

Female Hypogonadotropic Hypogonadism: About Two Cases

Niass Aminata*, Ndiaye Mame Diarra, Sall Ndeye Racky, Ndaw Aida, Philippe Moreira

Service de Médecine de la Reproduction Centre Hospitalier National Dalal Jamm, Dakar, Senegal

Email: *bguisse@orange.sn

How to cite this paper: Aminata, N., Diarra, N.M., Racky, S.N., Aida, N. and Moreira, P. (2025) Female Hypogonadotropic Hypogonadism: About Two Cases. *Open Journal of Obstetrics and Gynecology*, 15, 1030-1035.

<https://doi.org/10.4236/ojog.2025.157084>

Received: May 6, 2025

Accepted: July 8, 2025

Published: July 11, 2025

Copyright © 2025 by author(s) and Scientific Research Publishing Inc. This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

Abstract

Methods: This was a retrospective study in the Reproductive Medicine Department of the Dalal Jamm National Hospital. We collected two cases for which we collected sociodemographic data, circumstances of discovery, results of additional examinations that confirmed the diagnosis, as well as therapeutic and evolutionary data. **Results:** Delay in diagnosis was the rule, with an average age at diagnosis of 22.5 years. The main reason for consultation was the desire to become pregnant. The management of infertility is based on stimulations with gonadotropins, stimulations that have allowed the recruitment of follicles in both. However, the great particularity of the stimulation was the use of high doses of gonadotropins (response obtained only with 150IU of FSHr + LHr) which, in terms of cost, constituted an obstacle to the continuation of the treatment. **Conclusion:** Female hypogonadism remains a rare pathology and its late diagnosis in our practice is linked to a delay in the first consultation. Amenorrhea is the most common reason for consultation.

Keywords

Hypogonadism, Hypogonadotropic, Hypergonadotropic, Puberty, Amenorrhea, Infertility, Gonadotropins

1. Introduction

Female hypogonadism refers to any clinical-biological situation of deficiency of estrogen secretion by the ovaries [1]. When the deficit affects the hypothalamic-pituitary axis, it is called central hypogonadism or hypogonadotropic. The clinical picture is different depending on the time of occurrence (congenital or acquired), the location of the involvement (primary or secondary) and the nature of the abnormality (functional or lesional). Delayed puberty is the most common mode of disclosure. Acquired hypogonadotropic hypogonadisms are the most common

[2], most often due to pituitary adenomas, in particular prolactinomas, not to mention other etiologies such as infiltrative processes or overload diseases. Functional acquired hypogonadotropic hypogonadisms are most often the consequence of a nutritional deficit [3]. In Africa, particularly in Senegal, data are still scarce. Only a few sporadic cases have been reported [3]. In Senegal, a study conducted in 2016 on 10 patient observations collected over a period of 2 years found 7 cases of male hypogonadism and 3 cases of female hypogonadism [4]. Another study [1] on female hypogonadisms for a period of 9 years had found 11 cases. The aim of our work was to illustrate the diagnostic and therapeutic difficulties encountered in the management of female hypogonadisms in our practice.

2. Methodology

This is a retrospective case study over a 5-year period from January 2018 to December 2024. Two cases were collected. The analysis of the different circumstances of discovery of hypogonadism, the clinical characteristics of each patient, biological data and imaging as well as the therapeutic management and the outcome of the patients were carried out.

2.1. Clinical Observation No. 1

25-year-old patient, married for 2 years under a polygamous regime, nulligest with no particular history having consulted for a desire to become pregnant. As functional signs, she presented with a secondary amenorrhea of 9 months. The spouse is 45 years old, teacher, father of 4 youngest children aged 6 months. There is no notion of smoking found. The examination had found a weight of 70 kg, a height of 172cm, a normal corporeal mass index (23.66 kg/m^2), a female morphotype, normal external genitalia, secondary sexual characteristics were present and normal (Tanner stage 5). Hormonal assays performed returned with estradiolemia at 68.4 pg/mL, collapsed gonadotropin levels with FSH at 0.3 mIU/mL and collapsed LH at 0.31 mIU/mL. TSHus: 3.670 uIU/mL, FT4: 11.22 pg/mL, prolactinemia: 14.83 mIU/mL, testosterone: 0.29 mIU/mL. The cerebro-pituitary MRI had objectified a cystic dilation of the Virchow's spaces with regard to the posterior horns of the lateral ventricles and an absence of pituitary adenoma Pelvic ultrasound (done at the beginning of the follicular phase) had made it possible to visualize a gynecological-sized uterus with multi-follicular ovaries. The hysterosalpingography and the semen analysis of the spouse performed as part of the overall management of infertility were normal. The patient had been put on gonadotropins (fSHr + LHr) with an initial dose of 75 IU without follicular recruitment on day 8 of the cycle, increased to 112.5 IU. On day 12 of the cycle, two dominant follicles of 17 and 20 mm were observed. An induction by HGC was done then programming of sexual intercourse then support of the luteal phase with natural progesterone 200 mg per 8 hours combined with dydrogesterone 10 mg per 12 hours. After fifteen days of progesterone supplementation, the plasma BHCG assay came back negative. The patient reviewed her period around day 30

