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Case Report

Ovarian Hyperstimulation Syndrome: Severe Form (About a Case)

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Summary

Ovarian hyperstimulation syndrome is an iatrogenic complication of assisted reproductive technology and is characterised by cystic ovarian hypertrophy and fluid displacement from the intravascular space to a third space due to increased capillary permeability and ovarian neoangiogenesis. The mortality rate is not zero and is mainly related to the occurrence of thromboembolic accidents with the particularity of affecting both venous and arterial territories. Severe forms may require prolonged hospitalization in intensive care and the curative treatment is purely symptomatic.

Keywords: OHSS; Latrogenic; Complications; Thrombosis; Severe

Introduction

Ovarian Hyperstimulation Syndrome (OHSS) is an iatrogenic complication of assisted reproductive technologies. The syndrome is characterised by cystic ovarian enlargement and fluid displacement from the intravascular space to the third space due to increased capillary permeability and ovarian neoangiogenesis. Its occurrence is dependent on the administration of Human Chorionic Gonadotropin (hCG). The clinical picture varies in severity from simple ovarian enlargement to cardiovascular collapse. Mortality is mainly related to the occurrence of thromboembolic events.

The clinical picture varies in severity from a simple increase in ovarian size to cardiovascular collapse. Mortality is mainly related to thromboembolic events. The severe form is found in 0.5 to 5% of ovarian stimulations [1]. Severe forms may require prolonged hospitalization in intensive care because of life-threatening complications. In the context of the constant increase in the practice of MAP, this rare complication could become frequent. Through this clinical case we will detail the different clinical manifestations of the severe forms of this syndrome as well as the modalities of management in the intensive care unit.

Methods

Clinical Case

32 years old women with no medical history, developed dyspnea with abdominal pain 4 days after an ovarian puncture.

On admission the clinical examination revealed a conscious patient, tachypneic at 25 Cycles/min with 100% room air saturation, heart rate of 105 beats per minute, and blood pressure of 110/80mmHg without signs of hypoperfusion.

Biological evaluation revealed an inflammatory syndrome, with hyperleukocytosis at 20G/l, CRP at 6mg/l, D dimers at 6000ng/ml, normal troponin, acute renal failure with creatinine at 120umol/l, hyperkalemia at 5.7mmol/l, hyponatremia at 130mmol/l with hepatic cytolysis at 3-4 times normal.

Haemoglobin level at 12.9g/dl with a hematocrit of 40.1% Ultrasound shows a peritoneal effusion. A thoracic and abdominal angioscan was performed which showed no pleural effusion or pulmonary embolism with the presence of a large abdominal effusion.

The treatment was based on the correction of the blood vol-

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Table 1:

Clinical forms of OHSS	Symptoms
Mild	Abdominal discomfort or pain, vomiting, diarrhoea, increased ovarian volume but <12cm in diameter on ultrasound
Moderate	Serous effusion, haematocrit between 40-55%. Ovarian size >12cm in diameter
Severe	Tension ascites with or without pleural effusion, thromboembolic complication, ARDS, renal failure, Oligo-anuria/hematocrit >55% or white blood cells >25000/mm3, hepatic cytolysis, hyponatremia, hyperkalemia.

ume with cristaloids and albumin. The introduction of a multimodal analgesia for the abdominal pain. And in front of the major abdominal distension in addition to the renal repercussion, drainage of the liquid of ascites was made with compensation by albumin.

Furosemides in titration were used in the face of hyperkalaemia, ascites with a clear improvement in the patient's diuresis and a normalisation of hydroelectrolytic disorders, with strict biological monitoring of signs of hemoconcentration.

Strong preventive anticoagulation with the use of compression stockings while monitoring neurological and respiratory signs.

Our patient remained apyretic; a bacteriological study was done for the ascites fluid and came back negative.

After 10 days of hospitalization, the patient was weaned from furosemides, the renal function corrected, the liver balance improved.

