Introduction

Polycystic ovarian syndrome

Polycystic ovary syndrome (PCOS) was first described by Stein and Leventhal syndrome in 1935[1]. Polycystic ovary syndrome (PCOS) occurs in 4 to 8% of women in their reproductive years and is a most frequent endocrine disease[2]. PCOS is found approximately one in 15 women [3]. The combination of PCOS and amenorrhoea were the clinical features of the syndrome beside this it is a heterogenous disease.

PCOS was accepted to be an alternative to ovarian dysfunction with the presence of hyperandrogenism, according to Androgen excess society (AES) criteria; combination of anovulation and polycystic ovaries without hyperandrogenism was not accepted as PCOS[4].

PCOS is a condition in which woman has an imbalance of female sex hormone which may lead to changes in the menstrual cycle, cyst in the ovaries, trouble getting pregnant and other health problems[5]. PCOS is hormonal level changes or disbalances which make the ovaries difficult to release fully grown (mature) eggs. The reasons for these changes are unclear.

The hormones affected are[6] Estrogen and Progestrone, the female hormones that help a woman ovaries release eggs. Androgen a male hormone that is found in small amounts in women. Normally one or more egg are released during a woman’s cycle. This is known as ovulation. In PCOS mature egg are not released. Instead they remain in ovaries with a small amount of fluid around them.

PCOS is being considered as an ovarian dysfunction caused by male hormone androgen which inhibit folliculogenesis and lead to polyfollicular morphology and disturbs menstrual cycle and lead to anovulation[7]. Hypersecretion of androgen is most common biochemical feature in pcos women[8].

PCOS accounts for 70-80% of hyperandrogenism and there is elevation of serum total or free testosterone concentration[9]. Hyperandrogenism leads to hirusitism, acne and male pattern baldness.

PCOS is present in 60-90% of women with hirusitism[10,11,12] as increased androgen production leads to hirusitism.
ADIPONECTIN AND PCOS

Adiponectin is a 244-amino-acid-long polypeptide protein. A protein hormone which is called Adiponectin regulates a metabolic process like glucose regulation and fatty acid regulation.

Adiponectin is a protein hormone which is being secreted from adipose tissue. This hormone is present in plasma in adequate amount and is also being secreted from the placenta during the pregnancy period.[13]

The role of adipose tissue hormones adiponectin has been playing a major role in the pathogenesis of PCOS. Adiponectin has antiatherogenic, anti-diabetic, anti-inflammatory and insulin sensitizing effects, and is negatively related to the degree of adiposity in healthy individuals.

Adiponectin level is low in Pcos patients.

LEPTIN AND PCOS

Leptin is a new hormone "leptin" from the Greek lepto meaning thin was given by Friedman

In 1995 Several studies came to the conclusion that the db gene encodes the leptin receptor which is being expressed in the hypothalamus a region of brain that regulate the sensation of hunger and body weight.[14]

There is a strong correlation of Circulating leptin with obesity, which is ultimately associated with polycystic ovarian syndrome (PCOS), a major form of dysovulatory infertility in women. Leptin is important in regulating energy homeostasis, an impacts the reproductive systems in diverse ways.[15] Polycystic ovarian syndrome, the common dysovulatory infertility, is characterized by chronic anovulation, hyperandrogenemia, insulin resistance, and a high incidence of obesity; features which are often linked to leptin and its receptor

These facts thus make PCOS women a useful tool to assess the inter-regulatory phenomena between leptin and ovarian function. There is a results of positive correlation exists between serum leptin and PCOS[16].
Oxidative stress is an unbalanced system between the systemic manifestation of reactive oxygen species and a biological system ability to detoxify the reactive intermediates or to repair the resulting damage. Disturbances in the normal redox state of cells can cause toxic effects through the production of peroxides and free radicals that damage all components of the cell, including proteins, lipids, and DNA[17,18].

Oxidative stress may be "a state where oxidative forces exceed the antioxidant systems due to loss of the balance between them." Oxidative stress is associated with increased production of oxidising species or a significant decrease in antioxidant defences such as glutathione. There is a production of reactive oxygen species which is a quiet toxic part of oxidative stress. Some of the less reactive of these species (such as superoxide) can be convert by oxidoreductioreactions with transition metals or other redox cycling. Most long-term effects are caused by damage to DNA.[19,20] DNA damage induced by ionizing radiation is similar to oxidative stress.

Extensive attention is given to Oxidative stress (OS) in the last two decades, because of the discovery that abnormal oxidation status was related to patients with chronic diseases, such as cardiovascular, polycystic ovary syndrome (PCOS). OS is considered as a potential inducing factor in the pathogenesis of PCOS, which is one of the most common complex endocrine disorders and a leading cause of female infertility, affecting 4%-12% of women in the world, as OS has close interactions with PCOS characteristics, just as insulin resistance (IR), hyperandrogenemia, and chronic inflammation.

OS level is significantly increased in patients with PCOS compared with the normal, when oxidative status is evaluated by circulating markers, such as malondialdehyde (MDA), superoxide dismutase (SOD).[21]

MALONDIALDEHYDE(MDA)

Malondialdehyde (MDA) is the organic compound. It is a colourless liquid. Malondialdehyde actually occurs naturally and it is not seen in pure form. It is a highly reactive compound.

Malondialdehyde (MDA) is one of the major aldehyde derived from lipid peroxidation[22]. This is a biomarker that is being used to assess oxidative stress because the accurate measurement of such stress is necessary for investigation of its role in lifestyle diseases as well as to evaluate the efficacy of treatment.

Lipid peroxidation process revealing malyondialdehyde (MDA) is accepted to reflect oxidative stress [22] and MDA is increased in PCOS.
**SUPEROXIDE DISMUTASE (SOD)**

Superoxide dismutase (SOD) is an enzyme which catalyzes the superoxide radical into the dismutation (or partitioning) into either ordinary molecular oxygen (O₂) or hydrogen peroxide (H₂O₂).

- Superoxide is produced as a by-product of oxygen metabolism and, if not regulated, causes many types of cell damage [23].
- SOD is an important antioxidant enzyme serving as a defence mechanism of the body.
- SOD induces the conversion of superoxide to H₂O₂, a toxic substance that is converted to water by GPx.
- High SOD levels may explain the absence of endothelial dysfunction markers. There may be chances of an adequate antioxidant response against such an intrinsic oxidative load may provide proper functioning of vascular system.
- SOD levels were significantly higher in a PCOS group [24].