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Profile of Female Hyperandrogenism at the Medical Clinic II: About 19 Cases

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Abstract

Introduction: Female hyperandrogenism is essentially manifested by hirsutism. It is present in Senegal but not yet elucidated for lack of data. This is why we proposed to study the profile of hyperandrogenism in our context. **Patients and Methods:** This was an observational, cross-sectional cohort study of 19 patients of reproductive age followed for hyperandrogenism at the Medical Clinic II of Abass Ndao Hospital, from November 1st, 2019 to August 31st, 2021. **Results:** Our cohort consisted of women with an average age of 25.3 years, single (73.7%), with a low socioeconomic level (42.1%). A family history of hirsutism was found in 31.6% of cases. The main reasons for consultation were a menstrual cycle disorder in 94.7% of cases, and hirsutism in 78.9% of cases. The latter was post-pubertal (66.7%), with a slow or progressive evolution. The physical examination revealed: hirsutism (100%) with an average modified Ferriman Gallwey score (mFG) of 8.9 ± 5.8, acne (36.8%), hyperseborrhea (57.9%), major signs of virilization (10.5%), acanthosis nigricans (47.4%) and galactorrhea (5.3%). Hormonal explorations revealed an elevation of: testosterone (31.6%), 17-hydroxyprogesterone (5.3%), dihydrotestosterone (31.6%), and prolactin (10.5%). The ovarian morphology was micropolycystic (84.2%). The etiological profile corresponded to polycystic ovarian syndrome or PCOS (68.4%), ovarian hyperthecosis (10.5%), hyperprolactinemia (10.5%), congenital adrenal hyperplasia or CAH (5.3%). Idiopathic hirsutism was found in 5.3% of cases. **Conclusion:** Female hyperandrogenism is a less frequent reason for consultation in endocrinology. It deserves to be further evaluated in a large-scale study focused on epidemiological, clinico-biological and etiological investigation, in order to assess its prevalence and better define its profile in our context.

Keywords

Female Hyperandrogenism, Hirsutism, Senegal

1. Introduction

Hyperandrogenism is defined as an excessive secretion of androgens responsible for masculinization in women [1]. It is thought to be one of the most common endocrine abnormalities. Epidemiological studies carried out in the United States and Spain reported that 6% to 8% of women of childbearing age are hirsute [2] [3]. In a North American study, the prevalence was estimated at 5% in light phototypes and 8% in dark phototypes [2]. Its prevalence seems lower (4%) among Asians, according to diagnostic criteria re-evaluated in a study carried out on a population of 10,000 Chinese women [4]. Other studies reported a higher prevalence among Mediterranean (38% in Greece) or Australian (21%) women [5]. In sub-Saharan Africa, particularly in Senegal, we do not yet have a study to assess the exact prevalence of this phenomenon.

From a paraclinical point of view, the key element of the diagnosis is the demonstration of biological hyperandrogenism. This is usually based on the determination of plasma testosterone and/or its active metabolite, dihydrotestosterone [6] [7].

Polymicrocystic ovary syndrome (PCOS) is by far the most frequent etiology, observed in 71% of cases [8].

In addition to specific treatments depending on the etiology, anti-androgens are the cornerstone of the treatment of hyperandrogenism, especially hirsutism. Therefore, cyproterone acetate (a potent synthetic progestin with anti-androgenic activity) was the most widely used molecule in this context, which has now been replaced by estrogen-progestin contraception (EPC). It is now indicated for severe hyperandrogenism [9].

In view of these realities, it seemed necessary to us to contribute to a better knowledge of hyperandrogenism in our context.

2. Patients and Methods

This is an observational and cross-sectional study of cohort type, conducted over a period of 22 months, from November 1st, 2019 to August 31st, 2021. It concerned all patients of childbearing age seen in consultation at the medical clinic II of the Abass Ndao hospital center with signs of clinical and/or biological hyperandrogenism. Patients for whom the clinical and paraclinical data were not complete were excluded, as well as those who presented outside the inclusion period. For each patient, a file was opened. The clinical investigations were generally done at the first consultation, which allowed the following parameters to be collected: civil status, reasons for consultation, history of the disease, personal and family pathological history.

