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Isolated Pericardial Effusion Revealing an Ovarian Hyperstimulation Syndrome: about two Cases

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ABSTRACT

Ovarian hyperstimulation syndrome is the most severe iatrogenic complication of modern in vitro fertilization. It manifests itself as enlarged ovaries with many follicular cysts and is associated with a redistribution of body fluids. In most cases, this redistribution of liquids leads to the development of as cites. In some rare cases, it gives a massive unilateral pleural effusion without ascites and very rarely as a pericardial effusion.

We report the cases of two patients who developed ovarian hyperstimulation syndrome that initially manifested itself in a pericardial effusion of great abundance

KEYWORDS: Ovarian hyperstimulation syndrome - Pericardial effusion- fertilization

I. INTRODUCTION

Ovarian hyperstimulation syndrome is the most severe iatrogenic complication of modern in vitro fertilization [1]. The number of severe cases of ovarian hyperstimulation after ovarian stimulation is estimated around 2.7% [2]. This syndrome is expressed by multiple ovarian cysts as well as in the redistribution of body fluids that form a third sector. Fluid redistributions occur as ascites (87%), pleural effusion (21%), pericardial effusion (3%); and hemoconcentration in 71% of cases [3].

II. CASE REPORT

A. First case report:

We report the case of a 36-years-old female patient with no specific history. She was admitted to the emergency department for stage IV dyspnea according to the New York Heart Association (NYHA). There was no fever, nor chest pain, or even a hemoptysis, but the patient complained of suprapubic abdominal discomfort.

She had received ovarian stimulation in accordance with the long agonist protocol 15 days earlier in order to perform an in vitro fertilization.

It consisted in 14 days of administration of a gonadorelin agonist (Gnrh) (Triptorelin, Decapeptyl®) at a subcutaneous dose of 0.1 mg, followed by an injection of FSH (follitropin alpha, Gonal-F®) at a dose of 150 IU per day for 12 days subcutaneously.

Ovulation was then initiated by recombinant chorionic gonadotropin hormone (hCG) (choriogonadotropin alpha, Ovitrelle®) at the single dose of 500 µg subcutaneously. The clinical examination revealed that the patient was apyretic (37°C), tachypneic, and tachycardic at 120 cpm, with a blood pressure of 100/65 mmHg. Cardiac auscultation revealed a decrease in heart sounds, with no sign of right heart failure. The abdomen was soft, tympanic, and painless and the pleuropulmonary examination was normal.

The biology showed an hemoglobin rate of 11,4 g/dl , hemoglobin A1c at 40,4 %, and normal liver function testing. It was noted that there was an inflammatory syndrome (C-reactive protein at 15,7 mg/l , a sedimentation rate of 30). The β-hCG and oestradiol hormons were respectively containing 204 UI/l and 3,799 ng/l (standard : 24 to 195 ng/l). Because of an ongoing pregnancy, the radiological investigation was avoided. A transthoracic echocardiography was realized, finding an important pericardial effusion whitout any sign of collapse (Figure 1). The abdominal echography showed big ovaries bilaterally, with many follicular cysts, and a millimetric ascites in the pouch of Douglas.

