

# Hirsutism in the United Arab Emirates: a hospital study

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## Summary

**In a study of 102 women (Arabs and Asians) with hirsutism, polycystic ovary syndrome was diagnosed in 93 patients (91%) including 26 cases with hirsutism and regular menstrual periods. In more than half of the cases the diagnosis was made on the basis of transabdominal ultrasonography, which was the single test with the highest yield. Luteinising hormone hyperresponsiveness to gonadotropin-releasing hormone was positive in only seven of 50 patients tested. Late onset congenital adrenal hyperplasia due to 21-hydroxylase deficiency was identified in two cases, thus forming a prevalence of 8% in those who underwent adrenocorticotropin stimulation. In one case, polycystic ovary syndrome was an associated condition. In this study of a population of women with hirsutism and regular menses, the majority had polycystic ovaries. It is suggested that the term 'idiopathic or racial' hirsutism should not be applied unless a thorough evaluation has failed to reveal a diagnosable underlying disorder.**

**Keywords:** hirsutism, polycystic ovary syndrome, Asians, Arabs

Hirsutism is a common clinical problem with different aetiologies which vary in frequency in different populations.<sup>1,2</sup> The large range of familial and racial differences in the extent and acceptability of hair on various body sites makes objective assessment essential. However, various methods have been proposed to assess the degree of hirsutism.<sup>3–5</sup> Careful endocrinological evaluation of women defined as having hirsutism have shown increased androgen metabolism in up to 90%.<sup>6</sup> It has long been thought that racial or idiopathic hirsutism was the primary type found in the Middle Eastern community.<sup>7,8</sup> This current study evaluated the most common androgenic aetiologies of hirsutism in 102 consecutive patients presenting with hirsutism to an out-patient endocrinology service in Abu Dhabi.

## Subjects and methods

Over a period of four years, 102 women with hirsutism were studied. These patients attended the endocrine clinic in Al Jaziera Hospital in Abu Dhabi, the capital of the United Arab Emirates (UAE). This hospital

and two others cater to the medical needs of the Abu Dhabi population, and provide specialised medical services. The group includes UAE natives, non-UAE Arab expatriates and Asians (from the Indian subcontinent). The UAE community is multi-ethnic and these nationals reflect the bulk of the population.

All but 27 had a history of oligo/amenorrhoea since menarche. None had received any steroidal medications for at least eight weeks prior to being studied. Hirsutism was described as mild (Ferriman-Gallwey score 6–9), moderate (10–14), or severe (> 15).<sup>3</sup> Most of our patients fell between moderate to severe scores.

Eighty-seven patients (85%) underwent pelvic ultrasonography, which was evaluable. In the rest, the examination was either inconclusive or declined by the patient. The ovary was defined as being polycystic, if there were multiple cysts (10 or more), 2–8 mm in diameter, arranged either peripherally around the stroma or distributed throughout an increased amount of stroma, or both. Enlargement of the ovaries (dimensions of normal adult female ovary based on anatomic specimen length 2.2–5.5 cm, width 1.5–2 cm, AP diameter of 1.5–3 cm) was considered as a positive finding in this context.<sup>9,10</sup> Morning samples for hormonal assessment, collected during the follicular phase (days 5–9) of the cycle included basal levels of the following hormones: luteinising hormone, follicle-stimulating hormone, prolactin, testosterone, dehydroepiandrosterone, 17-hydroxyprogesterone, androstenedione and cortisol. Estimation of 24-hour free cortisol in urine was performed if Cushing's syndrome was clinically suspected. Fifty patients (49%) had gonadotropin-releasing hormone stimulation test. The test was performed by administration of a single intravenous injection of 100 µg gonadotropin-releasing hormone during the mid-follicular phase and blood samples were obtained before and 20 and 60 minutes after the injections.<sup>11</sup>

Twenty-five patients (24.5%), some with raised basal levels of dehydroepiandrosterone, testosterone and 17-hydroxyprogesterone (three to five times the basal level) and others who failed to respond to conventional treatment for polycystic ovaries (cyproterone acetate, spironolactone, clomiphene, contraceptive pills and bromocriptine for associated hyperprolactinaemia) underwent an adrenocorticotropin stimulation test. A single intravenous bolus of 0.25 mg of synthetic adrenocorticotropin was given in the mid-follicular phase at 08.00 h; blood samples were

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obtained at 0, 30 and 60 minutes for determination of serum 17-hydroxyprogesterone, progesterone, dehydroepiandrosterone, cortisol and androstenedione. Assays of 11-deoxycortisol, 17-hydroxypregnenolone and free testosterone were not available in our laboratories. Polycystic ovarian disease was diagnosed with a combination of clinical, biological and ultrasonographic parameters (box).

