

Implementing Neural Fuzzy Rough Set and Artificial Neural Network for Predicting PCOS

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Abstract—Polycystic ovarian syndrome (or polycystic ovary syndrome – PCOS) is a multifarious form in which a woman's ovaries are normally larger than standard. The term 'Polycystic' defines that the ovaries comprise of numerous cysts or follicles to facilitate hardly ever nurture towards ripeness or generate eggs accomplished of being fertilized. One third of women could contain polycystic ovaries observed on an ultrasound, however it does not all have PCOS. PCOS is comparatively universal, especially for sterile women. It concerns about 12 to 18 per cent of women of reproductive age (between late adolescence and menopause). In approximate 70 per cent of this kind of cases remain undiagnosed. In our previous researches, we have proposed a new feature selection technique and hybrid approach and in this present investigation, we implement these proposed algorithms to forecast the PCOS disease among women. In addition to above analysis, we evaluate the effect of the proposed algorithms with other existing methods.

Keywords-PCOS, ANN, NFRS, J48, ID3

I. INTRODUCTION

Polycystic Ovarian Syndrome (PCOS) have an effect on 4 to 12% of women are from reproductive age [1]. In the 1935, Stein and Leventhal primarily depicted the connection of polycystic ovaries, amenorrhea, hirsutism, and obesity. The major characteristics are essential for the identification of PCOS were comprehensive at the conference convened by the National Institute of Health in 1990 moreover, they were menstrual dysfunction and hyperandrogenism, through segregation of other major reasons of hyperandrogenism (congenital adrenal hyperplasia, androgen-secreting tumors, and hyperprolactinemia). The probable criteria included for perimenarchal onset, insulin resistance, elevated leutenizing hormone to follicle-stimulating hormone ratio and polycystic ovaries by ultrasonography (USG) [1].

PCOS was reclassified at a joint consensus meeting of the European Society of Human Reproduction and Embryology (ESHRE) and the American Society of Reproductive Medicine (ASRM), held in Rotterdam in may 2003. And it enclosed the existence of two of the following three major criterias: (a) oligo and/or anovulation, (b) polycystic ovaries on USG and (c) hyperandrogenism (clinical and/or biochemical), with the exclusion of other etiologies. The morphology of the polycystic ovary has been redefined as an ovary with 12 or more follicles measuring 2–9 mm in diameter and/or increased ovarian volume (more than 10 cm³) [2].

Polycystic ovaries are frequently observed in healthy women however it is more common in women with irregular cycles and hyperandrogenism. The polycystic form of the ovary is the distinctive sign of polycystic ovary syndrome

(PCOS) however there is a wide range of clinical and biochemical characteristics like e.g. elevated serum concentrations of androgens, insulin, LH and decreased insulin sensitivity. These conditions are frequently associated with obesity. Since insulin resistance in PCOS patients is predominantly extra-splanchnic [3] (Dunaif et al., 1992), the fasting blood sugar is normal. According to another group [4] (Franks et al., 1997), ovarian morphology is the vital sign of the syndrome and the wide range of associated phenotypes can be elucidated through the interaction of a small quantity of key genes with ecological features.

Since these symptoms are found in up to 10% of young women, PCOS is certainly the most frequent endocrine disorder diagnosed in these subjects. Despite the high prevalence of isolated polycystic ovarian morphology (22%), the syndrome may be accompanied by minimal clinical manifestations and, in particular, no uniformly deleterious effect on fertility has been reported [5] (Clayton et al., 1992). A controlled comparative study of patients undergoing an IVF programme found no significant difference in pregnancy and live birth rates between women with and without polycystic ovaries [6] (MacDougall et al., 1993). Nevertheless, in a large group of PCOS patients, a high prevalence of primary (46%) and secondary (26%) infertility was found [7] (Balen et al., 1995), while another group [8] (Regan et al., 1990) found an elevated rate of miscarriages in patients with raised LH concentrations in which PCOS was inferred.

II. PCOS PHENOTYPES IN VARIOUS POPULATIONS

In view of the high prevalence of affected individuals, a genetic cause of the syndrome was suggested 30 years ago [9]

(Cooper et al., 1968). This has been investigated in several studies on PCOS phenotypes in different populations.

A. Ethnic Groups

PCOS women (n = 75) from three different ethnic groups were studied [10] (Carmina et al., 1992) and it was concluded that although obesity and hirsutism varied with genetic and environmental factors, the prevalence of adrenal androgen excess and insulin resistance appeared fairly uniform. More recent studies, however, found that ethnicity and PCOS were associated with independent and additive defects of insulin action in Caribbean±Hispanic women [3] (Dunaif et al., 1992). There were also differences in insulin and glucose responses to glucose provocation tests between white and Indian women with the syndrome [11] (Norman et al., 1995). Other authors [12] (Legro et al., 1999) suggest that PCOS is a more important risk factor than ethnicity or race for glucose intolerance in young women.

