Evaluation of socio-demographic and clinical characteristics of PCOS patients attending a tertiary care institute in Colombo

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Abstract

Background and objectives: Polycystic ovary syndrome (PCOS) is a common endocrine disorder with heterogeneous aetiology. It is characterized by irregular menses and or oligo/anovulation, hyper-androgenism, and polycystic ovaries. The prevalence and diagnosis of PCOS changes depending on which clinical criteria are utilized to confirm the diagnosis. The prevalence can be high as 8–13% when the Rotterdam criteria are used. However, there is significant inter-individual variation in presentation. We have studied the socio-demographic and clinical characteristics of PCOS patients attending the Endocrinology clinic in a tertiary care institute in Sri Lanka.

Methods: A descriptive cross sectional study was conducted from September 2019 to September 2020 at the Endocrinology Unit of the National Hospital of Sri Lanka. All the patients who met the inclusion and exclusion criteria and who has a diagnosis of PCOS made according to Rotterdam criteria were recruited in to the study. After obtaining informed written consent, the data was collected using an interviewer administered questionnaire. HOMA-IR was calculated using the fasting insulin and blood glucose level.

Results: The study enrolled sixty females. The mean age was 26.7 years (range 18–44). The mean weight was 64.8 (SD = 11.9) kg and BMI was 27.1 (SD = 4.8) kg/m$^2$. According to Asian BMI cut-offs, 1 (1.7%) patient was underweight and 13 (21.7%) had normal weight. Forty six (76.7%) had their weight in the overweight or obese category. Fifty four (90.0%) patients had clinical or biochemical evidence of hyperandrogenism while 24 (40%) had polycystic ovaries on trans-abdominal ultrasound scan and 50 (83.3%) had irregular menstrual cycles. According to the body fat percentage assessed by the whole body DEXA scan 4.1% normal body fat, while 50.0% and 45.8% had overweight and obesity respectively. HOMA-IR detected 61.1% to have high insulin resistance. Out of the patients who had USS of the abdomen 27.5% had co-existent non-alcoholic fatty liver. Fifty four percent of the patients had sub/infertility.

Conclusions: The majority of the population were overweight or obese and had higher prevalence of insulin resistance and non-alcoholic fatty liver. Out of the clinical characteristics used to make the diagnosis of PCOS, the presence of clinical or biochemical evidence of hyperandrogenism and irregular menstrual cycles are more common than the detection of polycystic ovaries on trans-vaginal USS. The higher prevalence of overweight, obesity, insulin resistance and NAFLD associated with PCOS makes the diagnosis and management of the disease crucial to prevent long term consequences of the disease.
Keywords: PCOS, Socio-demographic, Clinical, Hyperandrogenism, Irregular menstrual cycles, Tertiary care institute in Sri Lanka

Background

Polycystic ovary syndrome (PCOS) is considered as the most common endocrine disorder prevalent in females. Multiple genetic and environmental factors play a complex role in its aetiology. Patients can present with varied symptoms, but predominant clinical manifestations include irregular menses and or oligo/anovulation, hyper-androgenism, and polycystic ovaries. The prevalence of PCOS is variable in different populations of people with varied geography and ethnicity. Moreover, the different criteria that are in practise to diagnose the disease contribute to the differences in the prevalence among different groups of people that are observed. The widely used Rotterdam criteria diagnose the disease in about 8–13% of females [1]. In the western countries, polycystic ovarian syndrome (PCOS) has a prevalence of 4–12% rendering it as the most prevalent endocrine disorder of reproductive-age women [2]. A prevalence of 6.5–8% has been reported in European countries [3].

Clinical manifestations of the disease include menstrual irregularities, hirsutism, and commonly infertility or subfertility. Commonly seen menstrual irregularities in PCOS patients include prolonged erratic menstrual bleeding, amenorrhea and oligomenorrhea [4]. Nevertheless, some of the females with PCOS will have normal menstrual cycles with or without anovulation [5]. On presentation, majority of females with oligomenorrhea and around half of females with amenorrhea will have PCOS [6]. Majority of females, with clinical features of androgen excess will eventually be diagnosed to have PCOS [7]. Androgen excess can be characterized by hirsutism, acne, androgenic pattern of hair loss etc. Hirsutism, which is excessive growth of terminal hair, is a frequent clinical feature of hyperandrogenism that can be seen in majority of females with PCOS [8]. Hirsutism can be objectively assessed using a modified Ferriman–Gallwey scoring system [9]. Majority (over 90%) of females with normal menstrual cycles and hirsutism are identified through ultrasound to have polycystic ovaries [10]. Furthermore, PCOS can be detected in 50% of women without significant hirsutism [11]. Acne is also a clinical feature indicative of hyperandrogenism, but is uncommonly seen in PCOS. Around one third of females with PCOS will have acne on diagnosis [7, 12]. Around forty percent of females with severe acne will have the diagnosis of PCOS made on presentation [13]. Infertility can also be objectively assessed using a modified Ferriman–Gallwey scoring system [9]. Majority (over 90%) of females with normal menstrual cycles and hirsutism are identified through ultrasound to have polycystic ovaries [10].

