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RESEARCH ARTICLE

ULTRASOUND IN THE PREVENTION, DIAGNOSIS AND MANAGEMENT OF OVARIAN HYPERSTIMULATION SYNDROME IN BAMAKO

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Abstract

Ovarian hyperstimulation or ovarian hyperstimulation syndrome (OHSS) is a direct complication of in vitro fertilization with heavy morbidity and, in some cases, even mortality. The physiology remains poorly defined but seems to be moving towards a cascade activation of coagulation through the vascular endothelial growth factor (VEGF). There are patient-specific or pacing-specific risk factors, but this complication can occur in the absence of these factors. Two types can be described: early OHSS linked to the onset of ovulation and late linked to pregnancy. The management is poorly defined and must adapt to the severity of OHSS by taking care of the added complications.The major complication is thrombotic with the particularity of affecting both the venous and arterial territories, which can induce serious complications such as strokes or myocardial infarction. Prevention of this thrombotic risk therefore remains the priority of OHSS management. Since few studies have been carried out on this subject, we have shown here the place of ultrasound in the prevention, diagnosis and treatment of ovarian hyperstimulation syndrome.

Aims:
- Identify the risk factors of OHSS.
- Describe the sonographic signs of OHSS.
- Show the role of the radiologist/sonographer in the management of OHSS.

Subjects And Methods: This was a cross-sectional, descriptive study concerning 350 women collected between March 2018 and March 2022 in different medically assisted procreation centers in Bamako. The study population consisted of consenting women presenting for assisted reproduction assessment in whom risk factors, signs of hyperstimulation and even complications were identified. The risk factors were: high anti-Müllerian hormone (AMH), the young age of the patient and the count of antral follicles (AFC) with more than 14 follicles. The patients had a biological assessment, the ultrasounds were made by endo-cavitary way and by high way with devices of General electric Voluson E8, Vivid 3 and Logic9.

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Results: 350 women were recruited into our. The average age was 26 years with extremes ranging from 18 to 37 years. 60.85% of the patients were between 18 and 25 years old. The risk factors were: high anti-Müllerian hormone (AMH) (7%), the young age of the patient (88%) and the count of antral follicles or AFC>14 follicles (89.8%). The clinical signs of OHSS were abdominal distension (8.5%), abdominal pain (8.5%), nausea and vomiting (2.28%). Ultrasound signs, i.e. ascites, increased ovarian volume, presence of large ovarian follicles were present in 8.57% of patients. The number of punctured follicles varied between 10 and 30. In sum, hyperstimulation was recorded in 30 patients, i.e. 08.57% of the 30 patients who had the OHSS, 12 had a pregnancy.

Conclusion: There are risk factors for OHSS that correspond to patients with good or even excessive ovarian reserves, as in the context of polycystic ovary syndrome. However, OHSS occurs in 33% of cases with no identified risk factor [1]. It is therefore advisable to carefully monitor ovarian stimulation in the context of In Vitro Fertilization with doses adapted to the patients and to remember that the best prevention remains the cancellation of the cycle without triggering or coasting, that is i.e. a cessation of gonadotropins. OHSS is a serious complication of PMA that can be life-threatening, so care and monitoring must be rigorous, adapted to each stage of the disease. The great variability of OHSS makes it difficult to draw up recommendations for clinical practice and it is therefore necessary to adapt the management according to each patient.

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Introduction:-

The incidence of ovarian hyperstimulation is estimated according to studies at between 3 and 6% with a severe form in 0.1 to 3% of cases [2-3]. In patients with risk factors for hyperstimulation, this rate can reach 20% [4]. This iatrogenic complication of medically assisted procreation (PMA) is mainly encountered during in vitro fertilization (IVF) treatment, especially since the generalization of co-treatment with analoguesof GnRH. Ovarian hyperstimulation syndromes (OHSS) have already been reported during spontaneous pregnancies without associated anomaly [5,6], or in the context of hydatidiform mole [7-8], hypothyroidism [9-10], mutation of the follicle stimulating hormone (FSH) receptor [11-12] or pituitary adenoma [13-14].

This complication can expose the patient to severe complications, in particular thromboembolic, which can be life-threatening, with an estimated mortality rate of between one in 45,000 and one in 500,000 [2].

