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VIRILIZING OVARIAN TUMOR WITH HYPERANDROGENISM : A RARE CASE REPORT

KEY WORDS: Androgenic alopecia, Sertoli-Leydig cell tumor, Virilizing ovarian tumor.

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ABSTRACT

Virilizing ovarian tumor (VOT) is a rare tumor which causes of hyperandrogenism; they account for 1–2% of all ovarian tumors being the Leydig cell tumor is the most common one. Most of the patients produce higher levels of testosterone which is the cause for the primary sign of virilization. We report a case of a 33-year-old woman with progressive signs of virilization, and secondary amenorrhea. Clinical analysis revealed high levels of serum testosterone and low levels of gonadotrophins. Transvaginal ultrasound showed enlarged ovary. Ovariotomy was performed and histopathology confirmed the diagnosis of Leydig cell tumor. After surgery, androgen levels return to normal, and there was regression of the signs of virilization.

INTRODUCTION:

Virilizing ovarian tumor or arrhenoblastoma is a rare androgen secreting ovarian tumor of unknown origin, occurring more frequently in reproductive age group women. More than 75% of incidence is observed in the age group below 30 years and less than 10 % in 30 years and above [1]. All patients showed hyper secretion of androgens [2, 3]. Diagnosis is based on the signs of virilization such as hirsutism in areas of the upper lip, chin, groin, thigh and chest, acne on the face, shoulders, back and chest and hair loss or hair thinning in the temporoparietal area, clitoromegaly [4].

CASE REPORT:

A 33 year old multiparous woman was presented to NRI General Hospital our gynecology department with complaints of one year progression of hoarse voice, an increased hair growth on face and body, amenorrhea and frontal balding. She has two healthy children, aged 3 and 1 year, both delivered vaginally without complications. She was tubectomised. The patient had amenorrhea for the past one year. No past history of hypothyroidism or diabetes mellitus. On examination it was found that she had a hoarse voice. She also had hirsutism which had affected the chin, chest, upper lip, forearms, thighs, buttocks and anterior abdominal wall. Chest examination showed breast atrophy. She was afebrile, with a pulse rate of 84 beats per minute and a blood pressure of 130/80 mmHg. On abdominal examination, the abdomen was found to be soft, no free fluid or mass was observed. Examination of pelvis revealed an enlarged clitoris, normal cervix, and a retroverted uterus with, no palpable mass and non tender fornices. Pelvis and other systems examinations were unremarkable. Blood investigations showed serum total testosterone 562 ng/dl (normal value: 14 - 76 ng/dl). 17-Hydroxyprogesterone, prolactin, FSH, LH, thyroid hormone levels, blood sugars and all other blood parameters were within normal limits. The Ultra Sonography of the abdomen and pelvis showed a bulky uterus (8.8 × 3.4 cm) with normal echotexture. Right ovary was normal in size (3.6×2.1 cm) but left ovary enlarged with loss of echo pattern measuring (6.2×3 cm) isoechoic lesion with cystic areas and minimal vascularity was observed. On consideration of patient's age and multiparity, it was suggested to perform ovariotomy by laparotomy.

DISCUSSION:

Ovarian neoplasms occur to an extent of less than 5% by virilizing ovarian tumors [5]. Hyperandrogenic secretions and signs of virilization raise high suspicion of a virilizing tumor. In females with signs of virilization, careful gynecological examination should be performed, to evaluate the presence of masses or clitoromegaly. Serum androgens are to be measured to exclude an ovarian or adrenal androgen-producing tumor.

In the present case, the high serum levels of total testosterone (740 ng/dl) confirmed the presence of a virilizing neoplasm, while normal serum levels of Dehydroepiandrosterone (DHEA-S) excludes adrenal cause. The suspicion of an androgen-producing tumor was made with testosterone serum levels above 200 ng/dl, and serum values of DHEA-S higher than 600 mg/dl may suggest an adrenal source [6]. High estradiol levels may be due to aromatization of testosterone, resulting in endometrial hyperplasia and vaginal bleeding. Leydig cell tumors are usually small, measuring less than 5 cm of diameter – just slightly bigger than a normal ovary [7]. It may be difficult to identify it by radiological imaging, in part because it is isoechoic to the uterus on ultrasound and isodense on Computerized Tomography (CT) scan [8]. Hilus cell tumours are encountered predominantly in postmenopausal women (average age 58 years) and cause hirsutism and/or virilization in 75% of cases. These tumours secrete testosterone and occasionally oestrogenic activity may be observed. The androgenic manifestations are milder than those associated with Sertoli-Leydig cell androblastomas. Their onset is less abrupt. Patients associated with these tumours usually have signs of virilization, including severe hirsutism, clitoromegaly, frontal balding, altered body fat, increased muscle mass, increased libido, deepening of voice, breast atrophy and pustular acne. In our patient, surgical intervention enabled the final diagnosis of Leydig cell tumor. After treatment, there was a return of serum androgen levels to normal values, and improvement of clinical hyperandrogenism [9].

To conclude, a patient who presents with virilism should be investigated systematically to determine whether the high testosterone levels are of ovarian or adrenal origin. In this patient, blood investigations ruled out an adrenal cause. Radiological imaging revealed and confirmed that the cause of virilization was of an ovarian etiology. Surgical intervention was sought and the diagnosis of virilizing ovarian tumor was made by histopathological examination.

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