of the menstrual cycle. Another cycle of stimulation with intrauterine insemination had been proposed but the husband refused, the patient has been lost to follow-up.

2.2. Clinical Observation No. 2

A 20-year-old patient who had been married for 13 months on a monogamous diet, with no particular pathological history, was seen for primary amenorrhea and desire for pregnancy. The spouse is 28 years old, mechanic, without children. The examination found a weight of 63kg, a height of 166cm, a body mass index of 22.86 kg/m². The morphotype was feminine with the secondary sexual characteristics not very marked (Tanner 3).

Hormonal assays revealed an estradiol level of 68.4 pg/mL, FSH collapsed to 0.1 mIU/mL, LH collapsed to 0.1 mIU/mL, TSHus: 3.670 uIU/mL, prolactinemia: 13 mIU/mL. Cerebropituitary MRI: normal. Pelvic ultrasound (done at the beginning of the follicular phase): uterine hypotrophy, normofollicular ovaries, normal hysterosalpingogram as well as the spermiogram. Intrauterine insemination was proposed but the couple decided to try spontaneous intercourse stimulation first.

The patient had been put on the pill for 3 months and then on gonadotropins (fSHr + LHr) with an initial dose of 75 IU without follicular recruitment on day 8 of the cycle, increased to 112.5 IU without recruitment and then to 150IU which allowed to have a dominant follicle on day 14 of the 19 mm cycle. An induction by HGC was done then programming of sexual intercourse and then support of the luteal phase with natural progesterone 200 mg per 8 hours combined with dydrogesterone 10 mg per 12 hours. Plasma BHCG was negative. On day 33 of the cycle, she reviewed her periods, but due to a problem of means, a therapeutic interruption was noted. The patient has been on the combined pill for 6 months waiting for ways to start a stimulation cycle with intrauterine insemination.

3. Discussion

- Epidemio-clinical, diagnostic and therapeutic aspects

Hypogonadism is a relatively rare condition that is underdiagnosed, with an unknown combined prevalence in men and women worldwide. In addition, data is scarce in Africa. The most common presentation is delayed or absent puberty according to Etoa Etoga MC *et al.* concerning a cross-sectional study over a period of 12 months, in 3 reference hospitals in Cameroon including men and women diagnosed with hypogonadism [3]. Delayed puberty is a common cause of consultation with a pediatric endocrinologist. The prevalence of delayed puberty is estimated to be around 3% [5]-[7]. Primary gonadal insufficiency or hypergonadotropic hypogonadism is thought to account for about 26% of PR in girls [8] [9]. During our study, which lasted 6 years, we were able to identify only 2 cases. The average age of our series is 22.5 years. Hypogonadism is diagnosed late in Africa, where some of our authors [3] [10] have pointed out that early diagnosis requires vigilance on the part of parents. Isselmou EBO points out that in our conservative