In the context of the constant increase in the practice of assisted reproduction, this rare complication could become frequent. Because few studies have been done in our context on ovarian hyperstimulation, this study details the current state of knowledge on the pathophysiology, the contribution of ultrasound in the prevention, diagnosis and treatment of OHSS.

Physiopathology:

Ovarian hyperstimulation associates a significant hypertrophy of the ovaries and one or more serous effusions. This passage into the third sector is therefore associated with hemoconcentration, oliguria and thromboembolic phenomena. The hormones and molecules involved in this pathology are as follows:

Estradiol:

High estradiol levels are considered an important risk factor for OHSS. Also, estradiol was initially considered as an actor of OHSS or even its triggering factor [15,16]. However, several publications have shown that a high estradiol level is not an essential condition for the occurrence of OHSS. Pellicer et al. [16] reported OHSS after IVF in a patient with a deficiency in 17,20desmolase activity, and who therefore could not have high estradiol levels. OHSS has been reported in pregnancies with normal or even low estradiol levels as in the case of spontaneous

pregnancies.Furthermore, high isolated estradiol is not sufficient to trigger OHSS since it must be associated with a high level of human chorionicgonadotropin (hCG) [17]. Moreover, estradiol has no direct vasoactive effect [18]. Therefore, high estradiol or a rapid rise in estradiol levels are risk factors for OHSS but not the triggering factor [19].

Luteinizing Hormone (LH) OR HCG:
Since hCG has the same beta chain as LH, it binds to the same receptors but with an affinity six to seven times higher and a much longer half-life [20]. These hormones and, in particular, hCG are therefore considered to be triggering factors for OHSS, explaining OHSS in hydatidiform moles. However, as reported by Nastri et al. [19], hCG alone cannot trigger OHSS. In the study by Michaelson-Cohen et al. [21] relating to 27 pregnancies with hCG levels above 150,000, none was accompanied by OHSS. Moreover, hCG has no direct vasoactive properties [22,23]. hCG and LH would therefore have a role in OHSS, but cannot be the only triggering factor in this cascade of events.

Vascular Endothelial Growth Factor (VEGF)
VEGF increases vascular permeability and promotes, in the context of OHSS, a reduction in the osmotic gradient at the origin of extravascular passage [24]. However, this phenomenon of increased vascular permeability is recognized as the essential mechanism of OHSS. In humans, five isoforms of VEGF have been identified. VEGF A, in particular, is produced by the ovary [22]. The two VEGFR-1 and VEGFR-2 receptors are present in the endothelia but also in the ovarian follicles. Wang et al. [25] have shown that vascular permeability, the level of VEGF and of these receptors are increased from the phase of ovarian stimulation by exogenous gonadotropins. And, 48 hours after the injection of hCG, we observe a peak in the expression of VEGF and VEGFR [26], in particular, at the level of the ovarian vessels. Exogenous (triggering injection) or endogenous (pregnancy) hCG stimulates the production of VEGF by luteinized granulosa cells. It is its supraphysiological lifespan compared to LH that makes it an essential player in this activation cascade.

Interleukins
The interleukins IL-2, IL-6, IL-8, IL-10 and IL-18 are found in the follicular fluid and in the ascites fluid of OHSS. These molecules are involved in the inflammatory process during follicular maturation, ovulation, luteinization and embryo implantation [20]. However, these molecules, in particular IL-6, are observed at high levels in the context of OHSS and would play a role in the cascade of events necessary for the appearance of OHSS, in particular on ovarian neovascularization, inflammation and inhibition of albumin production by the liver [27]. Conversely, IL-10 increasing after the onset of OHSS would have an anti-inflammatory and therefore corrective action of OHSS [28].

The Renin-Angiotensin System
A direct correlation between plasma renin activity and OHSS severity has been reported by Navot et al. [29]. However, the ovaries have the capacity to secrete prorenin [30] and renin [31]. Angiotensin II assays are also elevated in ascites and follicular fluid from OHSS patients [31]. However, this mechanism does not seem to play a promoting role on OHSS but would only be the consequence of hypovolemia secondary to vascular permeability. This hypovolemia would be the cause of the activation of the renin-angiotensin system [32].