PCOS diagnostic criteria had been evolved over past few decades. Up to present, three sets of diagnostic criteria are available for the diagnosis. The first criteria that had been described is the National Institutes of Health/National Institute of Child Health and Human Disease (NIH/NICHD) criteria followed by the European Society for Human Reproduction and Embryology/American Society for Reproductive Medicine (ESHRE/ASRM); Rotterdam criteria; and the latest Androgen Excess and PCOS Society diagnostic criteria [17–19]. All three diagnostic groups consider PCOS as a diagnosis of exclusion. Other alternative diagnoses such as Cushing syndrome, hyperprolactinemia, thyroid disorders congenital, adrenal hyperplasia, non-classic adrenal hyperplasia, androgen-secreting tumour, idiopathic hirsutism and idiopathic hyperandrogenism must be eliminated prior to the diagnosis of PCOS.

Pathogenesis of the development of PCOS is multifactorial. Family history has been described as a possible risk factor for PCOS based on the accumulation of cases within families [20]. Increased diagnosis of PCOS or its clinical features among first-degree relatives is indicative of genetic predisposition [21]. Furthermore, higher concordance has been detected in monozygotic versus dizygotic twins [22].

PCOS is connected with numerous other disease conditions. Fat accumulation with development of overweight and obesity often precedes the appearance of the clinical manifestations of PCOS. Adhering to a healthy lifestyle with dietary modifications and exercise therapy has been shown to decrease weight, improve insulin resistance, decrease abdominal fat, decrease testosterone and improve features of hyperandrogenism in females with PCOS [23, 24]. Diabetes mellitus including Type 1, Type 2, and gestational diabetes is linked with a higher occurrence of PCOS. Codner et al. screened 42 women...
Recruiting adult females aged > 18 years, who are premenopausal, who has a previous or a new diagnosis of PCOS according to the Rotterdam criteria without prior treatment were recruited to the study [18].

According to the European Society for Human Reproduction and Embryology/American Society for Reproductive Medicine (ESHRE/ASRM)/ Rotterdam criteria/ 2003, fulfilment of two out of three of the below criteria is necessary to make the diagnosis of PCOS, after excluding the other conditions that mimic the disease such as Cushing syndrome, hyperprolactinemia, thyroid disorders congenital, adrenal hyperplasia, non-classic adrenal hyperplasia, androgen-secreting tumour, idiopathic hirsutism and idiopathic hyperandrogenism [18].

- Oligo- and/or anovulation
- Clinical and/or biochemical signs of hyperandrogenism
- Polycystic ovaries (by ultrasound)

Females who are pregnant and who has not given informed written consent were excluded from the study. All the participants meeting the above mentioned inclusion and exclusion criteria was included to the study over a period of ten months.

Data collection
The selected participants who were attending the endocrinology clinic at the National Hospital of Sri Lanka was educated regarding the study by a team of researchers to explain about the research and to invite them for the study. Informed written consent was taken by the interviewer and the data was collected with the aid of an interviewer administered questionnaire administered by trained researchers and by measurement of necessary anthropometric measures (weight, height, waist circumference, modified Ferriman Gallway score, blood pressure). Body Mass Index (BMI) was calculated by dividing weight in kilograms by height in meters squared (kg/m2). All the participants were evaluated with...
Luteinizing hormone (LH) (Chemiluminescent immunoassay analyser), Follicular stimulating hormone (FSH) (Chemiluminescent immunoassay analyser), serum total Testosterone (Fully automated immunoassay analyser), Post prandial blood glucose (PPBG) (GOD- PAP5 method, Abbott architect analyser), Post prandial blood glucose (PPBG) (GOD- PAP5 method, Abbott architect analyser), Glycated haemoglobin levels (HbA1c) (HPLC method, HPLC analyser), Fasting Lipid profile including total cholesterol (TC), Triglyceride (TG) and Low density lipoprotein- cholesterol (LDL-C) (Fully automated chemistry analyser ABBOTT architect plus C8000), Aspartate aminotransferase (AST) (Fully automated chemistry analyser Bechman Coulter- AU 680), Alanine aminotransferase (ALT) (Fully automated chemistry analyser Bechman Coulter- AU 680) and serum fasting insulin (Chemiluminescent enzyme immunoassay, Immulite 1000 analyser) measurements. Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was calculated using the fasting insulin and blood glucose level [34]. Body fat percentage was measured using the whole body Dual-energy x-ray absorptiometry (DEXA) scan.

