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Secondary Amenorrhea Revealing A Rare Virilizing Ovarian Tumor, About A Case

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Summary: *Hyper androgenism is common in women. It can have two origins: adrenal and ovarian; It may be benign or malignant. The clinical symptoms are variable; It depends on the importation of the rate of androgens ranging from simple hirsutism to severe virilizing form with secondary amenorrhea. In all cases, a clinical and paraclinical approach is necessary to establish an etiological diagnosis and to adapt an effective therapy*
we report in this publication the observation of a patient with hyperandrogenism secondary to a virilizing ovarian tumor

Keywords: *Hyper androgenism,tumor; MRI, Testosterone , surgery*

I. Introduction

Hyper androgenism is common in women. It can have two origins: adrenal and ovarian; It may be benign or malignant. The clinical symptoms are variable; It depends on the importation of the rate of androgens ranging from simple hirsutism to severe virilizing form with secondary amenorrhea. In all cases, a clinical and paraclinical approach is necessary to establish an etiological diagnosis and to adapt an effective therapy (1)(2).
we report in this publication the observation of a patient with hyperandrogenism secondary to a virilizing ovarian tumor

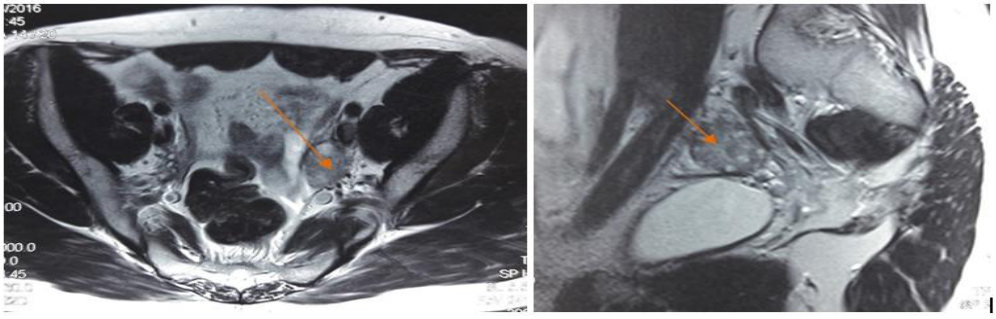
II. Observation

Patient 31 years old married for 3 years consults for secondary amenorrhoea for 2 and a half years associated with infertility. In these antecedents there is a normal pubertal development. The objective clinical examination of signs of virilizing hyper androgenism associating a score hirsutism at 27, Seborrhoeic skin with hair loss, a developed musculature with an android morphotype, a hoarse voice, clitoral hypertrophy at 3 cm. Signs of virilization were rapidly evolving. The BMI was 32 kg / m2. . There were no signs of hypercortisolism. The hormonal balance revealed a tumor hyper androgenism in favor of an ovarian origin (Table I).

TableI: Hormonal results

Parameter	Results	Normes
Testostérone (nmol/l)	36,08	0.24 – 2
Δ4 (ng/ml)	7,33	0,21-3,08
SDHEA (ug/dl)	132,65	30-333
17 OH Before SO(nmol/l) After SO	10,62 25,7	
FS H (Mu/ml)	0, 6	1,8-12
LH (Mu/ml)	< 0,3	0,5- 12

Pelvic ultrasound described normal internal female genitalia. On the other hand, pelvic MRI evoked a left ovotestis, a gonad measuring 36x19 mm, with a predominant tissue component enhanced by injection, surrounded by a peripheral arrangement of micro follicles (Figs 1 and 2). The karyotype was female gonosomal 46 XX.



Figs 1 and 2: Ovarian tumor objectived in M RI

The ovarian tumor markers CA125, CA19-9 and ACE were normal. We retained the diagnosis of virilizing ovarian tumor. The patient was operated by celoscopy benefiting from an ablation of the left ovary and biopsy of the right ovary. The histological study was performed by objectifying a tumor of the mixed sexual cords with Leydig and Sertoli cells.

The re-evaluation done one month postoperative noted a weight loss a decrease in hirsutism score to 17, a fall of testosterone to 2.7 ng / ml, a defect of the FHS gonadotrophins: 6.6 and LH: 3.93 Mu / ml. Pending the resumption of cycles ,the patient will be referred for rehabilitation of the penniform organ

III. Discussion

In women, androgens are produced from cholesterol in ovarian thec cells under the effect of LH and in the zona reticularis of the adrenal cortex under the action of ACTH.

Ovarian and adrenal steroids generate about 25% of the testosterone levels. The rest is produced by peripheral conversion in the liver, adipose tissue and skin cells from androgenic precursors Δ4 (androstenedione), DHEA (di hyro epi androsterone) and its sulfate form (1).

Depending on the elevated rate of circulating androgens, we can be oriented towards the cause. Indeed, SDHEA is exclusively adrenal; An elevation of its rate is seen in benign causes such as adrenal hyperplasia, or malignant causes such as corticosurrenaloma (3). On the other hand, Δ4 is produced both in adrenal gland (50%) and in the ovaries in 50%.

