

Figure 2: Ascatter plot shows correlation between Body Mass Index (IBM) and glucose in the study group($r = 0.02$, $p = 050$)

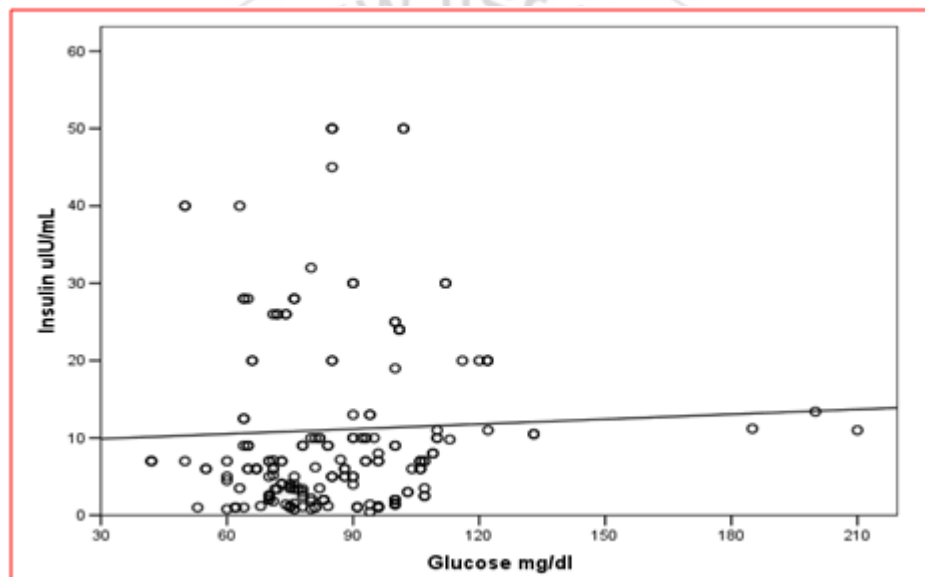


Figure 3: Ascatter plot shows correlation between insulin and glucose in the study group($r = 0.04$, $p = 060$)

4. Discussion

It has long been recognized that syndromes characterized by extreme insulin resistance are associated with ovarian hyperandrogenism. In the past decade, however, attention has focused on women who present with the polycystic ovary syndrome rather than on those with the typical phenotype of syndromes involving insulin resistance. Women with the polycystic ovary syndrome have a greater frequency and degree of both hyperinsulinemia [19, 20], and insulin resistance [21, 22], than weight-matched controls. Insulin resistance is independent of the effect of obesity; both lean and obese women with the polycystic ovary syndrome have evidence of decreased insulin sensitivity, but insulin resistance is most marked where there is an interaction between obesity and the syndrome [23, 24]. In our study hyperinsulinemia was observed according to data of statistic analysis there was significant increased Serum insulin level in patients with PCOS when compared to the control group. This agrees with a study done by Burghen, et

al [25], in 1980 who reported that there was association of PCOS with hyperinsulinemia. It has become clear that the syndrome has major metabolic as well as reproductive morbidities. Hyperinsulinemia caused higher level of androgen which is one feature of PCOS. Hyperinsulinemia in women with the polycystic ovary syndrome appears to reflect the hypersecretion of insulin itself, rather than of proinsulin and its split products. [26]. The cellular mechanism of insulin resistance in the polycystic ovary syndrome remains controversial. Results from studies of blood cells have suggested reduced binding of insulin to its receptor [27], whereas two recent studies [28], using peripheral adipocytes (recognized target cells for insulin action) have shown normal binding but reduced insulin-mediated glucose transport, suggesting a post receptor defect. This hyperinsulinemia may also lead to impaired lipolysis in adipocytes, which in turn may contribute to obesity often seen in PCOS patients [29, 30]. Present study shown insignificant and very weak positive correlation between the body mass index and insulin level in PCOS Patients (figure 1), 36.6% were obese (BMI > 30 Kg/m²)

The presence of hyperinsulinemia in PCOS women, independent of obesity, was confirmed by a number of groups worldwide [31].

In the present study, fasting plasma glucose levels in patients with PCOS significantly increased as compared to the control subjects (table 1), this result agrees with a study done by Burghen, et al[25], who reported that the elevation of fasting plasma glucose was associated with hyperinsulinemia also Kierland, et al[32], reported that there was insulin-resistant diabetes mellitus in patients with PCOS. The practical implication of these findings is that the polycystic ovary syndrome may be a marker of insulin resistance and dyslipidemia[33,34] Impaired glucose tolerance and frank type II diabetes mellitus are more prevalent in obese young women with the polycystic ovary syndrome than in weight-matched controls[19,22]. Recently published long-term follow-up studies of women with the syndrome show that the prevalence of type II diabetes is seven times higher in that group than in the reference population[35]. These women have hyperlipidemia and a greatly increased risk of cardiovascular disease[36].

The study showed insignificant and weak positive correlation between the body mass index and the plasma glucose levels (figure 2). Figure 3 showed a positive correlation between fasting insulin and fasting glucose level among PCOS patients. This is due to metabolic abnormalities caused by hyperinsulinemia

5. Conclusion

The current study demonstrated that the, PCOS causes significant increases of serum insulin and Plasma glucose in women and high body mass index. Weight reduction in obese women with the polycystic ovary syndrome should be encouraged [37] in an effort to limit the risk of hyperinsulinemia, type II diabetes and long-term cardiovascular disease. More investigations should be done to demonstrate the relationship between hyperinsulinemia, elevated glucose level, and insulin resistance obesity in PCOS patients especially glucose intolerance test, glucose insulin ratio, glucose clamp and other tests addition to the elevated BMI increases the risk for IR, IGT, and DM. Aggressive lifestyle interventions should be a priority in the

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