**Statistical analysis**
Data was analysed using Statistical Package for Social Sciences 18. Descriptive data was used to describe the population characteristics. Chi-square test was used to compare categorical variables.

**Ethical issues**
Ethical approval was obtained from the ethical review committee of the National Hospital of Sri Lanka as well as by the ethics review committee of the post graduate institute of medicine, University of Colombo, Sri Lanka (ERC/PGIM/2020/029). Documents were encoded with numerical values to avoid personal identification. All the measures were taken to ensure confidentiality of subjects.

**Definitions**
Modified Ferriman Gallway scores more than 8 is considered abnormal [9]. Furthermore, scores between 8–15 are considered as mild hirsutism, 16–25 as moderate, and scores >25 as severe hirsutism [9]. The diagnosis of diabetes was made if FPG ≥ 126 mg/dL (7.0 mmol/L), or 2-h plasma glucose ≥ 200 mg/dL (11.1 mmol/L) during an OGTT or HbA1C ≥ 6.5% according to the American Diabetes Association 2020 criteria [35]. In the age group of 20–39 years of Asian females, body fat >35% is considered as overweight and >40% is considered as obese while in the age group of 40–59 years of Asian females, body fat >36% is considered as overweight and >41% is considered as obese [36].

**Table 1** Study group Socio-demographic characteristics

| Ethnicity % (N)     | Number (N=60) (%) |
|---------------------|-------------------|
| Sinhalese           | 47 (78.3)         |
| Muslim              | 8 (13.3)          |
| Sri Lankan Tamil    | 2 (3.3)           |
| Others              | 3 (5.0)           |

| Education level % (N) | Number (N=60) (%) |
|-----------------------|-------------------|
| No education          | 1 (1.7)           |
| Grade 5               | 2 (3.3)           |
| Ordinary Level        | 21 (35.0)         |
| Advanced Level        | 24 (40.0)         |
| Tertiary              | 12 (20.0)         |

| Employment status % (N) | Number (N=60) (%) |
|-------------------------|-------------------|
| Employed                | 29 (48.3)         |
| Unemployed              | 31 (51.7)         |

| Marital status % (N)    | Number (N=60) (%) |
|-------------------------|-------------------|
| Married                 | 20 (33.3)         |
| Unmarried               | 39 (65.0)         |
| Other                   | 1 (1.7)           |

**Table 2** Study group Anthropometric measurements

| Minimum | Maximum | Mean (SD) |
|---------|---------|-----------|
| Height (cm) | 125 | 172 | 155.2 (7.5) |
| Weight (kg)  | 40 | 103 | 64.8 (11.9) |
| Waist circumference (cm) | 28 | 105 | 64.9 (28.8) |
| BMI (kg/m2)   | 17.2 | 41.8 | 27.1 (4.8) |

**Results**
60 participants who met the inclusion and exclusion criteria was included in to the study. Mean age of the population was 26.7 (SD ± 6.7) years with a minimum age of 18 years and the maximum age of was 44 years. The socio-demographic characteristics are summarised in Table 1.

The mean weight is 64.8 (SD ± 11.9) kg and BMI is 27.1 (SD ± 4.8) kg/m². The mean waist circumference is 64.9 (SD ± 28.8) cm. The study group anthropometric measurements are summarised in Table 2.

According to Asian BMI cut-offs, 1 (1.7%) patient was underweight and 13 (21.7%) had normal weight [37]. Forty six (76.7%) had their weight in the overweight or obese category. Body fat percentage measurement on DEXA scan detected 50.0% as overweight and 45.8% as obese (Fig. 1) [36].

The study extensively looked at the group characteristics such as birth weight, pubarche and menarche of the study participants. Hirsutism was assessed using the modified Ferriman Gallway scoring system. All the patients underwent multiple laboratory tests including...
LH, FSH, Serum total Testosterone, FBG, PPBG, Glycated haemoglobin (HbA1c), TC, TG, LDL-C, AST, ALT and Serum fasting insulin level as well as the body fat percentage by DEXA scan. The testosterone reference range for adults female is taken as 14–76 ng/dL according to the lab reference range. The results are summarized in the Table 3.

Known associations of the development of the PCOS is summarised in the Table 4. 21.7% patients had a family history of PCOS while none had a history of epilepsy needing anti-epileptic medications of maternal anti mul-lerian hormone (AMH) administration in the second trimester.