The general examination consisted mainly of the measurement and evaluation of body weight, height, BMI, waist circumference, constants.

Examination of the systems for manifestations of hyperandrogenism and others that may point to an etiology, including: the cutaneous-pituitary system (looking for alopecia, acne, hyperseborrhea, acanthosis nigricans and especially

for localizations of hirsutism; the latter was evaluated according to the modified Ferriman and Gallwey score); the gynecological system (looking for micromastia, galactorrhea, virilization of the external genitalia…).

Biological investigations based essentially on the determination of: total plasma testosterone, 17-hydroxyprogesterone (17-OHP), dehydroepiandrosterone sulfate (DHEAS), dihydrotestosterone (DHT) and urinary free cortisol (UFC), in order to confirm the hyperandrogenism In order to refine the etiological research, a morphological assessment mainly concerned pelvic ultrasound and exceptionally pelvic MRI.

A data sheet was established to collect the essential data for our study from each patient. The sample size was calculated by the Schwartz formula. The analysis was performed with the following software: Excel 2010 and Epi info 7.2. Thus, qualitative variables were described by frequency tables, bar charts, and pie charts. Quantitative variables were described by their positional (mean, median and mode) and dispersion (standard deviation, extremes) parameters.

All data provided in this study are anonymous. The work was carried out with respect to confidentiality.

Patients consent was sought for the taking and use of personal images.

3. Results

3.1. Epidemiological Data

The number of patients included in the study was nineteen (19). The mean age of the patients was 25.3 years with a standard deviation of 5.4 years. The extremes were 16 and 38 years. Fourteen patients or 73.7% were single and five patients or 26.3% were married. The socio-economic level was low in 8 patients (42.1%).

3.2. Clinical Data

3.2.1. Reasons for Consultation

The different reasons for consultation noted in our series were: menstrual disorders or menstrual cycle (94.7%), hirsutism (78.9%), pelvic pain (10.5%), external genitalia anomaly (5.3%), and galactorrhea (5.3%).

3.2.2. History and Terrain

These were noted in 57.9% of the patients, obesity in 5 patients, arterial hypertension (AH) in 2 patients. Other conditions included acne, Graves' disease, epilepsy, asthma, spasmophilia and smoking in two patients. Infertility was noted in 80% of the brides (4 of 5 patients). The notion of taking medication was found in 7 patients (36.8%), dominated by the use of Utrogestan (14.3%).

Twelve patients (63.2%) had a family history or background. Familial hirsutism was present in 6 patients (31.6%), polymicrocystic ovary syndrome (PCOS) in 2 and congenital adrenal hyperplasia (CAH) in none of the patients. Other familial conditions included diabetes, hypertension and Graves' disease.

3.2.3. Clinical Manifestations

➢ **Physical examination**

The mean weight of the patients was 69.9 kg with extremes of 51 and 124 kg. The mean height of the patients was 1.63 m and the extremes were 1.43 and 1.76 m. The average BMI of the patients was 25.5 kg/m^2 and the extremes were 19.1 and 48.4 kg/m^2. Ten patients (52.6%) had a normal BMI. Waist circumference was normal in 14 patients (73.7%) and high in 5 patients (26.3%). Blood pressure was normal in 18 patients (94.7%) and high in one patient.

➢ **Hirsutism**

The average modified Ferriman and Gallwey score of the patients was 8.9. Hirsutism was present in all 19 patients. Ten patients or 52.6% had a "non-pathological" score. These patients all shaved. Hirsutism was mild in 6 patients or 31.6% and moderate in 3 patients or 15.8%. Figure 1 shows the hirsutism in one of our patients.

➢ **Minor and major signs of virilization**

Minor signs of virilization were present in 12 patients. Hyperseborrhea associated with acne was present in 6 of the 12 patients (50%). Hyperseborrhea alone was present in 41.7% of patients. Acne was present in 8.3% of patients.