societies, there is a considerable delay in the first consultation [11]. In our study, the patients were 20 and 25 years old. This age interval corresponds to the post-pubertal period when the absence of secondary sexual characteristics begins to worry the patient. Amenorrhea is the main cause of the discovery of hypogonadism in our series. It is found in 100 of the cases. Amenorrhea is one of the main reasons for consultation in endocrinology and gynecology. In pathology, the existence of amenorrhea indicates damage to the hypothalamic-pituitary-ovarian axis or an abnormality of the reproductive tract [1]. According to Young J *et al.*, following a multicenter survey of a series of 104 patients, congenital hypogonadotropic hypogonadism is revealed by primary amenorrhea in more than 90% of cases in women [12]. As a first-line treatment, plasma assays of sex steroids, coupled with those of FSH and baseline LH, are the essential initial step in diagnosis [8]. An additional hormonal assessment with prolactin assays and the various actors of the corticotropic, thyrotropic and somatotropic axes is then necessary in order to direct the patient towards an endocrine pathology [12]. Hypogonadism can be isolated or combined with other anterior pituitary deficits in the context of developmental abnormalities of the anterior pituitary gland [9]. In our patients, the laboratory work-up is composed of plasma assays of gonadotropins LH, FSH and oestradiol. Other check-ups are only requested according to the clinical cases. Pelvic ultrasound is the first-line examination to be performed in the diagnosis of hypogonadism [13]. It is essential for girls and complements the clinical examination [13]. Abdominal pelvic ultrasound can be used to measure, with criteria of size and morphology, the degree of estrogen impregnation of the internal genitalia [9] [14]. MRI is the examination of choice in the study of the brain [9]. If a central pathology is suggested, a hypothalamic-pituitary MRI with measurement of the pituitary height, the pituitary shaft and images centered on the olfactory bulbs is performed to look for a congenital malformation of the hypothalamic-pituitary axis, agenesis of the olfactory bulbs or a tumor or infiltrative cause [11]. As a result, MRI was requested from all our patients. The correction of hypogonadism, based on indisputable biological results, will have multiple beneficial effects. The objectives of treatment are to induce a satisfactory peak of pubertal growth, the development of secondary sexual characteristics, then normal adult sexual activity and, if possible, fertility [6]. The standard treatment remains estrogen-progestin supplementation [6] [15].

- Fertility and hypogonadism

Hormonal disorders that can induce female infertility are not uncommon in our environment [5]. In women, when pregnancy is desired, ovulation will only be induced after several months of estrogen-progestin treatment to ensure proper uterine development [16]. Gonadotropic deficits in women are in the majority of cases of hypothalamic origin. Ovulation inductions can therefore be achieved through pituitary stimulation by pulsatile administration of GnRH but also by direct ovarian stimulation through the administration of gonadotropins. In the case of pituitary involvement, pulsatile GnRH is inoperative and only gonadotropin

treatment will be effective, as for example in severe mutations of the GnRH receptor. In case of failure of pulsatile administration of GnRH, when this treatment is not available, gonadotropins can be used, as is the case in our two patients. According to retrospective studies, the ovulation rate per cycle is estimated between 70% and 85%, the pregnancy rate per ovulatory cycle between 18% and 45%, and the cumulative pregnancy rate for six cycles between 70% and 100% [1]. These results concern stimulation by GnRH pump. The alternatives to be offered are egg donation, embryo reception, adoption and learning to “live happily together”. The ESHRE (European Society of Human Reproduction and Embryology) report from October 2015 analyzed results from 2011 regarding ART in Europe. This report publishes results obtained from various IVF and ICSI protocols in Europe each year. It covers 33 countries, 1314 clinics, and 609,973 treatment cycles, including 138,592 IVF and 298,918 ICSI procedures. There were 134,106 births recorded, of which 102,160 followed ICSI and 21,156 were embryo transfers [17]. These reports have shown us that it often takes a certain number of attempts before achieving a pregnancy, especially regarding ART management in general that is to say, not only in the case of women with HH. The difficulty is felt more in our context where assisted reproductive techniques are not accessible to everyone (no coverage by social security, the GnRH pump not available, the price of gonadotropins is expensive (about 400 dollars per unit), and the in vitro fertilization cycle can cost up to 5000 dollars).

4. Conclusion

Hypogonadotropic hypogonadism, or hypothalamic-pituitary hypogonadism, remains a rare pathology. The low oestradiol dosage can confirm female hypogonadism. And the determination of pituitary gonadostimulins (FSH and LH) makes it possible to differentiate a central origin (low gonadotropins defining a hypogonadotropic hypogonadism) from a gonadal origin (high gonadotropins, which defines a hypergonadotropic hypogonadism). In addition to managing the cause, if possible, treatment is based on hormone replacement therapy. Medical procreation will be considered in some cases.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Lèye, A., Ndiaye Sarr, N. and Lèye, Y.M. (2019) Hypogonadismes Féminins: Signes-Diagnostic-Traitement. In: *Pathologies Endocriniennes*, 1ère Edition, 181-183.
- [2] Brauner (2013) Puberté Pathologique. In: *EMC Pédiatrie*, Vol. 4, Elsevier Masson, 107-B-15.
- [3] Isselmou, E.B.O., Leye, A. and Ould Moctar, A. (2012) Hypogonadisme hypogonadotrope congénital associé à une obésité: À propos d'un cas. *Diabetes & Metabolism*, **38**, 63.
- [4] Alli, I.O.A. (2016) Aspects diagnostiques et thérapeutiques des hypogonadismes au

