lead to ovarian enlargement and multiple cysts; this stimulates the ovaries to secrete vasoactive substances, increasing vascular permeability, leading to fluid shifts and the accumulation of extravascular Fluid, resulting in renal failure, hypovolemic shock, ascites, and pleural and pericardial effusions. [8]

This acute shift produces hypovolemia, which may result in multiple organ failure, hemoconcentration (hematocrit > 45%), thrombosis, and disseminated intravascular coagulation from the increased viscosity of the blood, Factors associated with a high risk of developing OHSS include young age, low body weight, polycystic ovary syndrome, a high serum estradiol level, and a history of OHSS.[9]

Grading Of Ohss Is Based On:
Symptoms, Test Results, Imaging:
The severity of OHSS is classified as mild, moderate, or severe, with further grading as follows[8-10]:

Mild OHSS:
Grade 1:Abdominal distention and discomfort.
Grade 2: features of grade 1, plus nausea and vomiting, with or without diarrhea, and ovarian size of 5 to 12 cm.

Moderate OHSS:
Grade 3:Mild OHSS with imaging evidence of ascites.

Severe OHSS:
Grade 4:Moderate OHSS plus clinical evidence of ascites, with or without hydrothorax.
Grade 5: all of the above plus hypovolemia,hemoconcentration (hematocrit> 45%), Coagulation abnormalities, and oliguria.
Grade 6: all the features of grades 1 to 4 plus hypovolemia, hemoconcentration (hematocrit> 55%), anuria, renal failure, venousthrombosis, and adult respiratory distress syndrome. This can be life-threatening and may require hospitalization.

Treatment:
Treatment is generally conservative and includes management of ascites and pleural effusion and supportive care. Mild OHSS can be treated on an outpatient basis with bed rest, oral analgesics, limited oral intake, and avoidance of vaginal intercourse, and usually resolves in 10 to 14 days. Moderate and severe OHSS require bed rest and aggressive fluid resuscitation. OHSS in patients with renal failure, relentless hemoconcentration, or thrombovascular accident can be life-threatening and may require intensive- care monitoring. Paracentesis may be performed if tension ascites and oliguria or anuria develop 2Prophylactic anticoagulation with warfarin, heparin, or low molecular- weight heparin is indicated in women with a high tendency for thrombotic events who develop moderate to severe OHSS.[11],Surgical intervention may be necessary in patients with ectopic pregnancy, ovarian torsion, or ruptured ovarian cyst. Our patient was treated conservatively with supportive care and experienced a full recovery.

Conclusion:-
Hydatidiform mole diagnosis requires a high grade of suspicion. Quick intervention allows a better clinical resolution and it might avoid the development ofGestational Trophoblastic Neoplasia GTN. Spontaneous OHSS is a possible complication of hydatidiform mole. With this in mind, it is fundamental to understand the pathophysiology of spontaneous OHSS to manage this clinical entity. In our opinion that was due to a quick diagnosis and treatment. To conclude, it is important to remember that in cases of spontaneous OHSS, Hydatidiform Mole should be excluded. Communication between Endocrinology and Obstetrics services is fundamental to achieve control of thyroid functionand resuscitation for better care.

References:-
1. Arora R, Merhi ZO, Khulpateea N, Roth D, Minkoff H.Ovarian hyperstimulation syndrome after a molar pregnancy evacuation. FertilSteril 2008; 90:1197.e5–e7.
2. Alper MM, Smith LP, Sills ES (2009) Ovarian hyperstimulation syndrome: current views on pathophysiology, risk factors, prevention,and management. J ExpClin Assist Reprod 6:3. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2868304/. Accessed 29 May 2014
3. Nastri CO, Ferriani RA, Rocha IA, Martins WP (2010) Ovarian hyperstimulation syndrome: pathophysiology and prevention. J Assist Reprod Genet 27:121–128
4. Delbaere A, Smits G, Olatunbosun O, Pierson R, Vassart G, Costagliola S (2004) New insights into the pathophysiology of ovarian hyperstimulation syndrome. What makes the difference between spontaneous and iatrogenic syndrome? Hum Reprod 19:486–489
5. Zhou X, Duan Z (2012) A case of ovarian hyperstimulation syndrome following a spontaneous complete hydatidiform pregnancy. GynecolEndocrinol 28:850–852
6. Strafford M, Morno-Ruiz N, Stubblefied P (2009) Ovarian hyperstimulation syndrome in a spontaneous pregnancy with a complete hydatidiform mole. FertilSteril 92: 395.e1–3

7. Ludwig M, Germbruch U, Bauer O, Diedrich K (1998) Ovarian hyperstimulation syndrome (OHSS) in a spontaneous pregnancy with fetal anf placental triploidy: information about the general pathophysiology of OHSS. Hum Reprod 13:2082–2087
8. Whelan JG 3rd, Vlahos NF. The ovarian hyperstimulation syndrome. FertilSteril 2000; 73:883–896.
9. Mor YS, Schenker JG. Ovarian hyperstimulation syndrome and thrombotic events. Am J ReprodImmunol 2014; 72:541–548.
10. Golan A, Weissman A. Symposium: update on prediction and management of OHSS. A modern classification of OHSS. Reprod Biomed Online 2009; 19:28–32.
11. Practice Committee of American Society for Reproductive Medicine. Ovarian hyperstimulation syndrome. Fertil Steril 2008; 90(suppl):S188–S193.