As major signs, one patient had alopecia and clitoral hypertrophy. Another patient had a male morphotype with micromastia, muscular hypertrophy and penile clitoris. Figure 2 shows the clitoromegaly of this patient.

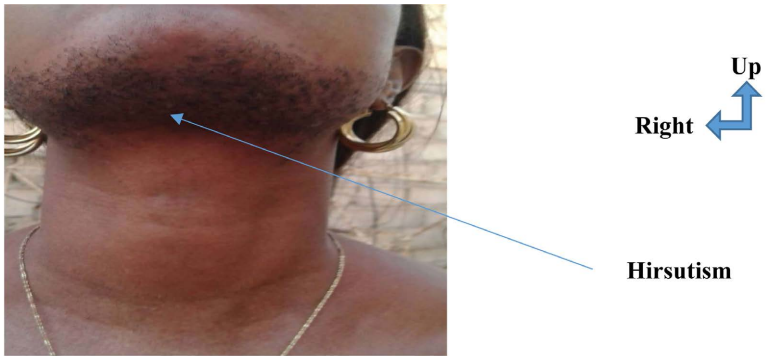

Figure 1. Hirsutism on the chin.

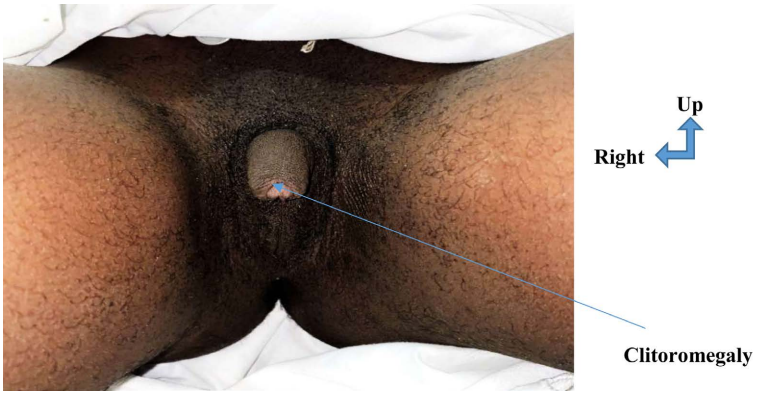

Figure 2. Clitoral hypertrophy in a patient.

➢ **Other clinical manifestations**

Acanthosis nigricans was noted in 9 patients or 47.4%. Bison hump, purple stretch marks and galactorrhea were noted in one patient each.

One patient had undergone type 1 genital mutilation (clitoridectomy).

3.3. Paraclinical Data

Sixteen patients (84.2%) had a total plasma testosterone measurement. Testosterone levels were normal in 10 patients (52.6%) and elevated in 6 patients (31.6%). Table 1 summarizes the results of the different assays performed in our study.

Seventeen patients (89.5%) had undergone pelvic ultrasound. This was normal in one patient. Polymicrocystic ovarian dystrophy was suspected in 16 patients (84.2%). One patient had a pelvic MRI with a normal result.

Polymicrocystic ovarian syndrome (PCOS) was considered in 13 patients (68.4%) and enzyme block due to 21-hydroxylase deficiency (congenital adrenal hyperplasia or CAH) in one patient.

4. Discussion

4.1. Epidemiological Characteristics

Our cohort was composed of 19 patients. This cohort was considered small

Table 1. Distribution of tests according to their number, average and results (N = 19).

TESTS	NUMBER OF TESTS	AVERAGE	RÉSULTS
Testosterone (ng/ml)	16	0.83	- Normal: 10 (52.6%) - High: 6 (31.6%)
17-OHP (ng/ml)	9	4.07	- Normal: 8 (42.1%) - High: 1 (5.3%)
D-4-A (ng/ml)	2	1.95	- Normal: 2 (10.5%)
DHEAS (ng/ml)	1		- Normal: 1 (5.3%)
DHT (ng/ml)	6	0. 39	- High: 6 (31.6%)
UFC (µg/J)	2	6.5	- Decreased: 2 (10.5%)
Prolactin (ng/ml)	11	13.44	- Normal: 9 (47.4%) - High: 2 (10.5%)
FSH (mUI/ml)	11	3.95	- Normal: 8 (42.1%) - Decreased: 3 (15.8%)
LH (mUI/ml)	11	8.52	- Normal: 8 (42.1%) - Decreased: 2 (10.5%) - High: 1 (5.3%)
Estradiol (pg/ml)	11	73.97	- Normal: 9 (47.4%) - Borderline: 1 (5.3%) - Collapsed: 1 (5.3%)