In conclusion, the pathophysiology of OHSS remains uncertain. However, it seems to emerge from the literature that VEGF and its VEGFR receptor would be the main players without its activation and regulation being clearly established. hCG seems to play an indispensable role in this cascade of events but cannot be the only initiator. Interleukins, in particular IL-6, should also have a role in this cascade. It would therefore be a set of mediators that must meet several conditions to initiate OHSS, which would explain why all ovarian stimulation, and that not all pregnant patients do OHSS despite high hCG levels.

Exploration Techniques:
Ultrasounds were performed by endocavitary and suprapubic route with General Electric Voluson E8, Vivid 3 and Logic9 devices, all equipped with Doppler.

Data Processing And Analysis:
The data collected on the technical sheets were entered and analyzed using SPSS software. Spearman's and Pearson's correlation tests were used to determine the degree of significance during comparisons at the 5% level.

Results:-

350 women were recruited into our. The average age was 26 years with extremes ranging from 18 to 37 years. 60.85% of the patients were between 18 and 25 years old. The risk factors were: high anti-Müllerian hormone (AMH) (8%), the young age of the patient (88%) and the count of antral follicles or CFA>14 follicles (94.84%). The clinical signs of OHSS were present in 30 women, i.e. 8.57%. Ultrasound signs, i.e. ascites, increased ovarian volume, the presence of large ovarian follicles were found in the same patients. The number of punctured follicles varied between 10 and 30. In sum, hyperstimulation was recorded in 30 patients, i.e. 08.57% of the 30 patients who had the OHSS, 12 had a pregnancy, including one case of thromboembolic complication.

Table I:- Distribution Of Patients According To Age.

AGE (YEARS)	NUMBER	PERCENTAGE (%)
18-25 YRS	**213**	**60.85**
25-35 YRS	95	27.14
35 YRSAND MORE	42	12.00
TOTAL=	**350**	**100%**

The most represented age group was 18-25 years old with nearly 61%. It should be noted that all the patients who underwent hyperstimulation were of this age group.The relationship between the young age of the patient and the occurrence of OHSS was significant ($p<0.005$).

Table II:- Distribution Of Patients According To Anti Müllerian Hormone (Amh) Values.

AMH	NUMBER	PERCENTAGE (%)
0.2-1ng/ml	11	03.14
1-3 ng/ml	**311**	**88.85**
>3ng/ml	28	08.00
TOTAL=	**350**	**100%**

AMH was normal in 88.85% of patients. It is the marker of ovarian reserve. It was high in 28 patients or 08%. No link was found between AMH level and OHSS (P=1).

Table Iii:- Distribution Of Patients According To The Number Of Antral Follicles On The Initial Ultrasound.

NUMBER OF FOLLICLES	NUMBER	PERCENTAGE (%)
≤10	4	01.14
10-14	14	04.00
15-20	26	07.42
>20	**306**	**87.42**
TOTAL=	**350**	**100%**

87.42% had a number of follicles greater than 20. Most of them were in the 18-25 age group and all cases of hyperstimulation were represented there. The correlation between the number of antral follicles at the initial ultrasound and the risk of hyperstimulation was significant ($p=0.001$).

Table IV:- Distribution Of Patients With Hyperstimulation According To Ultrasound Signs.

ULTRASOUND SIGNS	NUMBER	PERCENTAGE (%)
ASCITE AND MORE THAN 5 LARGE FOLLICLES>3 cm	**29**	**96.66**
PLEURESY	1	03.33
TOTAL=	**30**	**100%**

The most represented ultrasound signs were ascites associated with at least five large follicles of more than 3 cm with 96.66%. Serous effusions are constant signs that are almost always present.

The contribution of ultrasound in the treatment was identification during echo-guided punctures, measurements of the quantity of serous effusions, estimating their regression and the puncture of very large follicles.

Discussion:-

Risk factors for ovarian hyperstimulation syndrome (OHSS):

1. Age Of Patients:
61% of our respondents were between 18 and 25 years old and a cumulative percentage of 93% were under 35 years old. Young age is subject to a large number of follicles which would increase the risk of OHSS. Forman et al. (1990) [33], SART (1992) [34] reported that the young age of the patient undergoing medically assisted procreation increases the risk of OHSS by 22% and 25% respectively. The younger the woman, the higher the risk of OHSS ($p<0.005$).