Discussion

Ovarian hyperstimulation syndrome is a potentially fatal complication and can occur in up to 5% of patients undergoing in vitro fertilisation [2]. It is an iatrogenic complication of ovulation induction therapy and infertility treatments such as Clomiphene Citrate (CC), Human Menopausal Gonadotropin (hMG), Although OHSS occurs most often as an iatrogenic complication of ovulation induction, spontaneous ovarian hyperstimulation syndrome has been reported in rare cases during pregnancy [14,15].

Ovarian hyperstimulation syndrome occurs during the luteal phase of the induced menstrual cycle, on average 5-10 days after the start of stimulation. It is characterised by cystic enlargement of the ovaries and increased capillary permeability with plasma extravasation to the interstitial sector. These anomalies, depending on their severity, will cause a more or less significant oedematous syndrome with hydroelectrolytic disorders, haemoconcentration, coagulation disorders, and also sometimes hepatic and renal damage.

Certain factors were associated with an increased risk of developing OHSS: age <35 years, polycystic ovaries, higher concentration of Estradiol (E2) and large number of small follicles (8-12 mm) found by ultrasound during ovarian stimulation [16].

Two forms can be described: early OHSS related to ovulation induction and late OHSS related to pregnancy. Our study concerns the early form: onset of symptoms on the 4th day following ovarian puncture, exogenous hCG is responsible for this form. In the late form, in addition to exogenous hCG, endogenous hCG linked to pregnancy is added, which makes the symptoms more severe.

Three clinical grades of OHSS are distinguished, depending on symptomatology, biological findings and ultrasonographic findings. These are: mild, moderate and severe; the latter is life-threatening and will therefore involve the resuscitator. These clinical grades are sometimes difficult to distinguish (Table 1).

The incidence of OHSS has been estimated at 20-33% for mild cases, 3-6% for moderate cases and 0.1% and 2% for severe cases [17].

In our study the criteria of severity were: ascites and then the onset of acute renal failure with hydroelectrolytic disorders.

For most authors [4,5] the initial mechanism of the syndrome is an increase in capillary permeability. The pathophysiology of the symptoms can be explained by a circulatory dysfunction secondary to this permeability disorder. For other teams, a marked arterial vasodilatation would be the initiating mechanism of SHSO [6]. The increase in capillary permeability and/or arterial vasodilatation leads to a mobilisation of fluid from the intravascular sector towards the interstitial sector. This massive accumulation of exudates [7,8] can lead to the formation of significant ascites, pleural and/or pericardial effusion, hydroelectrolytic abnormalities, acute oliguric renal failure, haemoconcentration and hypovolaemia that may be haemodynamically poorly tolerated [15].

On the biological side, several abnormalities can be observed that have prognostic values including haemoconcentration and global inflammatory status. Note that a haematocrit greater than 55% places the patient in the life-threatening group [8.9], the elevated haematocrit should be considered as a marker of severity, as there is a good correlation with the level of plasma volume-dependent vasoactive substances. C-Reactive Protein (CRP) can also be used as a marker for monitoring the condition and/or as a marker of severity as demonstrated in a retrospective study of 19 women with moderate to severe OHSS with a correlation between ovarian size measured on ultrasound and CRP level [10].

SHSO is often associated with oliguria and acute renal failure with decreased urinary sodium excretion and hyponatremia indicative of plasma hypoosmolality [11].

Metabolic acidosis and hyperkalaemia may be related to decrease urinary sodium excretion. Severe forms of SHSO are marked by an increase in plasma creatinine [2].

A disturbance of the liver balance is found in 30% of severe SHSOs [2], but it corrects itself without sequelae after the improvement of the picture. It is most frequently a cytolysis that can sometimes be associated with an increase in gamma-glutamyl-transferase or alkaline phosphatases [13].

Conclusion

Severe forms of OHSS are rare but potentially lethal and must be known. A better understanding of the pathophysiology of OHSS, dominated by capillary hyperpermeability, currently allows conservative medical treatment to be envisaged. Nevertheless, prevention and early recognition of OHSS remain the best guarantees of effective management of patients at risk or who have it.

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