Abstract
To assess the possibility of monitoring ovulation induction in an out-patient basis in hypogonadotrophic amenorrhoea depending solely on abdominal ultrasound monitoring only.

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Introduction
For the majority of pubertal girls menstruation is the final results of a series of events, which results in sexual maturity. Maturation of the hypothalamus through several years of late childhood begins a cascade of events, which finally result in the establishment of the normal menstrual cycle and menstruation. Amenorrhoea will result when there is a failure of function in any of the organs involved in this cascade [1].

During childhood, gonadotrophin levels are low, both terms of pulse frequency and pulse amplitude but finally the establishment of a mature hypothalamus and gonadotrophin releasing hormone (GnRH) release leads to normal endocrine environment [2].

This results in ovarian stimulation, the production of estradiol from the follicles, which begin to develop at around the age of 8.5 years [3].

This rise in estrogen leads to secondary sexual development of the breasts and the establishment of menstruation.

Amenorrhoea is defined as the absence of menstruation. It may be classified as either primary or secondary amenorrhoea. There are, of course, physiological situation where amenorrhoea is normal, namely pregnancy, lactation and prior to the onset of puberty. Primary amenorrhoea, this condition relates to female who fail to menstruate by 16 years of age. Secondary amenorrhoea is defined as the cessation of menstruation for more than six months in a normal female of reproductive age that is not due to pregnancy.
There are various causes for hypogonadotrophic amenorrhea. This include pituitary insufficiency in which the pituitary fail to secrete gonadotrophins. In female, the most common cause is a result of severe obstetric hemorrhage causing necrosis of the pituitary (Sheehan's Syndrome).

Hypothalamus causes of hypogonadotrophic amenorrhea divided into functional and nonfunctional. The nonfunctional include space occupying lesions in which the hypothalamus partially or completely destroyed by lesions [4].

It is essential to assess the pituitary function fully in all patients with hypogonadotrophic hypogonadism and then instigate the appropriate replacement therapy. Ovulation may be induced with pulsatile subcutaneous GnRH or gonadotrophins. The administration of pulsatile GnRH provides the most physiological correction of infertility caused by hypogonadotrophic hypogonadism and will result in uni-follicular ovulation, whilst FSH therapy requires close monitoring to prevent multiple pregnancy. Purified or recombinant FSH preparations are not suitable for women with hypogonadotrophic hypogonadism (or pituitary hypogonadism) as these patients have absent endogenous production of LH and so whilst follicular growth may occur, oestrogen biosynthesis is impaired [5].

Thus, human menopausal gonadotrophins, which contain FSH and LH activity, are necessary for these patients.

Gonadotrophin therapy requires only an ovary with response oocytes, some 90% of patients will ovulate with it, and (50 – 70)% will conceive. Multiple gestations is a major complication of the method, with an incidence of (20 - 35) %. three or more fetuses per pregnancy will occur in 5% of women. Super ovulation can be kept to a minimum by withholding HCG if ultrasound shows more than from follicle or estrogen level exceeds 1500 pg/ml.

The second major drawback of gonadotrophin treatment is the hyperstimulation syndrome. Ovarian enlargement of 5 – 10 cm occurs in 30% of ovulating cycles, this can be reduced by withholdings HCG if signs of hyperstimulate by ultrasound or estrogen level above 1500pg/ml [6].

HMG is a hoemonal substance containing FSH and LH in the ratio of 1:1. In female, pergonal stimulate both the growth and the maturation of follicles and induces the secretion of estrogen and endometrial proliferation. The dose of FSH should be tolerated to the patient’s response. Patients should start treatment with two to four ampules of FSH daily.

When pelvic ultrasound confirms an increase in endometrial thickness and the presence of follicles of more than 14 mm in diameter, the serum oestradiol concentration should be measured and the same dose maintained. Patients should be monitored using ultrasound every 1 -2 days. Treatment should be adjusted until there is evidence of follicular growth and an increase in endometrial thickness.

When the largest follicle has a mean diameter of 18 mm, there are at least two other follicles of more than 14 mm, and the endometrial thickness of 8 mm or more 10000 IU HCG should be administrated, so ultrasonography is mandatory for the monitoring of ovarian stimulation.

**Material and Method**

Twenty hypogonadotrophic amenorrhoeic women from the history, examination and investigation they have no other abnormality except the low FSH and LH been married and infertile for more than two years to normal male with normal semen analysis .Subjected to ovulation induction for 42 treatment cycles by injectable gonadotrophins .Each cycle starts by human menopausal gonadotrophin( 75 IU ampule) two
ampules for the first two days and then one ampule for the remaining days depending on the patient response changing the dosage monitoring by abdominal real ultrasound every two days when the largest follicle has a mean diameter of 18 mm or more 10000 IU HCG given. Then follow the patient for the next cycles in the same way.

Diagnosis of pregnancy after one miss period by pregnancy test and ultrasound confirmation, then follow the pregnancy for each.

**Results**

Out of 42 treatment cycles in 20 patients, nine pregnancies resulted (45%), five aborted and four ended by a live babies (as in table 1). Mild ovarian hyperstimulation occurred in one patient. The incidence (2.5%) per treatment cycle (as in table 3) and (5%) per patient (as in table 2).

**Table 1**

<table>
<thead>
<tr>
<th>No. of Pat.</th>
<th>No. of Treat. cycle</th>
<th>Outcome ( Preg.)</th>
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<tr>
<td>20</td>
<td>42</td>
<td>9 (45%)</td>
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<td>Abortion</td>
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<td>5 (55.5%)</td>
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<td>Full term</td>
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<td>4 (44.4%)</td>
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**Table 2**

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<th>No. of pat.</th>
<th>OHSS</th>
<th>Percentage ( % )</th>
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<td>20</td>
<td>One</td>
<td>5 %</td>
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**Table 3**

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<th>No. of Treat. cycle</th>
<th>OHSS</th>
<th>Percentage ( % )</th>
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</thead>
<tbody>
<tr>
<td>42</td>
<td>One</td>
<td>2.5 %</td>
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**Conclusions**

We can depend solely on ultrasound for monitoring ovulation induction in hypogonadotrophic amenorrhoeic especially when facilities for serial Estradiol E<sub>2</sub> estimation are not available or different to perform. Monitoring has to be conducted by an experienced ultrasound trained doctors. Obstetrician should be careful for monitoring signs of ovarian hyperstimulation clinically and by ultrasound which are the main indication of Estradiol measurement.

**References**


2- Lee PA, Piotrick LP, Migeon CJ et al., 1978.