B. Twins

PCOS has been occasionally reported in identical twins [13] [14] (McDonough et al., 1972; Hutton and Clark, 1984). An Australian study found a 50% incidence of PCOS in 34 female twin pairs studied. The high degree of discordance in sonographic ovarian imaging between twins suggests a complex inheritance pathway and/or an important role of environmental factors in the genetic transmission mechanism [15] (Jahnfar et al., 1995). These authors suggest that the high prevalence of PCOS among twins may be explained by factors acting in prenatal life.

C. Families

Ovarian morphology, menstrual irregularities and signs of hyperandrogenism were the main symptoms investigated by studies on the familial clustering of PCOS cases, and premature balding was the male phenotype frequently found in the male relatives. All the studies showed a substantially increased risk of PCOS in first-degree female relatives of PCOS patients. Two additional studies on PCOS pedigrees [4] [16] (Franks et al., 1997; Govind et al., 1999), suggest the condition is passed down through either sex according to an autosomal dominant model of genetic transmission.

III. MECHANISM OF HERITABILITY

A. Ovarian Steroidogenesis

Although the secretion of androgens by the adrenal glands may be increased, the main source of androgen excess in PCOS is the ovary [17] (Franks et al., 1989). Oestrogen production may be linked either to abnormal stimulation of the ovary or to an intrinsic defect of ovarian steroidogenesis or increased extraovarian conversion of androgen in fat tissue.

B. Abnormal Ovarian Stimulation

Sensitive immunoassay systems have revealed the existence of microheterogeneity of human LH in a large population [18] [19] (Pettersson and Soderholm, 1991; Pettersson et al., 1992). Analysis of the structure of the LH β gene in women with immunologically anomalous LH revealed two nucleotide substitutions in codons 8. This common genetic variant induces higher LH bioactivity than the wild form [20] (Haavisto et al., 1995). While abnormal LH secretion may cause anovulation and luteal insufficiency, leading to PCOS, the frequency of LH mutations in women with PCOS is not different from that in healthy women, so the presence of the variant does not explain the abnormal steroidogenesis in polycystic ovaries [21] (Elter et al., 1999).

Follistatin binds to activin and affects its stimulatory activity on FSH secretion. A follistatin gene mutation in PCOS patients may play a role in the functional impairment of the FSH±granulosa cell axis. While evidence for such a link between the follistatin gene and PCOS has been found in a large study [22] (Urbanek et al., 1999), the association of PCOS with a polymorphism of the gene encoding follistatin was not confirmed by another recent study [23] (Liao et al., 2000).

C. Intrinsic Ovarian Defects

It is well known that there is a primary abnormality in the theca cells of PCOS patients leading to excessive production of progesterone and androgen [24] (Gilling-Smith et al., 1994, 1997). Therefore the abnormal steroidogenesis observed in PCOS is related to an intrinsic abnormality of the theca cells rather than to abnormal gonadotrophin stimulation [25] (Iban ez et al., 1996).

This finding prompted a study of the cholesterol side chain cleavage gene (CYP11a) as a possible cause of the deranged steroidogenesis. The segregation of CYP11a in 20 PCOS families was studied [26] (Gharani et al., 1997). The most common polymorphism of the gene (indicated as 216±) was significantly associated with PCOS families. A non-parametric linkage (NPL) analysis using polymorphic markers in that region similarly suggested that the steroid synthesis gene CYP11a is a very important locus for the genetic susceptibility of PCOS hyperandrogenism (NPL score 3.03, P = 0.003) [26] (Gharani et al., 1997).

D. Adrenal and Ovarian Hyperandrogenism

The increased ovarian and adrenal steroidogenic activity in PCOS can also be caused by enhanced lyase activity, exclusively by the cytochrome P450 C17a. Serine phosphorylation of this enzyme system selectively increases its enzymatic activity, leading to hypersecretion of ovarian and adrenal androgen, with no rise in adrenocorticotrophic hormone

(ACTH) or other steroidogenic activity [27] (Zhang et al., 1995).

E. Insulin Resistance

Insulin resistance is another common feature in women with PCOS. The cause is still unknown. Interestingly only women with an endocrine syndrome of hyperandrogenism and chronic anovulation appear to be insulin resistant and at high risk of glucose intolerance [28] [29] (Dunaif et al., 1987; Robinson et al., 1993).