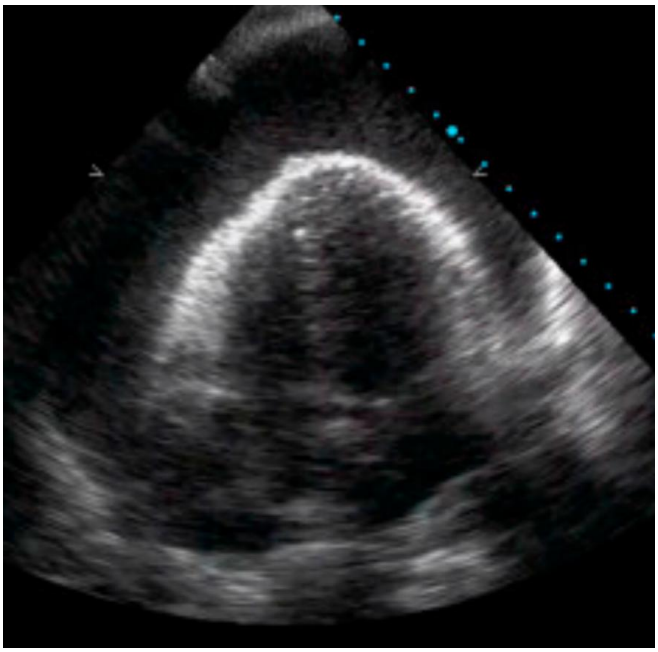

Figure 1. Echocardiographic image showing a significant pericardial effusion

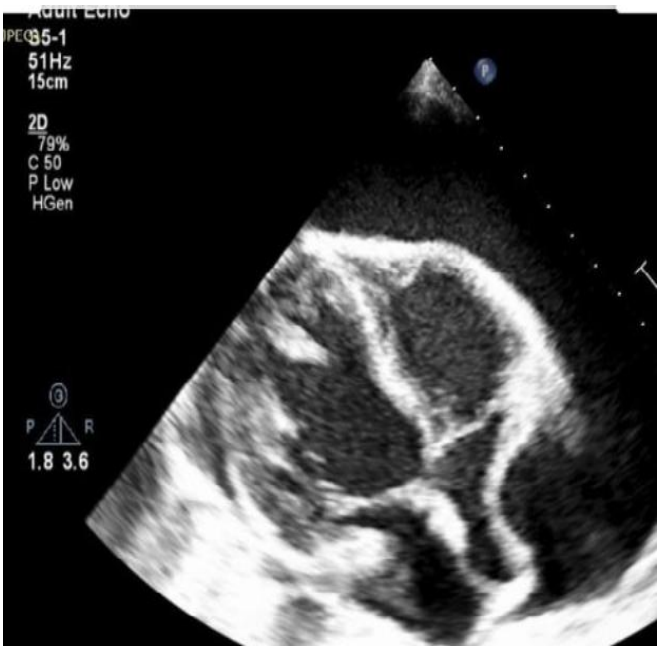

Figure 2. Echocardiographic image showing an important pericardial effusion with collapse of the right atrium

The diagnosis of the ovarian hyperstimulation syndrome is based on clinical criteria (especially the clinical and paraclinical context). The decision consisted on a strict clinical and echocardiographic monitoring. The patient remained stable in terms of cardio-vascular state, with an echocardiography of control, which showed a decrease of the effusion, becoming low, and a retention of the pregnancy until the 28th week of amenorrhea, with a vaginal delivery. The premature newborn died a few hours after the delivery because of a respiratory distress.

B. Second case report:

We report a case of a cardiac tamponade in a 30 years old woman after ovarian stimulation (14-day administration of a gonadorelin agonist (GnRH) (Triptorelin, Decapeptyl®) at a dose of 0.1 mg subcutaneously, followed by a subcutaneously injection of FSH (follitropin alpha, Gonal-F®) to the dose of 150 IU daily for 12 days. Ovulation was then triggered by recombinant chorionic gonadotropin (hCG) (choriogonadotropin alpha, Ovitrelle®) at a single dose of 500 µg subcutaneously.

Fourteen days after that, she developed a worsening dyspnea and was admitted to the intensive care unit with acute respiratory failure. The heart rate was 130 beats per minute, the blood pressure was 105/85 mm Hg with pulsus paradoxus, and the respiratory rate was 40 breaths per minute. The oxygen saturation on oximetry was 88% while the patient was receiving nasal oxygen (at a rate of 10 liters per minute).

Chest auscultation showed decreased breath sounds. Transthoracic two-dimensional echocardiography revealed a large anterior pericardial effusion with respiratory variation in transvalvular flow on Doppler imaging and impaired right atrial and ventricular filling (Figure 2).

Pelvic ultrasonography showed bilateral ovarian enlargement with presence of ascites of low abundance. Pulmonary ultrasonography revealed bilateral pleural effusion at the level of the pulmonary bases. Emergency percutaneous drainage of the subxiphoid pericardial cavity recovered 450 ml of clear fluid.