Fifty four (90.0%) patients had clinical or biochemical evidence of hyperandrogenism while 24 (47%) had polycystic ovaries on trans-vaginal ultrasound scan and 50 (83.3%) had irregular menstrual cycles (Table 5). According to the Rotterdam criteria the presence of ≥ 12 follicles in each of two ovaries measuring 2–9 mm in diameter and/or ovarian volume ≥ 10 mL on trans-vaginal ultrasound scan (USS) is considered as polycystic ovaries [18]. Elevated LH: FSH ratio > 2 was seen in 20 (33.3%) patients although it is not a component of the diagnostic criteria.

Clinical or biochemical hyperandrogenism and polycystic ovaries on USS was seen in 36.6% of the study group while 80% had clinical or biochemical hyperan-droensim and menstrual irregularities. Menstrual irregularities and polycystic ovaries on USS was seen on 33.3% of the sample population. According to this findings it is evident that majority of the study population was diagnosed with PCOS using the presence of clinical and biochemical hyperandrogenism and menstrual irregularities.

Acne is considered as a feature of hyperandrogenism and it was detected in 16 patients (26.7%). The results are summarised in the Fig. 2.

Out of the population, 4 patients (6.7%) was diagnosed to have diabetes mellitus though 14 patients (23.3%) had evidence of clinical insulin resistance demonstrated by the presence of Acanthosis nigricans. Few patients had pregnancy complications while 21.7% had subfertility/ infertility. From the females who has actively tried to conceive, fifty four percent had sub/infertility. Out of the patients who had USS of the abdomen 27.5% (n = 14) had co-existent non-alcoholic fatty liver (Table 6).

HOMA-IR detected 61.1% to have high insulin resist -ance. HOMA- IR value < 2 is considered as normal in the calculations [34].

When the above factors were compared according to the Asian BMI cutoffs, the presence of Acanthosis nigricans (p = 0.006), fatty liver on ultra sound scan (p = 0.005) had a significant association with the BMI category (Table 7). Furthermore, the presence of Acanthosis nigrians were significantly higher in clinically hirsute patients (p = 0.008).

**Discussion**

PCOS remains as a significant health burden due to the associated metabolic, psychological and reproductive complications. The current study has demonstrated that 76.7% of the study group had their weight in the over-weight or obese category according to the Asian BMI cut-offs [37]. Body fat percentage measurement on DEXA scan detected 50.0% as overweight and 45.8% as obese depicting the metabolic complications posed by the
PCOS as well the future risk of development of an array of non-communicable diseases. In a study done by Hes-tiantoro et al. body fat percentage is found to be better

Table 3  Study group characteristics

|                      | Minimum | Maximum | Mean | Standard. Deviation |
|----------------------|---------|---------|------|---------------------|
| Age                  | 18      | 44      | 26.7 | 6.7                 |
| Birth Weight (Kg)    | 1.5     | 5.1     | 2.8  | 0.65                |
| Pubarche age         | 9       | 18      | 11.9 | 1.7                 |
| Menarche age         | 9       | 18      | 12.2 | 1.7                 |
| Hirsutism (Ferriman Gallway Score) | 3     | 26      | 14.5 | 5.5                 |
| LH Level (IU/L)      | 1.1     | 23.1    | 10.2 | 5.8                 |
| FSH Level (IU/L)     | 1.1     | 16.9    | 5.9  | 2.5                 |
| Serum Total Testosterone (ng/dL) | 8.6  | 193     | 63.8 | 40.9                |
| FBS (mg/dL)          | 66      | 345     | 96.8 | 42.9                |
| PPBS (mg/dL)         | 77.8    | 172     | 103.8| 27.2                |
| HbA1C (mg/dL)        | 4.5     | 10.5    | 5.7  | 1.2                 |
| TC (mg/dL)           | 130     | 323     | 209.8| 50.5                |
| TG (mg/dL)           | 50      | 366     | 132.5| 67.3                |
| LDL-C (mg/dL)        | 62      | 141     | 133.7| 42.5                |
| AST (U/L)            | 11      | 214     | 35.5 | 37.2                |
| ALT (U/L)            | 10      | 148     | 38.2 | 31.8                |
| Serum fasting insulin (mIU/ml) | 3.8 | 44.9     | 14.6 | 10.0                |
| Fat % on whole body DEXA | 29.6  | 47.9    | 39.0 | 4.6                 |

