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Ovarian Hyperstimulation Syndrome (OHS): A Rare Cause of Acute Renal Failure: A Case Report

Dr. Djiguiba Karamoko[1,2*], Coulibaly A[1], Atabieme K[3], Sy Seydou[3,4], Ibrahimi A[2], Bénamar L[2], Rhou H[2], Ezzaitouni F[2], Yattara H[3,4], Ouzzedoune N[2], Bayahia R[2]

[1]Service Nephrology and Hemodialysis of Peritoneal Dialysis and Kidney Transplantation, Hospital Luxembourg of Bamako in Mali
[2]Department of Nephrology and Hemodialysis of Peritoneal Dialysis and Kidney Transplantation, CHU Ibn Sina (Faculty of Medicine) of Rabat-Morocco
[3]Department of Nephrology and Hemodialysis of Peritoneal Dialysis and Kidney Transplantation, CHU –Point G of Bamako in Mali
[4]University of Sciences Technic and Technology of Faculty Medicine and Bamako in Mali

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***Corresponding author:** Dr Djiguiba Karamoko
Medical Doctor, MD Nephrology of Luxembourg Hospital Bamako Mali

Abstract **Case Report**

We report in this work, the evolution and management of a case of ovarian hyperstimulation syndrome (OHS) complicated by severe acute renal failure (ARI). A young 31-year-old patient pregnant with twins after 17 weeks of amenorrhea was admitted to nephrology department for rapidly progressive acute renal failure (AKI) (creatinine increases in 48 hours from 28mg/l to 55mg/l) and was complicated by major hydro-electrolyte disorders and multi-organ failure. Her past medical history revealed an ovulation induction before her last pregnancy and a spontaneous abortion seven years ago before her hospitalization which occurred in the 2nd trimester of her 1st twin pregnancy obtained by medically assisted reproduction. Despite optimal care, the patient improves her renal function and her multiorgan failure after abortion in gynecological intensive care. Because of worldwide increase of infertility cases and the use of new ovulation induction techniques with these risks of serious complications, we report this interesting clinical case of OHS.

Keywords: ovarian hyperstimulation syndrome (OHS); acute renal failure (ARI).

1- INTRODUCTION

Ovarian hyperstimulation syndrome (OHS) is an iatrogenic complication that can occur during ovarian stimulation, or during medically assisted reproduction techniques (MAR) for the treatment of infertility [1]. This complication is a rare pathology when it occurs spontaneously. It is a very serious complication, because it is generally associated with significant maternal and infant morbidity and mortality.

OHS causes an increase in the ovaries volume, ascites, oliguria, hemoconcentration, and hydro-electrolyte disorders which can be life-threatening in the young patients treated.

Its pathophysiology is currently poorly understood. But it is necessary to identify its risk factors in this population of young women to avoid its several complications. Among these complications we have thromboembolic events, dysthyrodism and acute renal failure with hydro-electrolyte disorders.

An etiological investigation must be the rule in all cases of OHS. Its management should be empirical, and symptomatic, to improve the renal perfusion and to prevent the occurrence of thromboembolic events [2, 3].

This work reports a case of ovarian hyperstimulation syndrome (OHS) after treatment with ovulation inducers in a 31-year-old pregnant woman with twin pregnancy after 17 weeks of amenorrhea complicated by rapidly progressive acute renal failure.

2- CLINICAL OBSERVATION

The patient was referred to us by her gynecologist for rapidly progressive acute renal failure (increasing creatinine from 28mg/l to 55mg/l in 48 hours).

Mrs. H.R, a 31-year-old young patient, with a twin pregnancy of 17 weeks of amenorrhea, after having received a protocol for ovulation induction consisting of a dose of gonadotropin (HMG) associated with Decapetyl.

Her past medical history includes a spontaneous abortion occurring in the 2nd trimester of her 1st pregnancy obtained by medically assisted reproduction (MAR) 7 years ago and Graves' disease treated with carbimazole (neomercazole) and propranolol (Avlocardyl) .