compared to other studies, in particular that of Nacer *et al*. [10], and was explained by the fact that our patients consulted more dermatology and gynaecology. Hyperandrogenism is generally manifested at a young age. In our series, the mean age was 25.3 ± 5 years. Our results are similar to those of different studies on hyperandrogenism. In Algeria, Nacer *et al*. [10] found a mean age of 23 years.

4.2. Clinical Characteristics

4.2.1. History of Hirsutism

In our study, hirsutism constituted 78.9% of the reasons for consultation. The mode of onset of hirsutism is crucial. It appears during the puberty period and develops slowly, suggesting a functional cause. In our series, hirsutism appeared after puberty in the majority of patients (66.7%). In contrast, in the study by Aynaou *et al*. [11] in Morocco, hirsutism appeared for the most part at puberty (58.8%). However, the onset of this symptom is more decisive than the age of onset. Older hirsutism may be post-pubertal. The evolution of hirsutism was either slow or progressive in our study. This finding can be explained by the slow evolution of the symptomatology, also reported by Peigné *et al*. [1].

4.2.2. Gynecological History

Menstrual cycle disorders concerned almost all patients (94.7%). Our results are much higher than those reported by Carmina *et al*. [12] in Italy who reported a frequency of 54.8%. In the study by Aynaou *et al*. [11] in Morocco, cycle disorders were reported in 52.9% of cases. This can be explained by the fact that the majority of our patients seen for cycle disorders came from gynecology. This is in contrast to other series, especially in the West, where patients were directly recruited mainly for hirsutism.

4.2.3. History and Conditions

In our study, the proportion of patients with a personal history or pathology was 57.9%. Obesity was found in 26.3% of cases, hypertension in 2 patients. Obesity in hyperandrogenism may reveal insulin resistance or metabolic syndrome, often associated with PCOS [13]. The hypertension, associated with other signs of hypercorticism, may suggest a Cushing's syndrome. It may also be part of a metabolic syndrome.

In addition, the notion of taking medication was found in 7 patients (36.8%). Some therapeutic classes (synthetic progestins, anticonvulsants) are responsible for hyperandrogenism [1]. However, no cause essentially of drug origin was diagnosed in our patients.

In our series, a family history of hirsutism was found in 31.6% of cases. Our results are consistent with those observed in the literature. Parlak *et al*. [14] reported a family history of hirsutism in the idiopathic hirsutism group, the SOMPK group and the congenital adrenal hyperplasia group respectively in 47%, 50% and 52%. Laassara *et al*. [15] in Morocco and Puri *et al*. [16] in India found a family history of hirsutism in 20.7% and 14% of cases respectively.

4.2.4. Clinical Manifestations

The mean modified Ferriman and Gallwey score (FGm) was 8.9 ± 5.8. Only 9 patients or 47.4% had definite hirsutism (FGm score greater than 8). The threshold value associated with excessive hair growth is 8 based on the 95th percentile [17]. However, a value greater than or equal to 6 is sometimes frequently associated with hirsutism [8]. In our study, hirsutism was mild in 31.6% and moderate in 15.8% of cases. As demonstrated in the literature, the severity of hirsutism varies greatly between series but is not correlated with the severity of the causative condition [8] [10] [18].

Acne present on a single area cannot be considered on its own as a clinical sign of hyperandrogenism [1] [19]. In our study, acne was present in 36.8% of cases. Our results are similar to those observed in the series of Laassara *et al*. [15] in Morocco where acne was present in 30.4% of patients.