- Centre National Hospitalier de Pikine (CNHP). Thèse, Université Cheikh Anta Diop de Dakar, 123 p.
- [5] Ould Isselmou, E.B. and Leye, A. (2012) P134 Syndrome de turner associé à une hypothyroïdie: À propos d'un cas. *Diabetes & Metabolism*, **38**, A63.
[https://doi.org/10.1016/s1262-3636\(12\)71236-3](https://doi.org/10.1016/s1262-3636(12)71236-3)
 - [6] Ben Souda, M., Amziane, F. and El Ouahabi, H. (2017) L'hypogonadisme hypogonadotrope féminin. *Annales d'Endocrinologie*, **78**, 306-307.
<https://doi.org/10.1016/j.ando.2017.07.284>
 - [7] Duranteau, L. (2019) Retard pubertaire chez la fille. In: Letombe, B., Jonard, S. and Robin, G., Eds., *Endocrinologie en Gynécologie et Obstétrique*, Elsevier, 41-55.
<https://doi.org/10.1016/b978-2-294-75965-9.00004-0>
 - [8] Bouhours-Nouet, N., Donzeau, A., Coutant, R., Illouz, F. and Rodien, P. (2015) Conduite à tenir devant un retard de croissance staturale à l'adolescence. *MCED Janvier-Février*, **74**, 61-69.
 - [9] Cabrol, S. (2007) Le syndrome de Turner. *Annales d'Endocrinologie*, **68**, 2-9.
 - [10] Iraqi, N., Boufares, F. and Belmejdoub, G. (2013) Hypogonadisme Hypogonadotrope. *Maroc Médical*, **32**, No. 3.
<https://doi.org/10.48408/IMIST.PRSM/mm-v32i3.1241>
 - [11] Garel, C. and Léger, J. (2006) Contribution of Magnetic Resonance Imaging in Non-Tumoral Hypopituitarism in Children. *Hormone Research in Paediatrics*, **67**, 194-202. <https://doi.org/10.1159/000097755>
 - [12] Issoufou, D.M., Altine, H., Sani, M.M., Soli, I., Ado, A.I. and Issoufou, M. (2019) Profil hormonal en cas d'infertilité chez la femme à l'Institut des radioisotopes de niamey. *Health Sciences and Disease*, **21**, No. 1.
 - [13] Edouard, T. and Tauber, M. (2010) Retard Pubertaire. *Archives de Pédiatrie*, **17**, 195-200. <https://doi.org/10.1016/j.arcped.2009.09.017>
 - [14] Christin-Maitre, S., Pasquier, M., Donadille, B. and Bouchard, P. (2006) L'insuffisance ovarienne prématurée. *Annales d'Endocrinologie*, **67**, 557-566.
[https://doi.org/10.1016/s0003-4266\(06\)73007-4](https://doi.org/10.1016/s0003-4266(06)73007-4)
 - [15] Roze, C., Touraine, P., Leger, J. and de Roux, N. (2009) Hypogonadisme Hypogonadotrope Congénital. *Annales d'Endocrinologie*, **70**, 2-13.
<https://doi.org/10.1016/j.ando.2008.06.005>
 - [16] Young, J. (2007) Hypogonadisme hypogonadotrophique congénital: Procréation, grossesses et descendance. In: Caron, P., Ed., *Pathologie hypophysaire et grossesse*, Springer, 147-159. https://doi.org/10.1007/978-2-287-35572-1_11
 - [17] Kupka, M.S., D'Hooghe, T., Ferraretti, A.P., de Mouzon, J., Erb, K., Castilla, J.A., et al. (2016) Assisted Reproductive Technology in Europe, 2011: Results Generated from European Registers by ESHRE. *Human Reproduction*, **31**, 233-248.