Diagnoses of Cushing's syndrome, acromegaly, prolactinoma, or thyroid disorders were excluded by performing the appropriate static and dynamic hormonal assays and in appropriate patients, radiological tests including computed tomography (CT).

## Results

Of the 102 patients, 72 were Emirian nationals, 18 were Arab expatriates (non-UAE Arabs), and 12 were Asians from the Indian subcontinent. Their age ranged from 14–37 years with an average of 22.3 years. Sixty-three patients were obese (body mass index > 25 kg/m<sup>2</sup>). In the 50 patients who underwent a gonadotropin-releasing hormone stimulation test, an enhanced response as defined by Marshall *et al*<sup>11</sup> (ie, at least a four-fold rise of the basal level of luteinising hormone at 20 minutes after the gonadotropin-releasing hormone injection) was observed in only seven patients (14%). Of these, only one had abnormal ultrasonographic findings consistent with polycystic ovary syndrome. An enhanced response of 17-hydroxyprogesterone (10 times or more the basal level) to adrenocorticotropin stimulation was obtained in only two cases of the 25 patients tested. These two patients were thus diagnosed as late-onset congenital adrenal hyperplasia due to 21-hydroxylase deficiency. In the one patient in whom 17-hydroxyprogesterone suppression was attempted, the basal level was suppressible by dexamethasone; this patient also had ultrasonographic features consistent with polycystic ovary syndrome. In the whole group, 93 patients (91%) were diagnosed as having polycystic ovary syndrome alone. These included 26 patients who had previously been considered to have idiopathic hirsutism (hirsutism with regular menstrual periods). In this group, hormonal changes consistent with polycystic ovary syndrome (high basal level of serum luteinising hormone, testosterone, androstenedione, and high luteinising hormone/follicle-stimulating hormone ratio) were observed in 20/26 (77%) and positive ultrasonographic changes of polycystic ovary syn-

drome in 12/23 (52%). The relevant data are shown in table 1. An ovarian tumour was diagnosed in one patient who presented with hirsutism, virilisation and recent menstrual changes. She showed a very high basal level of serum testosterone along with positive radiological findings including CT scan. Another patient was proven to have bilateral dermoid cyst by laparotomy and histopathology. Five remaining patients were classified as having 'uncategorised hirsutism' on the basis of upper limit values of serum testosterone alone. The rest of their hormonal assays and ultrasonographic findings were within normal limits. These patients underwent adrenocorticotropin stimulation test to rule out partial or minor forms of late onset congenital adrenal hyperplasia. Their responses were also within normal limits. The aetiology of the diagnosis of hirsutism is shown in table 2.

## Discussion

Both ovaries and adrenals are the sources of androgens. A classic cause of hirsutism is polycystic ovary disease; this was first recognised as the clinical triad of amenorrhoea, hirsutism and obesity by Stein and Leventhal some 60 years ago.<sup>12</sup> Many women who complain of excess hair, however, are not obese, nor do they have menstrual disturbances. These women are often labelled as having idiopathic hirsutism. This diagnosis, however, was seriously challenged by Adams *et al* after reporting a high prevalence of polycystic ovary syndrome (92%) in a sample of women with hirsutism and normal menses.<sup>10</sup>

**Table 1** Laboratory findings of 93 patients with polycystic ovary syndrome alone. Normal ranges in parentheses

Test	n	(%)
Positive ultrasound	50/87	(57)
Raised serum concentration of luteinising hormone (0.7–8.0 mIU/ml)	44/93	(47)
Raised luteinising hormone/follicle-stimulating hormone ratio (> 2)	40/93	(43)
High serum concentration of testosterone (0.06–0.8 ng/ml)	60/93	(65)
High serum concentration of androstenedione (0.2–3.1 ng/ml)	29/69	(42)
High serum concentration of dehydroepiandrosterone (350–4300 ng/ml)	15/77	(19.5)

**Table 2** Aetiology of the diagnosis of hirsutism in the whole group

Diagnosis	n	(%)
Polycystic ovary syndrome (alone)	93	91
Polycystic ovary syndrome and congenital adrenal hyperplasia	1	(1)
Congenital adrenal hyperplasia (alone)	1	(47)
Ovarian tumour	1	
Bilateral dermoid cyst	1	(1)
Uncategorised	5	(5)