Hyperseborrhea was noted in 57.9% of cases. This is significantly higher than the results reported by Laassara *et al*. [15] with 19.6% of cases. Sharma *et al*. [20] reported hyperseborrhea in 4% of cases. These differences may be explained by a greater sensitivity of the sebaceous glands to androgens in our patients.

In our study, certain signs generally observed in cases of tumor etiology were found in 2 patients, *i.e*. 10.5%. These signs are: alopecia and male morphotype with micromastia, muscular hypertrophy and clitoral hypertrophy in the shape of a penis. However, in view of the long-standing appearance of hirsutism and its slow evolution, investigations were in favour of an enzymatic block by 21-hydroxylase deficiency. Aziouaz *et al.* [21] in Morocco found these signs in 10% of cases. Lassara *et al.* [15] in Morocco reported alopecia in 18.5% of their patients, without any other major sign of hyperandrogenism. Similarly, Puri *et al.* [16] in India, Sharma *et al.* [20] in India and Carmina *et al.* [12] in Italy reported androgenic alopecia in 16%, 16% and 3.2% of cases respectively.

Acanthosis nigricans is a marker of hyperinsulinemia [22] in the context of insulin resistance, often associated with PCOS or ovarian hyperthecosis. In our study, it was found in 9 patients (47.4%). Among these, 6 had PCOS, 2 other patients had ovarian hyperthecosis and 1 patient had hyperprolactinemia. Hyperprolactinemia is sometimes associated with SOMPK, which consolidates our results. Our results are much higher than those observed in the literature: Puri *et al.* [16] (India), Laassara *et al.* [15] (Morocco) and Mahajan *et al.* [23] (India) reported Acanthosis nigricans in 14%, 2.2% and 3.3% respectively. This seems to be a particularity of the dark phenotype.

In our series, a buffalo hump and mixed obesity were noted in one patient, purple vergette and hypertension in another. However, Cushing's syndrome was ruled out in these patients. Galactorrhea was noted in one patient. No patient presented with a tumor syndrome. No tumor cause was found in our patients.

These different etiological signs are as rare as their causes, in the context of hyperandrogenism. Laassara *et al.* [15] in Morocco, Parlak *et al.* [14] in Türkiye, Mahajan *et al.* [23] in India, Carmina *et al.* [12] in Italy, had not reported any of these signs. On the other hand, Aziouaz *et al.* [21] in Morocco found signs of

hypercorticism in 20% of the cases in their study.

4.3. Paraclinical Aspects

Elevation of the total testosterone level above 0.8 ng/ml was observed in 6 patients (31.6%). In contrast, in the series by Laassara *et al.* [15], this elevation was noted in 50% of cases. This difference can be explained by the lower threshold in this series (0.6 ng/ml) than in ours.

Total testosteronemia did not correlate with the degree of hirsutism. One of our patients had a moderate Ferriman and Gallwey score (FGm) of 19 in contrast to a normal testosterone level. Conversely, another patient had an FGm score of 2 with an elevated testosterone level of 0.9 ng/ml. This is sometimes explained by the non-reproducibility of the FGm score due to recent hair removal. However, a normal testosterone level, if there are clear signs of clinical hyperandrogenism, should be interpreted with caution. In case of doubt for a SOMPK, it is recommended to repeat the assay or to choose an optional assay (SHBG, free testosterone, D-4-A or 17-OHP assay) [24]. In our study, patients with normal testosteronemia (52.6%) had SOMPK (8 patients, 42.1%), hyperprolactinemia or idiopathic hirsutism.

One patient had a testosteronemia of 2 ng/ml with no evidence of tumors. The diagnosis of tumor was ruled out after the available investigations. Therefore, testosterone levels are only indicative, neither confirming nor eliminating an androgen-secreting tumor [1].

Like testosterone, dihydrotestosterone (DHT) assay allows confirmation of biological hyperandrogenism. In our study, 6 DHT assays were performed. The results were all high, despite normal testosterone levels. Idiopathic hirsutism was diagnosed when there was an isolated elevation of this androgen associated with favorable elements.