Lab Reference ranges
LH (Adult females)- Follicular phase 1.9–12.5 IU/L, Midcycle phase 8.7–76.3 IU/L, Luteal phase 0.5–16.9 IU/L, Post menopausal 15.9–54 IU/L
FSH (Adult females)- Follicular phase 2.5–10.2 IU/L, Midcycle phase 3.4–33.4 IU/L, Luteal phase 1.5–9.1 IU/L, Post menopausal 23–116.3 IU/L
Total Testosterone 14–76 ng/dL.
FBS 70–110 mg/dL, PPBS < 140 mg/dL, HbA1c ≥ 6.5%- Diabetes, 5.7%-6.4%- Prediabetes, < 5.7%- Normal, TC < 200 mg/dL, TG < 150 mg/dL, LDL < 100 mg/dL, AST < 35 U/L, ALT < 35 U/L, Serum fasting insulin 2–20 mIU/ml

Table 4  Known association of PCOS

| Category                                 | Number | Percentage (3.1%) |
|------------------------------------------|--------|-------------------|
| Family history of PCOS                  | 13     | 21.7              |
| Epilepsy                                 | 0      | 0.0               |
| Maternal AMH administration in the 2nd trimester | 0  | 0.0               |
| Congenital adrenal hyperplasia (CAH)    | 1      | 1.7               |

Table 5  Diagnostic criteria for PCOS

| Criteria                                          | Number (%) | Number (%) |
|---------------------------------------------------|------------|------------|
| Clinical or biochemical hyperandrogenism          | 54 (90%)   | 53 (88.3%) |
| Hirsutism (Ferriman Gallway score ≥ 8)            |            |            |
| Elevated Serum Testosterone (≥ 76 ng/dL)          | 16 (26.6%) |            |
| Polycystic ovaries on USS                         | 24 (47%)   |            |
| Menstrual irregularity                            | 50 (83.3%) |            |

Fig. 2  Overlap of the Rotterdam criterion used to diagnose PCOS
marker for measuring inflammation in PCOS patients when compared to the BMI [38].

In a recent systemic review, a significantly higher proportion of dyslipidaemia, cardiovascular risk markers and metabolic syndrome was seen in patients with PCOS with higher BMI [39]. Nevertheless, the study populations has a low prevalence of chronic diseases such as diabetes, dyslipidaemia, hypertension, cardiovascular disease and cerebrovascular disease which can be explained by a relatively young age of the sample with a mean age of 26.7 (SD ± 6.7) and such analysis was not carried out. Majority of the study population has been educated up to or more than ordinary level indicating that the disease detection is higher in well-educated populations. In a study done in India the majority of PCOS fell in to the category of middle socio economic class [40]. Furthermore the majority (65%) were unmarried at the time of study which might indicate delays in marriage in these patients. Moreover, the study group had a mean FBG of 96.8 mg/dL (SD ± 42.9) and a HbA1c 5.7 (SD ± 1.2) which indicates the tendency towards development of frank diabetes. Furthermore, the diagnosis of PCOS increases insulin resistance contributed by the obesity associated with the disease [28]. The study group had a high prevalence of 61.1% of increased insulin resistance detected by the HOMA-IR assessment. Thus, intervening early to

| Category                                | Number (N = 60) | Percentage from the whole population (%) |
|-----------------------------------------|----------------|----------------------------------------|
| Diabetes mellitus                       | 4              | 6.7                                    |
| Dyslipidaemia                           | 4              | 6.7                                    |
| Hypertension                            | 3              | 5.0                                    |
| Cardiovascular disease                  | 0              | 0.0                                    |
| Cerebrovascular Disease                 | 0              | 0.0                                    |
| Venous thromboembolism                  | 0              | 0.0                                    |
| Mental Health disorders                 | 0              | 0.0                                    |
| Thyroid disease                         | Hypothyroidism | 10                                     | 16.6                                  |
|                                          | Hyperthyroidism| 3                                      | 5.0                                    |
| Endometrial cancer                      | 0              | 0.0                                    |
| Pregnancy complications                 | Miscarriage    | 2                                      | 3.3                                    |
|                                          | Preterm birth  | 1                                      | 1.7                                    |
|                                          | GDM            | 1                                      | 1.7                                    |
|                                          | PIH            | 0                                      | 0.0                                    |
| Non alcoholic fatty liver disease       | 4              | 6.7                                    |
| Fertility                               | Normal         | 11                                     | 18.3                                   |
|                                          | Sub-fertile    | 12                                     | 20.0                                   |
|                                          | Infertile      | 1                                      | 1.7                                    |
| Acanthosis nigricans                    |                | 28                                     | 46.6                                   |
| Fatty liver on USS                      |                | 14                                     | 23.3                                   |

| Asian BMI cut-offs | Underweight (n) | Normal weight (n) | Over weight (n) | Obese (n) | Significance (p value) |
|--------------------|-----------------|-------------------|----------------|----------|------------------------|
|                    | Complications of PCOS                                      |
| Acanthosis nigricans| 0.006                                                      |
| Yes                | 0               | 1                 | 4              | 23       |                        |
| No                 | 1               | 12                | 5              | 14       |                        |
| Fatty liver on USS | 0.005                                                      |
| Yes                | 0               | 0                 | 1              | 13       |                        |
| No                 | 1               | 13                | 8              | 15       |                        |
modify the risk factors can improve the future prospects of this relatively young group of individuals.