2. The Amh:
Anti-Müllerian hormone was elevated in 8% of cases. Although we did not find a correlation between this increase and the occurrence of OHSS, it was elevated in 93% of those who had this syndrome. LaMarca et al. [35] reported a higher cancellation rate for OHSS risk for an AMH level above 7 ng/ml. However, this rate suggests a more specific condition, that of polycystic ovary syndrome (PCOS) [36].

3. The Number Of Follicles Greater Than 14 Follicles At The Initial Ultrasound:
87.42% had a number of follicles greater than or equal to 20 follicles at the initial ultrasound. The antral follicle count on the initial ultrasound has the same predictive value as the AMH. The threshold retained in the review of the literature is 14 follicles with a sensitivity of 82%. Our figure is very close to the recent meta analysis by Broer et al. that polycystic ovary syndrome is responsible for hyperstimulation in 89% [37].

Ultrasound signs:
The most represented were ascites associated with at least five large follicles of more than 3 cm with 96.66%. Serous effusions are constant signs that are almost always present. Our figures are confirmed by studies by Delvigne A. et al. [38] and Kesler R. et al. [39] who respectively found 95.6% and 96.4% serous effusion in severe OHSS.

The evacuating puncture of ascites makes it possible to give patients comfort because ascites creates discomfort, heaviness, nausea/vomiting and pleurisy creates breathing difficulty [40].

Conclusion:-
There are risk factors for OHSS that correspond to patients with good or even excessive ovarian reserves, as in the context of polycystic ovary syndrome. However, OHSS occurs in 33% of cases with no identified risk factor [1]. It is therefore advisable to carefully monitor ovarian stimulation in the context of PMA with doses adapted to the patients and to remember that the best prevention remains the cancellation of the cycle without triggering or coasting, i.e. say a discontinuation of gonadotropins to allow a drop in estradiol levels. OHSS is a serious complication of PMA that can be life-threatening, so care and monitoring must be rigorous, adapted to each stage of the disease. The great variability of OHSS makes it difficult to draw up recommendations for clinical practice and it is therefore necessary to adapt the management according to each patient.

Declaration of conflict of interest:
All authors declared having no conflict of interest.

Iconography:

FIG A: Poly micro follicular ovaries

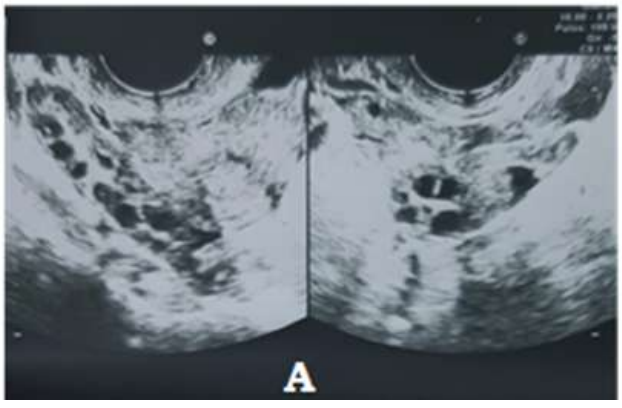


FIG B: polycystic ovaries with peri-ovarian fluid effusion

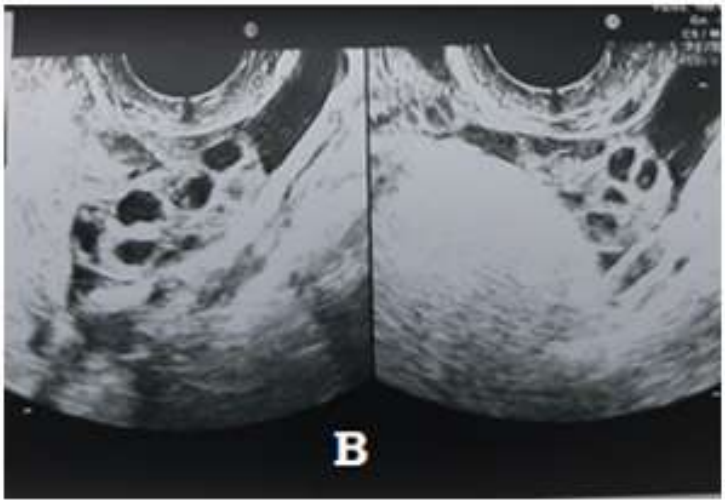


FIG E: Large follicles with high risk of ovarian hyper stimulation syndrome

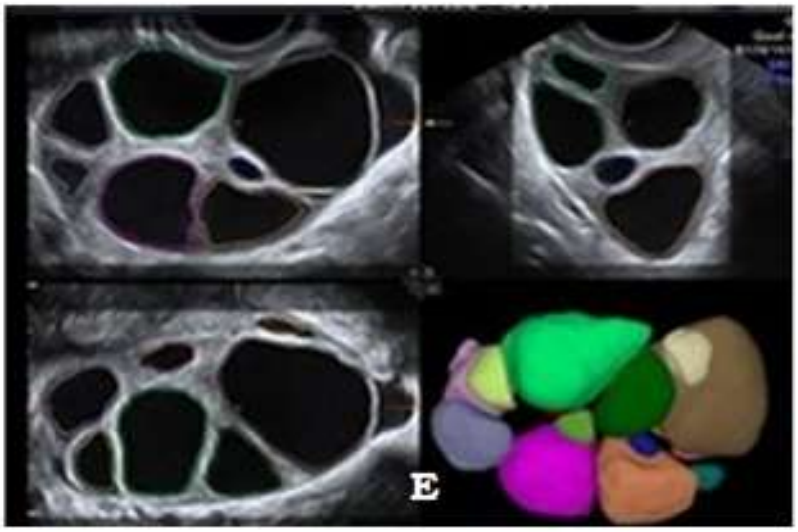


FIG C: Ascite between the liver and right kidney

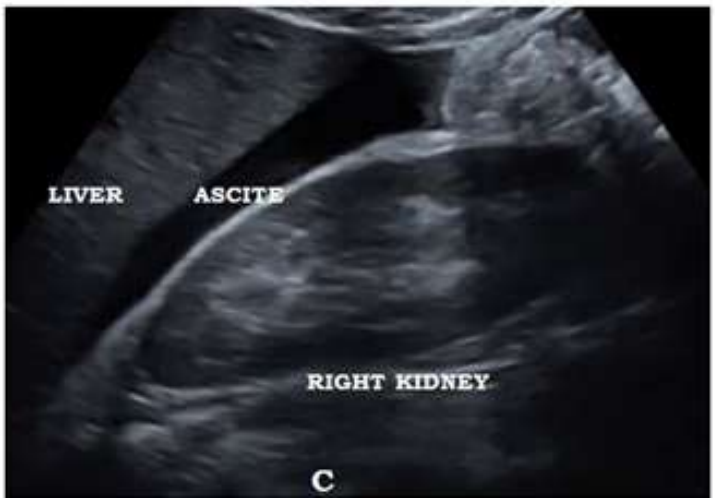
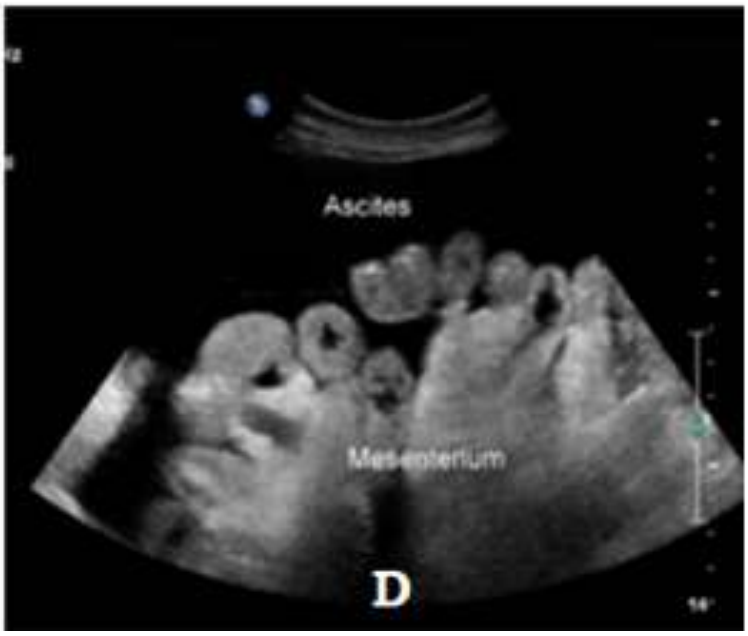


FIG D: Ascites of great abundance between the handles



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