About 84.2% had shown an appearance of polymicrocystic ovarian dystrophy. This contributed to the diagnosis of PCOS in 13 patients. Otherwise, the diagnosis of ovarian hyperthecosis was made in 2 patients and hyperprolactinemia in one patient. Pelvic ultrasound helps in the diagnosis of SOMPK. However, this remains a diagnosis of elimination.

4.4. Etiological Characteristics

4.4.1. Micropolycystic Ovary Syndrome (MPOS)

This is the most common cause of hirsutism [25], ranging from 72% to 86% [26]. In our study, PCOS was the most frequent cause of hyperandrogenism at 68.4%. Elsewhere, its variability is increased, ranging from 4% to 21% depending on the study, probably due to variations in definitions, study populations, and sensitivity of the tests used for diagnosis [27] [28]. The following **Table 2** shows the prevalence of SOMPK according to the literature.

4.4.2. Other Etiologies

Ovarian hyperthecosis was diagnosed in 2 patients in our study. We did not find

Table 2. SOMPK prevalence in different studies.

Series	Year	Country	Prevalence
Our study	2021	Senegal	68.4%
Puri *et al.* [2]	2012	India	40%
Mahajan *et al.* [4]	2021	India	29.5%
Aynaou *et al.* [5]	2017	Morocco	70.6%
Azziz *et al.* [6]	2004	USA	82%
Nacer *et al.* [7]	2011	Algeria	59.9%
Carmina *et al.* [8]	2006	Italy	72.1%
Laassara *et al.* [9]	2017	Morocco	41.3%

a prevalence of this condition in the literature. This can be explained by the fact that it represents a severe form of SOMPK and is classified in this register.

Endocrinopathies are rare causes of hyperandrogenism. In our study, hyperprolactinemia was found in 2 patients (10.5%). In the series by Laassara *et al.* [15], it affected 4 patients out of 92, *i.e.* 4.4%. Elsewhere, Nacer *et al.* [10] found it in 8 patients out of 2452, *i.e.* a prevalence of 0.33%. The discrepancy in these different results may be explained by the sample size of the population studied.

Congenital hyperplasia mainly concerns 21-hydroxylase deficiency. In our study, one patient, or 5.3% of the cases, was affected by this pathology. Our results are similar to those observed in the literature, such as Carmina *et al.* [12] in Italy in 2006 who reported 4.3% of HCS.

Idiopathic hirsutism corresponds to 5% to 15% of the causes of hyperandrogenism in the United States and in Italy [1] [6] [12]. In our series, one patient or 5.3% of cases had idiopathic hirsutism. Our results are therefore consistent with the literature. However, in India, the study by Puri [16] showed a much higher proportion, *i.e.* 50%. This may be explained by the prevalence of familial hirsutism in this region.

Ovarian and adrenal virilizing tumors are relatively rare, affecting 0.5% to 2% of hirsute women [26]. In our study, no tumor cause was objectified. In the series of Carmina *et al.* [12], only 2 patients out of 950, *i.e.* 0.2%, had presented androgen-secreting tumors. In the series of Nacer [10], out of 2452 cases, only one patient had an adrenal tumor. This proves the rarity of these pathologies in the causes of hyperandrogenism.

4.5. Limitations of Our Study

Female hyperandrogenism is a frequent cause of consultation in endocrinology, gynecology, dermatology or general medicine. However, we did not receive enough patients during our study period. This can be explained by the fact that they consult more in the "appropriate" services such as dermatology for hirsutism or gynecology for menstrual disorders or menstrual cycle.

Clinically, hirsutism has been underestimated in some patients. Indeed, in

these patients, abnormal hairy areas were masked by recent or repetitive hair removal.

The collection of paraclinical data was not exhaustive. Most of the patients were not able to undergo all of the prescribed examinations because of the high cost of certain explorations. This had an impact on our etiological research.

Differential diagnoses were not formally eliminated in patients with an incomplete workup.

Pelvic ultrasound was accessible on the whole. However, the reports were incomplete, especially in the follicular count.