Clinical examination at the entrance to the emergency room, we noted on the functional level, a very marked asthenia, an alteration of his general condition associated with incoercible vomiting and orthopnea. Physical examination revealed mucocutaneous pallor. The cardiopulmonary examination found normal blood pressure at 120/60mmHg in a half-sitting position, tachycardia 130 heartbeats per minute and polypnea at 30-minute cycles. Pulmonary auscultation revealed bilaterally lung bases crackles going up to the tops. Abdominal examination showed minimal ascites with audible fetal heart sounds on auscultation. In addition, absence of lower limbs edema and the presence of clinically visible goiter. The rest of our examination was normal.

Her laboratory test investigations revealed a multi-visceral failure with a rapidly progressive acute renal failure (plasma creatinine goes up from 28 mg/l to 55 mg/l in 48 hours) associated with major hydro-electrolyte disorders with severe hyperkaliemia at 7.4 meq/l, true hypoosmolal hyponatremia at 122 meq/l, without metabolic acidosis. Urinary test results showed sodium-potassium ratio < 1, moderate proteinuria at 0.6g/24h without microscopic hematuria and a urinary infection with Escherichia coli.

An abdomino-pelvic ultrasound performed urgently showed no obstruction of the urinary excretory tract, and the kidneys were of normal size, well differentiated, without pyelocaliceal dilatation and non multicystic ovaries.

The blood count found severe anemia with a hemoglobin level of 7.4g/dl of normochromic normocytic non- regenerative type and hyperleukocytosis at 18,700/mm3 with neutrophil predominance. Platelets count was normal. A thyroid assessment showed hyperthyroidism with an elevated T3 at 6.97pmol/l and an elevated T4 at 4.63 pmol/l, but the ultra-sensitive TSH was zero.

Immunologically, the complement fractions of C3 and C4 were normal. The anti-DNA, AAN, ANCA type MPO and PR3 Abs were all negative. On the inflammatory level, the CRP was at 148mg/l, an accelerated sedimentation rate at the 1st hour at 30mm and at the second hour at 60mm.Hyperferritinemia at 1718 ng/ml was noted. Moreover, in the context of her multi-visceral failure, hepatic cytolysis syndrome was found with a level of ALT=138UI/l and AST=107UI/l, a cholestasis syndrome of GGT at 103UI/l and hepatic ALP at 121UI/l. A rhabdomyolysis with a CPK level at 1081UI/l associated with an LDH of 503UI/l. The schistocytes and haptoglobin were normal in her hemolysis test. Viral B and C serologies and toxoplasmosis were negative, as well as syphilitic serologies (TPHA and VDRL) but she had positive IgG rubella serology.

Faced with this clinical picture with a rapidly progressive AKI, hydro-electrolyte disorders, and respiratory distress, in a context of life-threatening overload, a hemodialysis session was performed through a temporary right femoral catheter removed 48 hours later.

In the context of treatment with ovulation inducers, twin pregnancy, hyperthyroidism, non-obstructive acute renal failure, absence of glomerular damage, absence of tubulo-interstitial damage and multiorgan failure, diagnosis of hyperstimulation syndrome ovarian (SHO) was retained.

Despite adequate symptomatic management (restoration of blood volume, prevention of thromboembolic accidents, antiemetics, analgesics and antispasmodics). The evolution of our patient was marked in our service by the appearance of anarchic uterine contractions, threatening the fetus.

She was urgently sent to the obstetrics gynecology department of the same university hospital where she had a spontaneous abortion. Post-abortion, she rapidly improved her renal function with a plasma creatinine of 8mg/l, and normalizes her liver function test and her inflammatory syndrome.

The patient was seen in consultation clinic 2 months later with a good general condition, no pallor and her BP was at 110/60mmHg. Her laboratory investigations assessment showed blood creatinine at 4.9mg/l, blood urea at 0.14g/l, and hemoglobin level at 12.3g/dl.

Normal white blood count at 8460/mm3. A normal liver test without cytolysis or cholestasis (ALAT=35UI/l, ASAT=25UI/l PAL=192UI/l). Disappearance of rhabdomyolysis with a control CPK of 47UI/l and normal inflammatory marker (CRP<6mg/l).

