Bilateral Pleural Effusion as Unique Manifestation of Atypical Ovarian Hyperstimulation Syndrome

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Abstract

Ovarian Hyperstimulation Syndrome (OHSS) is a complication associated with In Vitro Fertilization (IVF) treatment. The clinical manifestations are due to an increase of vascular permeability and extravasations of fluid in a third space. Herein we report an unusual case of OHSS revealed by an isolated bilateral pleural effusion. A 37-year-old patient presented with dyspnea and orthopnea in addition to dry cough and chest pain 2 weeks after an IVF cycle with 13 oocytes retrieved, E2: 2435 pg/ml and 2 cleavage stage embryo transferred. Patient was afebrile, tachycardic, tachypneic with normal oxygen saturation. Decreased breath sounds of both lung bases were detected. Laboratory tests showed Albumin 2.8 g/dl, Human Chorionic Gonadotropin (hCG) 1494 mU/ml, fibrinogen > 500 mg/dl, D-Dimer 666 ng/ml. Hematocrit, renal and liver function tests were in normal range. Electrocardiogram was normal. Transvaginal sonography did not show free fluid. Chest X-ray revealed bilateral pleural effusion and angio-CT discarded Pulmonary Embolism (PE). Patient was admitted to the intensive care unit for seven days. She was symptomatically treated with intravenous diuretics and albumin, oxygen and low molecular weight heparin for thromboembolic prophylaxis. Patient remained hemodynamically stable, afebrile, with normal urine output and negative fluid balance, with improvement in respiratory symptoms, so that oxygen therapy was reduced and chest tube was symptomatically treated with intravenous diuretics and albumin, oxygen and low molecular weight heparin for thromboembolic prophylaxis. Patient remained hemodynamically stable, afebrile, with normal urine output and negative fluid balance, with improvement in respiratory symptoms, so that oxygen therapy was reduced and chest tube...
that pleural effusion could be secondary to transfer of ascites fluid
from the vascular space to third space, with intravascular volume depletion, hy-
potension, oliguria, ascites and hemoconcentration [4]. It is proposed
that the vascular space can be disrupted in OHSS by a variety of mediators
such as: vascular endothelial growth factor (VEGF), produced by granulosa cells,
which depends on hCG [2,4,7,8]. Although the etiology of OHSS is unknown,
the physiopathology is an increase of vascular permeability to high molecular weight
proteins due to mediators such as VEGF, produced by granulosa cells, which depends on hCG
[2,4,7,8]. This causes displacement of fluids and proteins from intra-
vascular space to third space, with intravascular volume depletion, hy-
potension, oliguria, ascites and hemoconcentration [4]. It is proposed
that pleural effusion could be secondary to transfer of ascites fluid
through the diaphragmatic lymphatic or diaphragmatic anatomical defects, which are more frequent on the right side of the dia-
aphragm [6-12].

Predisposing factors for OHSS includes: age under 35 years, num-
ber of follicles over 15, plasma concentration of estradiol higher than
3,000 pg/ml, polycystic ovary syndrome and pregnancy [10,11,13]. None of them was relevant in our case and atypical OHSS could only
be associated to pregnancy and a particular patient reliability. Given
the possible recurrence of the syndrome in future cycles of stimulation
in this patient, it will be necessary to consider preventive measures,
such as: cancel the cycle before administering hCG with less follicles
or lower levels of estradiol to the previous cycle; reduce dose of hCG:
embryo cryopreservation to avoid the production of endogenous
hCG; and delayed transfer [8,14]. Other secondary prophylaxis strat-
egies (albumin administration, corticosteroids or dopamine agonists)
are controversial [14,15].

Conclusion
Isolated pleural effusion is an uncommon feature in severe cases
of OHSS. It is a life-threatening complication that has to be differenti-
ated from pulmonary embolism, a common complication associated
with hyperestrogenism situations [11]. Physicians should be aware of
this complication and keep in mind that it may occur without other
most common symptoms. Early diagnosis and treatment is crucial to
enhance patient recovery. We must prevent its occurrence analyzing
the risk factors of each patient and developing an individualized treat-
ment plan.

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