The commonest presentation of the disease is clinical or biochemical hyperandrogenism (90%) and menstrual irregularities (83.3%) in the study population as similar to the previous studies [4, 8]. Eighty percent of patients had a combination of above symptoms. Previous literature specifies acne as an uncommon clinical feature and was seen only in one third of the patients diagnosed with the disease [7]. The current study has demonstrated similar findings with only 26.7% patients suffering from acne. Nevertheless, high suspicion of the disease by the clinicians are paramount important as these are common clinical presentations of many other diseases.

The study has looked extensively in to the clinical, biochemical and radiological derangements that is associated with the disease. Although only 4 patients (6.7%) patients recruited to the study came up with a previously made diagnosis of dyslipidaemia, the study detected 18 patients (30.0%) to have a TC >200 mg/dL, while 26 patients (43.3%) had LDL >100 mg/dL indicating a higher prevalence of dyslipidaemia than previously diagnosed. The mean TC of the population is 209.8 mg/dL (SD ± 50.5) and the mean LDL is 132.5 mg/dL (SD ± 67.3) which is higher than the lab reference range. Furthermore, in the data collection, only 4 patients (6.7%) came up with a diagnosis of non-alcoholic fatty liver disease (NAFLD), while the study diagnosed NAFLD in 14 patients (23.3%). This corroborate the importance of detailed assessment of the patients diagnosed with PCOS in order to detect undiagnosed associated non communicable disease which might have disastrous complications in the future if not addressed early.

Reproductive complications associated with PCOS leads to numerous other problems including adverse psychological outcomes. The current study detected few patients to have had pregnancy complications while many more had concerns with subfertility/infertility (21.7%) consistent with the pathophysiology of the disease [14]. From the females who had or having fertility wishes fifty four percent had sub/infertility indicating the importance of addressing the fertility issues as well as the psychological effects arising from such complications of the disease.

The current study detected a significant association between high BMI categories and the presence of Acanthosis nigrians which is an indicator of insulin resistance and the presence of fatty liver on ultra sound scanning similar to the findings obtained in the recent systemic review [39]. Thus, addressing overweight and obesity might in turn will improve the detrimental metabolic parameters which will improve the long term outcome of PCOS patients.

The current study has several limitations. As this is a single centre study, the population may not represent the general population in the country which involve urban, sub-urban and rural areas. Furthermore, even though the study has extensively looked at clinical, biochemical, radiological features of the disease, we have not assessed the psychological impact of the diseases to the study participants. Moreover, as the study is a cross sectional study, follow up for the participant was not carried out to assess the response to the lifestyle and medical interventions that patients has undergone as a part of the routine management of the disease.

Conclusions
The study detected the majority of the study population to be overweight and obese according to the BMI cut-offs as well as body fat percentage detected by whole body DEXA scan which indicates an alarmingly high incidence of metabolic complications occurrence in PCOS patients in the future. The most common manifestations of the disease includes the presence of clinical or biochemical evidence of hyperandrogenism and irregular menstrual cycles in contrast to the detection of polycystic ovaries on trans-vaginal USS which indicates the importance of clinical suspicion and clinical diagnosis of the disease. The higher BMI is associated with the presence of acanthosis nigrians and fatty liver which mark the importance of weight reduction in bringing down the insulin resistance and the development of fatty liver in patients with PCOS. The higher prevalence of non-communicable diseases in this group of young females marks the importance of the early diagnosis and management of not only the PCOS itself but also the associated metabolic, reproductive and psychological complications of the primary disease. Successful management of the disease will eventually ease the future health expenditure strain of the country. Future studies including multi-centre, prospective studies will contribute to the understanding of the clinical features and productive management of the disease.