Its elevation, even if preferentially ovarian, can be seen both in polycystic ovary syndrome and in adrenal hyperplasia (2).

Among the etiologies of hyper androgenism, polycystic ovary syndrome is by far the most frequent up to 86% followed by congenital adrenal hyperplasia. Ovarian and adrenal virilizing tumors are rare; They are found in 2% (4).

Androgens act via a nuclear receptor expressed at the level of the skin cells, and the external and internal genital organs. The clinical hyper androgenicity is based on the circulating levels of androgens mirrored by that of testosterone (1)(2).

At the level of the pilosebaceous follicle, hyper androgenia leads to a hirsutism which is a frequent reason for consultation. It will be evaluated according to the score of Ferriman and Gallway. A score> 9 is considered pathological. It may be the first sign of a tumor cause especially when it is severe. Our patient had a score of 27 (1).

When androgen levels are high, this results a freination of pituitary gonadotropins which will be responsible for disorder of gonadal function, cycle disorder type spaniomenorrhea up cold amenorrhea, and infertility (3).

Higher rates can induce bodily changes in women and result in a picture of virilism that combines frontal golf, muscular development with an android morphotype, a change in the voice that becomes more severe and hoarse, and clitoral hypertrophy. Such a picture of hyper-androgenic virilizing must evoke a tumoral cause.

Before a picture of hyper androgeny, the etiological investigation must be minimal. The interrogation must trace its history and its evolution. An old-fashioned picture, with little or no progression of para-pubertal and familial antecedents, tends towards a benign cause such as polycystic ovary syndrome or congenital adrenal hyperplasia (4).

On the other hand, a recent post-pubertal installation, rapidly evolutive, with no family history, associated with signs of virilism oriented towards a tumoral cause alone capable of producing very high levels of androgens.

The starting hormone exploration must include the dosage of total testosterone, SDHEA and the basic 17 OH progesterone optionally supplemented by an ordinary synacthene test (2).

A rise of testosterone above 1.5 ng / ml or even more than 2 ng / ml must obligatorily and rapidly seek a tumoral cause. In adrenal causes, the clinical picture usually associates signs of hypercortisolism because adrenal cortex usually has a variegated androgen-cortisolic secretion (3).

The morphological exploration makes it possible to find the adrenal tumor because usually voluminous and easily identifiable to the CT. In ovarian cases, tumors may be small, included in the ovarian stroma and escaping pelvic ultrasound in the hands of a non-expert. The ultrasound performed in our patient was normal. MRI provides better sensitivity for the exploration of the ovary. In our patient, she had a left ovary measuring 336 x 19 mm, with a predominant tissue component enhancing after injection, a peripheral disposition of micro follicles, evoking an ovostestis. On a CT control, it was concluded to an ovary Left with a central suspect tissue component enhanced after injection(6)

The virilizing tumors of the ovary preferentially secrete testosterone and give a very evocative hormonal profile with a normal SDHEA rate, a testosterone level greater than 1.5 ng / ml, a ratio Δ4 on testosterone of less than 1 (6).

Sex cord-stromal tumors known as androblastoma are very rare tumors. They represent 0.5% of ovarian tumors. Like our patient, they most often reach young women with an average age of 25 years but can see themselves at any age. They are most often manifested in a virile picture in 50% of cases proportional to the number of Leydig cells. Often unilateral tumors (97% of cases). Their size is variable with an average of 15 cm but can reach 35 cm. They are soft in consistency, pale yellow in color, or gray (7) (8)(9) as in our patient.

Microscopically, these tumors are composed of variable proportions of Lydigue cells, Sertoli cells, and in the case of tumors with moderately or slightly differentiated primary gonadal stroma and sometimes heterologous epithelial or mesenchymal elements. These tumors are classified into well, moderately or slightly differentiated. In well-differentiated tumors, Sertoli cells are arranged in tubes, the stroma is scarce, the mitotic activity is low, without capsule breakage. These elements of good prognosis are found in our patient (10)(11)(12) .

Indeed, the prognosis of this tumor depends on the degree of differentiation, the existence or not of capsule breaker and the heterologous elements.

The well-differentiated Sertlo-Leydig mixed tumors have a benign course. On the other hand, almost 60% of tumors with little differentiation have a malignant evolution. In addition to surgery, these poorly differentiated tumors require an assessment of extension and regular monitoring (12)(13).

In addition to the physical repercussions, we must not ignore the psychoaffective repercussion of these tumors which alters the image of oneself and disrupts the life of couple. Psychological support must be provided throughout the care process.

IV. Conclusion

Amenorrhea is a common reason for consultation in endocrinology that may reveal a very rare ovarian tumor. The examination and the clinical examination allow a first etiological orientation and to suspect a tumoral cause in front of a table of acute and evolutive virilism. The hormonal balance will make the difference between

Benign hyperandrogenism and hyper androgenic tumor especially when the testosterone level is greater than 2 ng / ml. Androgenic precursors allow etiological orientation and guide morphological exploration, Pelvic MRI may have a better sensitivity than ultrasound although it may underestimate the size of the tumor.

The surgical management by celoscopy when the tumoral status allows it is preferential, it will leave less scar in these patients already affected

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