5. Conclusion

Female hyperandrogenism is a less frequent reason for consultation in endocrinology. It deserves to be further evaluated in a large-scale study focused on epidemiological, clinico-biological and etiological investigation, in order to assess its prevalence and better define its profile in our context.

Conflicts of Interest

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

References

[1] Peigné, M., Villers-Capelle, A., Robin, G. and Dewailly, D. (2013) Hyperandrogénie féminine. *La Presse Médicale*, **42**, 1487-1499. https://doi.org/10.1016/j.lpm.2013.07.016

[2] Azziz, R., Woods, K.S., Reyna, R., *et al.* (2004) The Prevalence and Features of the Polycystic Ovary Syndrome in an Unselected Population. *The Journal of Clinical Endocrinology & Metabolism*, **89**, 2745-2749. https://doi.org/10.1210/jc.2003-032046

[3] Asunción, M., Calvo, R.M., San Millán, J.L., *et al.* (2000) A Prospective Study of the Prevalence of the Polycystic Ovary Syndrome in Unselected Caucasian Women from Spain. *The Journal of Clinical Endocrinology & Metabolism*, **85**, 2434-2438.

[4] Li, R., Qiao, J., Yang, D., *et al.* (2012) Epidemiology of Hirsutism among Women of Reproductive Age in the Community: A Simplified Scoring System. *European Journal of Obstetrics & Gynecology and Reproductive Biology*, **163**, 165-169. https://doi.org/10.1016/j.ejogrb.2012.03.023

[5] Escobar-Morreale, H.F., Carmina, E., Dewailly, D., *et a*l. (2012) Epidemiology, Diagnosis and Management of Hirsutism: A Consensus Statement by the Androgen Excess and Polycystic Ovary Syndrome Society. *Human Reproduction Update*, **18**, 146-170. https://doi.org/10.1093/humupd/dmr042

[6] Azziz, R., Sanchez, L.A., Knochenhauer, E.S., *et al.* (2004) Androgen Excess in Women: Experience with over 1000 Consecutive Patients. *The Journal of Clinical Endocrinology & Metabolism*, **89**, 453-462. https://doi.org/10.1210/jc.2003-031122

[7] Reingold, S.B. and Rosenfield, R.L. (1987) The Relationship of Mild Hirsutism or Acne in Women to Androgens. *Archives of Dermatological Research*, **123**, 209-212. https://doi.org/10.1001/archderm.123.2.209

[8] Bachelot, A., Catteau-Jonard, S., Chabbert-Buffet, N., *et al.* (2020) Nouvelles re-

commandations pour le traitement des hyperandrogénies. SFE.

[9] Bachelot, A., Catteau-Jonard, S., Chabbert-Buffet, N., *et al*. (2020) Nouvelles recommandations pour le traitement des hyperandrogénies de la SFE. Society For Education.

[10] Nacer, K.M. (2011) L'hirsutisme en Algérie: Étude étiologique. *Annales de Dermatologie et de Vénéréologie*, **138**, A277. https://doi.org/10.1016/j.annder.2011.10.362

[11] Aynaou, H., Elilie Mawa Ongoth, F. and Latrech, H. (2017) Profil clinique et étiologique de l'hyperandrogénie chez la femme au CHU Mohammed VI d'Oujda. *Annales d'Endocrinologie*, **78**, 366. https://doi.org/10.1016/j.ando.2017.07.490

[12] Carmina, E., Rosato, F., Jannì, A., Rizzo, M. and Longo, R.A. (2006) Extensive Clinical Experience: Relative Prevalence of Different Androgen Excess Disorders in 950 Women Referred Because of Clinical Hyperandrogenism. *The Journal of Clinical Endocrinology & Metabolism*, **91**, 2-6. https://doi.org/10.1210/jc.2005-1457

[13] Dewailly, D., Hieronimus, S., Mirakian, P. and Hugues, J.N. (2010) Le syndrome des ovaires polymicrokystiques (SOPMK). *Annales d'Endocrinologie*, **71**, e9-e14. https://doi.org/10.1016/j.ando.2009.12.004

[14] Parlak, M., Parlak, A.E. and Toslak, I.E. (2016) Etiologic Factors and Clinical Characteristics of Turkish Adolescent Girls Diagnosed with Hirsutism. *Journal of Clinical and Analytical Medicine*, **7**, 205-209.