Abbreviations
PCOS: Polycystic ovary syndrome; NIH/NICHD: National Institutes of Health/National Institute of Child Health and Human Disease; ESHRE/ASRM: European Society for Human Reproduction and Embryology/American Society for Reproductive Medicine (ESHRE/ASRM); BMI: Body mass index; FPG: Fasting plasma glucose; PPBG: Post prandial blood glucose; OGTT: Oral glucose tolerance test; HbA1C: Glycated haemoglobin; TG: Triglycerides; HDL-C: High density lipoprotein-cholesterol; LDL-C: Low density lipoprotein-cholesterol; LH: Luteinizing hormone; FSH: Follicular stimulating hormone; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; DEXA: Dual-energy x-ray absorptiometry; AMH: Anti mullerian hormone; CAH: Congenital adrenal hyperplasia; USS: Ultrasound scan; NAFLD: Non-alcoholic fatty liver disease.
Acknowledgements
None

Authors’ contributions
IR, MS and NPS designed the study and were involved in data collection, statistical analysis, interpretation of data and drafting the manuscript. TGA and IR were involved in data collection, data entry and data analysis. All authors read and approved the final manuscript.

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Funding
Not funded.

Availability of data and materials
The data analyzed in this paper can be made available to researchers.

Requests for access to the dataset used in this paper should be directed to the corresponding author.

Competing interests
None of the authors have any financial or non-financial competing interests to disclose.

Consent for publication
Not applicable.

Received: 7 August 2022. Accepted: 9 November 2022
Published online: 21 November 2022