The patient's condition improved dramatically, and she was weaned from the ventilator 1 hour later. The plasma level of human chorionic gonadotropin beta subunit was 127 IU per liter. The patient was discharged from the intensive care unit on day 11 but had a spontaneous abortion on day 30.

III. DISCUSSION

Ovarian hyperstimulation syndrome is the most severe iatrogenic complication of modern fertilization methods. This syndrome is characterized by a massive cystic ovarian enlargement associated with an acute body fluid shift. Ascites is the most frequent manifestation of this syndrome. In some rare cases, ovarian hyperstimulation syndrome is complicated by massive unilateral pleural effusion without ascites and very rarely by a pericardial effusion without ascites.

According to Delvigne and al. [4], different risk factors enable the development of this syndrome such as the young age of patients, a low body mass index and a history of ovarian hyperstimulation or of pregnancy. The atopic patients and those who have micropolycystic ovaries are a population at risk. Among the different protocols, a stimulation with an agonist protocol seems to face higher risk than the antagonist one.

The agonist protocol consists in inhibiting the ovulation and putting the ovaries at rest using the GnRH agonist on the first day of menstrual bleeding. It consists afterwards in stimulating the ovaries fourteen days later with a FSH

analogue [5]. In the antagonist protocol, we stimulate the ovaries with a FSH analogue and then we inhibit the ovulation with a GnRH antagonist 6 to 8 days later [6].

In case of administration of high dosage of exogenous HCG for inducing the ovulation, we notice the increase in the frequency of the ovarian hyperstimulation syndrome. The high level of the œstradiol basic rate in the blood and a late peak after stimulation are also correlated with the onset of this syndrome.

The redistribution of body fluids encountered in the ovarian hyperstimulation syndrome is secondary to the increase of the vascular permeability, which is dependent of the release of vasoactive factors by the ovary such as the Vascular Endothelial Growth Factor (VEGF) [7].

A relationship between the administration of HCG and the expression of VEGF's messenger RNA has been reported. The VEGF increases the vascular permeability and the passage of macromolecules by promoting the mitosis of the endothelial cells, and thus, induces to an exudate in the serous membranes [7].

The interleukin-6 has also been correlated to this syndrome by an increase of the capillary permability, and especially, by a decrease of the production of albumin in the liver. The decrease of the oncotic pressure, secondary to the decrease of albumin, and the increase of the VEGF promote these fluid exchanges according to the Frank–Starling law [7].

High rates of VEGF and IL-6 were found in the ascites, the pleural effusions, and in the serum of patients who are carrying the ovarian hyperstimulation syndrome [7, 8]. However, the role of the VEGF predominates, which explains the exuding nature of the effusions and of the ascites.

In the two cases described above, there was no significant quantity of ascites, but the patients had an abundant pericardial effusion.

We are not aware of previous reports of the ovarian hyperstimulation syndrome with cardiac tamponade and bilateral pleural effusions. Rare cases of isolated right sided pleural effusion have been described [9]. In a Belgian multicenter study of 128 patients with the ovarian hyperstimulation syndrome, only four patients (3%) had a pericardial effusion, without tamponade. Because individual responses to the induction of ovulation are unpredictable, prevention of the ovarian hyperstimulation syndrome is difficult.

In 2003, a total of 122,872 assisted reproduction procedures were reported in the United States [10]. The widespread use of such procedures underscores the need for recognition of the complications. Physicians should be aware that cardiac tamponade is a rare but life threatening potential complication of the ovarian hyper stimulation syndrome.

IV. CONCLUSION

The ovarian hyperstimulation syndrome is a potentially lethal complication of the administration of gonadotropin used for the induction of ovulation. This condition occurs in up to 5% of patients undergoing in vitro fertilization. Typical manifestations include marked ovarian enlargement associated with shifts in extravascular fluid, leading to ascites and pleural and pericardial effusions.

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