There appears to be a genetic target cell defect as a cause of the metabolic condition [30] (Holte, 1996). The same hyperphosphorylation process described for cyto-chrome P450 C17 lyase activity, leading to adrenal and ovarian hyperandrogenism, has been implicated as the cause of a specific post-receptor defect of transduction of the insulin signal in fibroblasts [31] (Dunaif et al., 1995). In these patients, auto-phosphorylation of the serine (rather than tyrosine) residue impairs insulin signal transduction and contributes to the 50% insulin resistance observed. Thus a single molecular defect leading to the activation of a serine kinase might explain the two main biochemical disturbances in these patients: hirsutism and insulin resistance.

F. Abnormal Insulin Secretion

Hyperinsulinaemia has been reported in patients with PCOS and the syndrome is one of the major risk factors for non-insulin-dependent diabetes mellitus (NIDDM) [32] ((Holte et al., 1995). The b cell dysfunction is not obesity-dependent and in the majority of PCOS women is not associated with glucose intolerance [33] (Dunaif and Finegood, 1996).

The direct role of the insulin gene in the aetiology of hyperinsulinaemia was investigated in three groups of PCOS patients (one of which included 17 families with several affected individuals). All three populations showed an association between class III alleles at the variable number of tandem repeats (VNTR) 5¢ to the insulin gene and PCOS [34] (Bennett et al., 1995). The association was stronger in anovulatory patients, who more frequently have hyperinsulinaemia. A non-parametric linkage analysis in the PCOS families showed excess allele sharing at the same locus (NPL score 3.250, P = 0.002) [35] (Waterworth et al., 1997). The authors concluded that the VNTR 5¢ region to the insulin gene is a major locus for PCOS-associated hyperinsulinaemia.

IV. COMPARISON OF NEURAL FUZZY ROUGH SET WITH FEATURE SELECTION TECHNIQUES

In our previous research paper [36], we have proposed a feature selection algorithm called Neural Fuzzy Rough Set

(NFRS). In this paper, we are implementing this algorithm using MATLAB is used here to compare with the existing clinical reports of PCOS with different age group, Menstrual Cycle with obese and without obese, presence of clinical hyperandrogenism and absence of clinical hyperandrogenism. Here, first the proposed algorithm of feature selection called Neural Fuzzy Rough Set Evaluation with Correlation feature selection, Information gain, Principle Component Analysis according to the above test results using feature selection techniques.

TABLE 1 FEATURE SELECTION TECHNIQUES OF DIFFERENT AGE GROUP WITH OBESE PATIENTS RECORD

Different Age Group	Feature Selection Techniques			
	PCA	IG	Gain Ratio	Proposed Algorithm (NFRS)
<i>Total No of features Selection from 100 patients of each age category</i>				
Less than 20	50	61	45	20
21 – 25	67	70	55	22
26-30	71	68	59	30
35-35	48	69	63	21
36-40	53	45	66	18
More than 41	66	60	53	33

The above table 1 represents that each feature selection technique selects the number of attributes from total number of 100 attributes of each age group category. The records of dataset of the patients with obese of different age group. Depend upon the obese report of the each patient, the attributes are filtered. From the above table our proposed algorithm of NFRS gives the better less number of features than the other techniques.

TABLE 2 RESULT OF THE PATIENTS OF DIFFERENT MENSTRUAL CYCLE WITH OBESE USING FEATURE SELECTION TECHNIQUES

Type of Menstrual Cycle	Feature Selection Techniques			
	PCA	IG	Gain Ratio	NFRS
<i>Each Type have 100 patients records with Obese</i>				
Abnormal	75	72	76	50
Withdrawal	65	69	70	45
Bleed	73	70	66	30
Delayed	66	78	72	51
Early	69	66	63	32
Variable	78	69	77	35
Normal	70	63	69	22
Oligo-Ovulation	63	60	55	18
Normal-Ovulation	80	75	63	28

In this table 2 and table 3, the records of each different menstrual cycle with obese and without obese is given for selecting features using feature selection techniques and proposed technique of NFRS. The NFRS produces the less number of attributes than the other methods.