References
1. Bozdog G, Mimusoglu S, Zengin D, Karabulut E, Yildiz BO. The prevalence and phenotypic features of polycystic ovary syndrome: a systematic review and meta-analysis. Hum Reprod. 2016;31(12):2841–55.
2. Aziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. J Clin Endocrinol Metab. 2004;89(6):2745–9.
3. Ansúnez M, Calvo RM, San Millán JL, Sancho J, Avila S, Escobar-Morreale HF. A prospective study of the prevalence of the polycystic ovary syndrome in unselected Caucasian women from Spain. J Clin Endocrinol Metab. 2000;85(7):2344–8.
4. Farquhar C. Introduction and history of polycystic ovary syndrome. In: Kovacs G, Norman R, editors. Polycystic Ovary Syndrome. 2nd ed. Cambridge, UK: Cambridge University Press; 2007. p. 4–24.
5. Balian AH, Conway GS, Kaltzas G, Techasaisak K, Manning PJ, West C, et al. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod. 1995;10(8):2107–11.
6. Hart R, Hickey M, Franks S, Definitions, prevalence and symptoms of polycystic ovaries and polycystic ovary syndrome. Best Pract Res Clin Obstet Gynaecol. 2004;18(5):671–83.
7. Aziz R, Sanchez LA, Knochenhauer ES, Moran C, Lazenby J, Stephens KC, et al. Androgen excess in women: experience with over 1000 consecutive patients. J Clin Endocrinol Metab. 2004;89(2):453–62.
8. Fauser BJC, Tarlatzis BC, Rebar RW, Legro RS, Balian AH, Lobo R, et al. Consensus on women’s health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESPRE/ASRM-Sponsored 3rd PCOS consensus workshop group. Fertil Steril. 2012;97(1):28–38.e25.
9. Ferriman D, Gallwey JD. Clinical assessment of body hair growth in women. J Clin Endocrinol Metab. 1961;21:1440–7.
10. Adams J, Polson DW, Franks S. Prevalence of polycystic ovaries in women with anovulation and idiopathic hirsutism. Br Med J (Clin Res Ed). 1986;293(6543):355–9. Available from: https://pubmed.ncbi.nlm.nih.gov/3089520/
11. Souter I, Sanchez LA, Perez M, Bartolucci AA, Aziz R. The prevalence of androgen excess among patients with minimal unwanted hair growth. Am J Obstet Gynecol. 2004;191(6):1914–20.
12. Wijeyaratne CN, Balian AH, Barth JH, Belchetz PE. Clinical manifestations and insulin resistance (IR) in polycystic ovary syndrome (PCOS) among South Asians and Caucasians: is there a difference? Clin Endocrinol (Oxf). 2002;57(3):343–50.
13. Eden JA. The polycystic ovary syndrome presenting as resistant acne successfully treated with cyproterone acetate. Med J Aust. 1991;155(10):677–80.
14. Teede H, Deeks A, Moran L. Polycystic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. BMC Med. 2010;8(1):41. https://doi.org/10.1186/1741-7015-8-41.
15. Chang RJ, Cook-Andersen H. Disordered follicle development. Mol Cell Endocrinol. 2013;373(1–2):51–60.
16. Glueck CJ, Phillips H, Cameron D, Sieve-Smith L, Wang P. Continuing metformin throughout pregnancy in women with polycystic ovary syndrome appears to safely reduce first-trimester spontaneous abortion: a pilot study. Fertil Steril. 2001;75(1):46–52.
17. DA Zawadzki JK. Diagnostic criteria for polycystic ovary syndrome. In: Givens JH, Merriman G, editors. The Polycystic Ovary Syndrome. Cambridge, MA: Blackwell Scientific; 1992. p. 377–84.
18. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril. 2004;81(1):19–25. https://doi.org/10.1016/j.fertnstert.2003.10.004.
19. Aziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al. Positions statement: criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an androgen excess society guideline. J Clin Endocrinol Metab. 2006;91(11):4237–45.
20. Franks S, Gharani N, Waterworth D, Batty S, Williamson R, et al. The genetic basis of polycystic ovary syndrome. Hum Reprod. 1997;12(2):2641–8.
21. Amato P, Simpson JL. The genetics of polycystic ovary syndrome. Best Pract Res Clin Obstet Gynaecol. 2004;18(S5):707–18.
22. Vink JM, Sadrzadeh S, Lambalk CB, Boomsma DI. Heritability of polycystic ovary syndrome in a Dutch twin-family study. J Clin Endocrinol Metab. 2006;91(6):2100–4.
23. Salehi M, Bravo-Vera R, Sheikh A, Gouller A, Poretsky L. Pathogenesis of polycystic ovary syndrome: what is the role of obesity? Metab Clin Exp. 2004;53:358–76.
24. Moran LJ, Hutchinson SK, Norman RJ, Teede HJ. Lifestyle changes in women with polycystic ovary syndrome. Cochrane database Syst Rev. 2011;(7):CD007506. https://doi.org/10.1002/14651858.cd007506.pub3.
25. Codner E, Soto N, Lopez B, Trejo L, Avila A, Eyzaguirre FC, et al. Diagnostic criteria for polycystic ovary syndrome and ovarian morphology in women with type 1 diabetes mellitus. J Clin Endocrinol Metab. 2006;91(6):2250–6.
26. Peppard HR, Marfori J, Iuorno MJ, Nestler JE. Prevalence of polycystic ovary syndrome among premenopausal women with type 2 diabetes. Diabetes Care. 2001;24(6):1050–2.
27. Kashanian M, Fazy Z, Pirak A. Evaluation of the relationship between gestational diabetes and a history of polycystic ovarian syndrome. Diabetes Res Clin Pract. 2008;80(2):289–92.
28. Dunaif A, Segal KR, Futterweit W, Dobrjansky A. Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. Diabetes. 1989;38(9):1165–74.
29. Herzog AG. Menstrual disorders in women with epilepsy. Neurology. 2006;66(Suppl 3):523–8.
30. Rosenfield RL. Clinical review: Identifying children at risk for polycystic ovary syndrome. J Clin Endocrinol Metab. 2007;92(3):787–96.
31. Tsouma I, Koukouni E, Demeridou S, Boutisiku M, Hassakios D, Chaisakou A, et al. Lipid lipoprotein profile alterations in Greek infertile women.
with polycystic ovaries: influence of adipocytokines levels. In Vivo (Brooklyn). 2014;28(5):935–9.
32. Lo JC, Feigenbaum SL, Yang J, Pressman AR, Selby JV, Go AS. Epidemiology and adverse cardiovascular risk profile of diagnosed polycystic ovary syndrome. J Clin Endocrinol Metab. 2006;91(4):1357–63.
33. Wild S, Pierpoint T, McKieuge P, Jacob H. Cardiovascular disease in women with polycystic ovary syndrome at long-term follow-up: a retrospective cohort study. Clin Endocrinol (Oxf). 2000;52(5):595–600.
34. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412–9.
35. American Diabetes Association. Classification and diagnosis of diabetes. Diabetes Care. 2017;40(Suppl 1):S11-24.
36. Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr. 2000;72(3):694–701.
37. WHO Expert Cotationnul. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet (London, England). 2004;363(9403):157–63.
38. Hestiantoro A, Kapnosa Hasani RD, Shadrina A, Situmorang H, Ilma N, Muharam R, et al. Body fat percentage is a better marker than body mass index for determining inflammation status in polycystic ovary syndrome. Int J Reprod Biomed. 2018;16(10):623–8.
39. Gilbert EW, Tay CT, Hiam DS, Teede HJ, Moran L. Comorbidities and complications of polycystic ovary syndrome: An overview of systematic reviews. Clin Endocrinol (Oxf). 2018;89(6):683–99. https://doi.org/10.1111/cen.13828.
40. Mangalath AAM, Alias A, Sajith M, Nimbargi V, Kumdale S. Sociodemographic characteristics and clinical presentation of infertile women with polycystic ovary syndrome in a tertiary care hospital. Int J Infertil Fetal Med. 2018;9(1):14–8.

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