3- DISCUSSION

Ovarian hyperstimulation syndrome (OHS) is a rare complication of medically assisted reproduction in the management of female infertility by ovarian stimulation [4]. 3-8% of in vitro fertilizations are

complicated by OHS, especially if risk factors are not identified before the procedure in these young women. OHS is a very serious iatrogenic complication because it often affects the fetal maternity prognosis.

Physio pathological mechanisms are not clear, but the most involved factor is the increase of the ovaries size after stimulation by the chorionic gonadotrophin hormone (HCG). This phenomenon is immediately followed by the formation of multiple yellow bodies and the release of ovarian hormones in a significant way and several vasoactive substances such as cytokines, endothelial vascular growth factor (VEGF) and nitrogen monoxide (NO). These excessively produced substances lead to an activation of the ovarian aldosterone renin system and increase of capillary permeability [5]. The rapid progressive renal failure that our patient presented by could be caused by volume overload and third spacing effusion. Unlike a clinical case published by Chauvet *et al.*, for a 32 -year -old patient who was admitted for severe high blood pressure complicated by a thrombotic angiopathy associated with catastrophic antiphospholipid syndrome, triggered by an induction of ovulation [6]. Our patient was not hypertensive, and her immunological assessment was negative but she had a history of spontaneous abortion in her first pregnancy. Also Ravel *et al.*, reported a case of acute coronary syndrome after ovarian stimulation [7]. Thromboembolic events are other complications of OHS and all vessels can be affected. After ovarian stimulation, arterial thrombosis occurs a week to 10 days after the injection of HCG in most of the published cases. So, arterial thrombosis is early complication of OHS. On the other hand, the venous attacks are late, they occur in most cases up to 20 weeks after the induction of ovulation. The most concerned organs are the heart, the large vessels, and the brain, so prophylaxis anticoagulation should be considered if the patient has risk factors. The mechanisms of these thrombosis events are not well known so a prophylactic anticoagulant treatment prevented our patient from thromboembolic event.

Early manifestations of OHS after the induction of ovulation at the beginning of pregnancy are vomiting, nausea, and abdominal distension, capillary leak with serous effusion syndrome, hemoconcentration, hypovolemia, hypoproteinemia, oliguria, hyponatremia and acute renal failure [8, 9]. Our patient presented with a typical clinical picture of OHS, so once the diagnosis was made, we did complete etiological investigations and we identified the main risk factors.

Among her risk factors, we found poly cystic ovary syndrome, granulosa cell tumor, FSH receiver mutation (FSH-R), multiple pregnancies, pituitary adenoma, dysthyroidism (hypothyroidism or hyperthyroidism) [10, 11], that was the case with our patient who was carrying a goiter with biological and clinical hyperthyroidism.

Despite appropriate management (maintenance of blood volume, antispasmodic, analgesic, anti-emetics, carbimazole) the evolution of our patient was marked by a spontaneous abortion at 17 weeks of amenorrhea of a twin pregnancy.

Because of serious complication of OHS, we recommend an adequate clinical and paraclinical examination for any young woman who undertake this therapy of ovulation induction for infertility, to be able to identify the factors favoring the occurrence of OHS.

Acute renal failure in this context of OHS can reveal a catastrophic anti-phospholipid syndrome, lupus, and even thrombotic microangiopathy. We suggest through this case, the realization of a nephrological examination and a preliminary renal assessment, before and during all treatment by medically assisted reproduction techniques.

CONCLUSION

We illustrate through this clinical case that ovarian hyperstimulation syndrome (OHS) is a serious and potentially fatal iatrogenic complication, either for the fetus or for the mother herself. Ovarian induction can be complicated by severe OHS, as in our case. It is therefore important to carry out a meticulous examination in search of risk factors for OHS, before submitting all young women to these medically assisted reproduction protocols [15]. Once the risk factors have been identified, multidisciplinary care can be used to properly plan pregnancies in these women and should reduce the risk of maternal-fetal loss [16].

CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

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