TABLE 3 FEATURE SELECTION RESULT OF THE PATIENTS OF DIFFERENT MENSTRUAL CYCLE WITHOUT OBESE USING FEATURE SELECTION TECHNIQUES

Type of Menstrual Cycle	Feature Selection Techniques			
	Each Type have 100 patients records without Obese			
	PCA	IG	Gain Ratio	NFRS
Abnormal	60	59	56	30
Withdrawal	70	66	63	25
Bleed	63	69	70	24
Delayed	71	75	72	40
Early	65	70	63	25
Variable	68	63	60	20
Normal	73	60	59	18
Oligo-Ovulation	63	58	51	26
Normal-Ovulation	70	66	52	19

TABLE 4 FEATURE SELECTION RESULT OF PATIENT WITH OBESE AND CLINICAL HYPERANDROGENISM USING DIFFERENT FEATURE SELECTION TECHNIQUES

Clinical Hyperandrogenism	Feature Selection Techniques			
	Each Type have 100 patients records with Obese			
	PCA	IG	Gain Ratio	NFRS
Hirustism	50	60	58	19
Acne & Oily skin	58	58	61	23
Hirustism, Acne & Oily Skin	64	54	71	26
Absence of Hyperandrogenism	60	63	65	28

From the table 4 and table 5, the patient with obese and clinical hyperandrogenism, the observation of different feature selection and proposed algorithm shows that proposed NFRS generates the less number of features than the other for predicting the severe PCOS with patients.

TABLE 5 FEATURE SELECTION RESULT OF PATIENT WITHOUT OBESE AND CLINICAL HYPERANDROGENISM USING DIFFERENT FEATURE SELECTION TECHNIQUES

Clinical Hyperandrogenism	Feature Selection Techniques			
	Each Type have 100 patients records without Obese			
	PCA	IG	Gain Ratio	NFRS
Hirustism	54	56	60	25
Acne & Oily skin	60	64	62	26
Hirustism, Acne & Oily Skin	62	63	61	20
Absence of Hyperandrogenism	50	54	53	18

V. COMPARISON OF HYBRID ALGORITHM USING NEURAL FUZZY ROUGH SET AND ARTIFICIAL NEURAL NETWORK

In our previous research paper [37], we have proposed a hybrid algorithm which correlates the Neural Fuzzy Rough Set

and Artificial Neural Network. It correlates the feature selection and classification techniques for reducing the feature selection and classification separately for predicting the PCOS women with different problems like different types of Menstrual cycle, and different age group of people.

TABLE 6 CLASSIFICATION ACCURACY OF THE PATIENTS WITH OBESE OF DIFFERENT AGE GROUPS USING CLASSIFICATION TECHNIQUES

Different Age Group	Classification Techniques			
	Classification Accuracy in (%)			
	ANN	J48	ID3	NFRS+ANN
Less than 20	80	75	72	85
21 – 25	72	78	79	80
26-30	69	72	76	81
35-35	82	73	80	88
36-40	65	70	66	75
More than 41	82	74	69	83

The table 6 represents the classification accuracy of the various classification techniques like Artificial Neural Network, J48 , ID3 and proposed algorithm of NFRS+ANN. The result shows that NFRS+ANN gives the best classification accuracy of the patient with obese of different age group.

TABLE 7 CLASSIFICATION ACCURACY OF THE PATIENTS WITH OBESE OF VARIOUS MENSTRUAL CYCLE USING CLASSIFICATION TECHNIQUES

Type of Menstrual Cycle	Classification Accuracy (in %)			
	Patient with Obese			
	PCA	IG	Gain Ratio	NFRS+ANN
Abnormal	78	75	70	80
Withdrawal	72	63	60	73
Bleed	70	66	64	75
Delayed	74	70	78	80
Early	71	68	69	79
Variable	69	65	93	78
Normal	80	72	76	85
Oligo-Ovulation	82	83	84	89
Normal-Ovulation	78	74	72	86

TABLE 8 CLASSIFICATION ACCURACY OF THE PATIENTS WITHOUT OBESE OF VARIOUS MENSTRUAL CYCLE USING CLASSIFICATION TECHNIQUES

Type of Menstrual Cycle	Classification Accuracy (in %)			
	Patient without Obese			
	ANN	J48	ID4	NFRS+ANN

Abnormal	69	63	60	73
Withdrawal	70	65	64	75
Bleed	71	66	69	78
Delayed	72	68	70	79
Early	74	70	72	80
Variable	78	72	76	80
Normal	78	74	78	85
Oligo-Ovulation	80	75	84	86
Normal-Ovulation	82	83	78	89

From the table 7 and table 8, the proposed technique of NFRS and ANN gives the best accuracy result of classification with patient who are affect with obese and without obese who are possessing different menstrual cycle.

VI. CONCLUSION

From the above tables, we can say that the proposed algorithms of Neural Fuzzy Rough Set feature selection and correlation of NFRS and artificial neural network gives the better result when it is compared with various feature selection and classification technique with the records of people who are affect with obese and without obese when it calculating with different age group, hyperandrogenism and types of menstrual cycle.

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