Nothing to Disclose

Androgen profile differs to adults in adolescent girls with polycystic ovary syndrome

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Background

Polycystic ovary syndrome (PCOS) is characterised by ovulatory dysfunction and hyperandrogenism. There is a strong association with obesity, and the prevalence of adolescent PCOS is increasing in line with the increase in childhood and adolescent obesity. A diagnosis of PCOS in adolescence has lifelong implications. It is the commonest cause of infertility in women and is associated with increased risk of metabolic syndrome. The criteria used to diagnose PCOS in adults may not be appropriate for the evaluation of adolescents as some criteria may be features of normal puberty, including menstrual irregularity, acne and polycystic appearances of the ovaries.

In adults the assessment of hyperandrogenism is based upon either clinical or biochemical criteria, however most expert groups include a measurement of testosterone (total, unbound or free) in the evaluation of PCOS[2]. The ovary is the major source of androgen excess in PCOS. Androstendione is the immediate precursor to testosterone. Elevated levels of LH in PCOS stimulate theca cells to secrete androstendione, which is converted peripherally to testosterone. DHEAS is a primary adrenal androgen and has a much lesser impact on the clinical signs of hyperandrogenism.

Objectives

To describe androgen profiles in adolescent girls presenting with clinical features of PCOS, to identify the most sensitive marker of hyperandrogenism in adolescent PCOS.

Methods

A retrospective case review was undertaken of patients with clinical characteristics of PCOS (clinical hyerandrogenism plus oligomenorrhoea / primary or secondary amenorrhoea, with or without polycystic ovaries on ultrasound) attending a single centre between 2005 and 2015.

Results

Data are reported as median (range). Data were collected from 40 patients, age 15.49 (11.84 – 18.02) yrs. BMI-SDS was 2.42 (-1.25 – 3.91), and 25/40 (62.5%) were overweight/obese (BMI SDS >1.75). Androgen profiles and sex hormone binding globulin (SHBG) levels at presentation are given in Table 1.

Table 1: Androgen and sex hormone binding globulin levels in 40 patients

Biochemical Marker (Normal Range)	Number of patients with abnormal level (%), median level (range)
Testosterone (0-3.5nmol/l)	2 (5%), 1.7 (<0.7-4.7)
DHEAS (1.6-7.8 umol/l)	11 (28%), 5.7 (1.8-14.9)
Androstendione (2-5.4 nmol/l)	26 (65%), 6.6 (1.3-18.2)
SHBG (25-145 nmol/l)	20 (50%), 24.5 (9-200)
LH: FSH ratio (<2)	14 (35%), 1.71(0.1-5.8)
Free Androgen Index (0.5-6.5)	19 (48%), 5.99 (0.72-33.5)

Conclusions

In this population of girls, with clinical feature of PCOS, hyperandrogenism would not have been diagnosed in 60% of patients if only testosterone was measured.

The biochemical profile in our adolescent cohort contrasts to data reported from adult population, where only 20-30% of patients have increased androstendione levels[2]. There is little data describing normal androgen levels during puberty. Puberty itself causes insulin resistance leading to increased androgen production from ovarian theca cells[3]. Obesity is a common finding in our cohort, so it may be that elevated androstendione levels simply reflect the age and body composition of the patients, however, it should be noted that these biochemical features are seen in the presence of hirsuitism, which would not be expected in healthy girls of this age, and it is this observation that leads us to propose that androstendione should be included in the diagnostic evaluation of girls presenting with features of PCOS.

References

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