### Diagnostic criteria for polycystic ovaries

- Two of the following groups occurring together:
- amenorrhoea, oligomenorrhoea, non-mechanical sterility or hirsutism
  - biological hyperandrogenism (increased testosterone and/or androstenedione)
  - ultrasonographic abnormalities<sup>9,10</sup>

In our group the relatively high yield of positive pelvic ultrasonography in the diagnosis of polycystic ovary syndrome appeared to make it the investigation of choice in the evaluation of hirsutism. The result is comparable to that reported in a series of 350 patients in the UK (60%).<sup>2</sup> Even higher results were obtained by Adams *et al* among hirsutes with menstrual disturbances (87%) and in others with regular periods (92%).<sup>10</sup> These rates are operator-dependent and possibly reflect the more accurate assessment provided by the use of a vaginal transducer which usually allows closer access to the ovaries.<sup>13</sup> Such a procedure was not feasible in our patients for socio-legal reasons.

Variations in basal levels of luteinising hormone in polycystic ovary syndrome are not unusual. Franks reported that 50–70% of patients with polycystic ovary syndrome exhibit a high basal level of luteinising hormone.<sup>14</sup> Adams<sup>10</sup> and Grulet<sup>15</sup> observed the same finding at rates of 66% and 62%, respectively. The proportion of patients with a raised luteinising hormone/follicle-stimulating hormone ratio in our series was lower than those reported by Adams *et al*<sup>10</sup> (66%) in the UK and by Yilmaz *et al* in their series of 31 Turkish women (55%).<sup>16</sup> The value of dynamic endocrine investigation in polycystic ovary syndrome has been extensively emphasized and luteinising hormone hyperresponsiveness to the gonadotropin-releasing hormone stimulation test has been advocated as the mainstay of diagnosis.<sup>15,17,18</sup> Since few patients in our series showed an enhanced response of luteinising hormone to gonadotropin-releasing hormone stimulation, the stimulation test was of less value than ultrasonography in the diagnosis of polycystic ovary syndrome in our patients.

The frequency of polycystic ovary syndrome in different populations is variable. It appeared extremely high in our group (91%), compared to the 62% and 60% found in the studies of Adams<sup>10</sup> and O'Driscoll,<sup>2</sup> respectively. This study indicates that an initial clinical diagnosis of idiopathic hirsutism in a Middle Eastern population may more often be found to be polycystic ovary syndrome after investigation. This is in agreement with the views of Adams and colleagues who suggested that the idiopathic label was inappropriate for most patients with hirsutism and regular periods.<sup>10</sup>

While polycystic ovary syndrome remains

the commonest cause of hyperandrogenemia and hirsutism, other more morbid causes need to be ruled out. Late onset congenital adrenal hyperplasia due to 21-hydroxylase deficiency appears to have variable ethnic distribution. Elder-Geva *et al* reported a prevalence of 8% of this enzymatic defect in 170 Israeli Jewish women with hirsutism,<sup>1</sup> whereas this defect has been found in 2–6% of patients with oligomenorrhoea or hirsutism in the UK.<sup>19,20</sup> Others have estimated frequencies ranging from 1–30%.<sup>1</sup> Obviously, in our series, the number of late-onset congenital adrenal hyperplasia cases due to 21-hydroxylase deficiency is comparatively small, and neither of the two patients with this defect is of Arab origin. The blunted response of dehydroepiandrosterone to adrenocorticotropin stimulation in our series may argue against the diagnosis of 3 $\beta$ -hydroxylase deficiency in these patients. Nevertheless, when facilities become available to us, the screening for appropriate steroid intermediates (17-hydroxypregnenolone, and 11-deoxycortisol for 11-hydroxylase deficiency) will be imperative. The presence of co-existing polycystic ovary syndrome in one patient with late-onset congenital adrenal hyperplasia emphasizes that this ovarian finding does not rule out the presence of other important pathology.

Some authors advocate the use of the adrenocorticotropin stimulation test in every patient with hirsutism,<sup>1</sup> but we tend to believe that their opinion may be influenced by the fact that late-onset congenital adrenal hyperplasia is frequently encountered in their community. The test is not cost-effective and the majority of our patients have radiological and hormonal changes in keeping with polycystic ovary syndrome, thus performing the adrenocorticotropin stimulation test in hirsutism routinely is probably of limited practical value in this community. The test is better reserved for those patients who do not respond to the conventional treatment for polycystic ovary syndrome. Finally, there is a need for further studies to evaluate hirsutism in each major ethnic group of this community. The magnitude of 'racial hirsutism' remains obscure. Large number of families will need to be evaluated in order to determine its prevalence.

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