[15] Laassara, W. (2017) Profil métabolique des patientes ayant une hyperandrogénie (A propos de 92 cas). Thèse de doctorat, Université Sidi Mohamed Ben Abdallah, Fès.

[16] Puri, N. (2012) A Study on the Clinical and Hormonal Profile of Patients with Hirsutism. *Our Dermatol Online*, **3**, 88-91. https://doi.org/10.7241/ourd.20122.18

[17] Hatch, R., Rosenfield, R.L., Kim, M.H. and Tredway, D. (1981) Hirsutism: Implications, Etiology, and Management. *American Journal of Obstetrics and Gynecology*, **140**, 815-830. https://doi.org/10.1016/0002-9378(81)90746-8

[18] Mokni, S., Hammami, H., *et al*. (2011) Caractéristiques épidémio-cliniques, facteurs de risque, étiologies de l'hirsutisme: À propos de 84 cas. *Annales de Dermatologie et de Vénéréologie*, **138**, A168. https://doi.org/10.1016/j.annder.2011.10.113

[19] Catteau-Jonard, S., Proust-Richard, C., Robin, G., *et al.* (2013) Hyperandrogénie chez l'adolescente. *Encycl Méd Chir, Gynécologie*, 802-A-36.

[20] Sharma, N.L., Mahajan, V.K., Jindal, R., Gupta, M. and Lath, A. (2008) Hirsutism: Clinico-Investigative Profile of 50 Indian Patients. *Indian Journal of Dermatology*, **53**, 111-114. https://doi.org/10.4103/0019-5154.42387

[21] Aziouaz, F., Bensbaa, S., Damoune, I., Yassine, I. and Ajdi, F. (2013) Hirsutisme: Aspects Cliniques, Biologiques et étiologiques (à propos de 20 cas). *Annales d'Endocrinologie*, **74**, 316-317. https://doi.org/10.1016/j.ando.2013.07.254

[22] James, W.D. (2005) Acne. *The New England Journal of Medicine*, **352**, 1463-1472. https://doi.org/10.1056/NEJMcp033487

[23] Mahajan, V.K., Singh Chauhan, P., Chandel, M., *et al*. (2021) Clinico-Investigative Attributes of 122 Patients with Hirsutism: A 5-Year Retrospective Study from India. *International Journal of Women's Dermatology*, **7**, 237-242. https://doi.org/10.1016/j.ijwd.2020.11.007

[24] Déchaud, H., Raverot, V., Plotton, I. and Pugeat, M. (2012) Stratégie d'exploration des hyperandrogénies. In: Letombe, B., Catteau-Jonard, S. and Robin, G., Eds., *Endocrinologie en gynécologie et obstétrique*, Elsevier Masson, Paris, 129-138. https://doi.org/10.1016/B978-2-294-70154-2.00011-X

[25] Liu, K., Motan, T. and Claman, P. (2017) No 350-Hirsutisme: Évaluation et traite-

ment. *Journal of Obstetrics and Gynaecology Canada*, **39**, 1069-1084. https://doi.org/10.1016/j.jogc.2017.08.012

[26] Droumaguet, C., Salenave, S., Brailly-Tabard, S. AND Young, J. (2010) Hyperandrogénie et hirsutisme. EMC (Elsevier Masson SAS, Paris), Endocrinologie, Nutrition, 10.033-G-10.

[27] Azziz, R., Carmina, E., Chen, Z., *et al*. (2016) Polycystic Ovary Syndrome. *Nature Reviews Disease Primers*, **2**, Article No. 16057. https://doi.org/10.1038/nrdp.2016.57

[28] Lizneva, D., Suturina, L., Walker, W., *et al*. (2016) Criteria, Prevalence, and Phenotypes of Polycystic Ovary Syndrome. *Fertility and Sterility*, **106**, 6-15. https://doi.org/10.1016/j.fertnstert.2016.05.003