REPRODUCTIVE

PP-R-01

OVARIAN LEYDIG CELL TUMOR: A MYSTERIOUS CAUSE OF SEVERE VIRILIZATION

https://doi.org/10.15605/jafes.038.AFES.152

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CASE

A 41-year-old woman with uterine bleeding and progressive virilization for 5 years was referred to our hospital because of worsening anemia. Abdominal CT and transvaginal ultrasound showed an enlarged uterus and left ovary without any tumor. She underwent abdominal hysterectomy and was diagnosed with leiomyoma then evaluated for virilization. Laboratory findings were a total testosterone level of 644 ng/dl (reference, 14-53 ng/dl), DHEAS and 17-OHP levels were normal. 8 am cortisol level after 1 mg dexamethasone suppression test was suppressible. According to markedly high testosterone levels and normal adrenal androgen levels, an androgen-secreting ovarian tumor was highly suggestive. Since the patient suffered from severe virilization and did not desire fertility, she underwent bilateral oophorectomy. The histopathology result showed a Leydig cell tumor on the left ovary. At the follow-up clinic, where she is receiving estrogen replacement, her testosterone level returned to a normal level and virilization was gradually improved.

KEYWORDS

Leydig cell tumor, virilization, hirsutism, androgen-secreting tumor

PP-R-02

46,XY 5-ALPHA REDUCTASE 2 DEFICIENCY SYNDROME IN A 19-YEAR-OLD PHENOTYPIC FILIPINO FEMALE: A CASE REPORT

https://doi.org/10.15605/jafes.038.AFES.153

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CASE

Steroid 5 alpha-reductase 2 deficiency is a rare autosomal recessive disease caused by genetic mutation which results in the non-conversion of Testosterone to Dihydrotestosterone. The case is a nineteen-year-old, born from a none consanguineous marriage, reared as a female, who presented with primary amenorrhea. At the age of thirteen, she developed virilization with no development of secondary female sex characteristics. There were two palpable soft and non-tender inguinal masses measuring three centimeters. The external genitalia showed an acuminate pubic hair, a three-centimeter phallus-like structure, with fused labio-scrotal folds with ruggae, an empty scrotal sac, and a three-centimeter blind vaginal pouch where urine passes through. The testosterone level was at a normal level, with a testosterone-dihydrotestosterone ratio of less than ten. The karyotype result revealed a male, 46,XY chromosome with no aberrations. The genetic analysis showed two pathogenetic variants of the SRD5A2 gene. After a multidisciplinary discussion, the patient adopted the male gender.

KEYWORDS

disorder of sexual development, 5-alpha-reductase 2 deficiency