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Hyperprolactinemia alters insulin resistance profiles in overweight males

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Objective. Elevated prolactin levels (hyperprolactinemia) is an endocrine disorder associated with metabolic dysfunctions such as insulin resistance, obesity, dyslipidemia, and glucose intolerance on many occasions. Type 2 diabetes mellitus (T2DM) is a major risk factor for insulin resistance, which is one of the most important determinants of overweight males for hyperprolactinemia. The purpose of this study was to investigate the impact of hyperprolactinemia on insulin resistance parameters including HOMA-IR, QUICKI, TG/HDL, and TyG index values in overweight male patients.

Patients and Methods. A total of 90 participants were involved in a case-control study including 45 overweight males with hyperprolactinemia and 45 age- and BMI-matched healthy controls. Blood samples were collected for measurement of fasting glucose, insulin, lipid profile, testosterone, and prolactin. HOMA-IR, QUICKI, TyG index, TG/HDL ratio were used to assess insulin resistance.

Results. In hyperprolactinemic individuals, fasting insulin levels, HOMA-IR, and the TyG index were elevated, which indicates increased insulin resistance. In addition, these patients had lower QUICKI values corresponding to lower insulin sensitivity. This was also related to the fact that there was a significant elevation in triglycerides and LDL levels as well as a decrease in HDL. Testosterone was turned negatively with high prolactin level ($p<0.01$), followed by insulin resistance markers, which were positively correlated with increased prolactin ($p<0.001$).

Conclusions. The results suggest that hyperprolactinemia has a strong association with insulin resistance in overweight males. The elevated prolactin levels found in testosterone deficiency also led to a dysregulated glucose metabolism and lipid abnormalities, and hormonal imbalance underlying the rationale for early screening and metabolic interventions.

Keywords: hyperprolactinemia, insulin resistance, HOMA-IR, obesity, prolactin

Hyperprolactinemia (HP), which is caused by an overproduction of prolactin (PRL) by any means (i.e. pituitary adenoma, hypothalamic dysfunction, medications and primary hypothyroidism) is a common endocrine disorder (Haidenberg et al. 2024). Hyperprolactinemia is usually linked with reproductive dysfunction, but recent evidence is pointing to strong associations of hyperprolactinemia symptoms with metabolic disease such as insulin resistance, obesity, and dyslipidemia (Parak et al. 2020; Faron-Gorecka et al. 2023; Wang et al. 2023).

Insulin resistance is strongly linked with obesity, where cells lose the ability to respond to insulin resulting in hyperglycemia and an increased chance

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of developing type 2 diabetes mellitus T2DM (Matikainen et al. 2021; Roden et al. 2024). Metabolic syndrome, including hypertension, dyslipidemia, and central obesity that greatly increases cardiovascular disease (CVD) risk is also linked to insulin resistance (Lombardi et al. 2020).

Prolactin regulates insulin secretion and β cell proliferation in pancreatic β cells, which express prolactin receptors (PRLRs) (Alexander and Dimke 2023). Chronic hyperprolactinemia, however, impairs insulin signaling and glucose uptake, lipid metabolism, and inflammatory regulation (Han et al. 2021). Hyperprolactinemia has been shown to increase insulin resistance by inhibiting insulin receptor signaling pathways (Gierach et al. 2022), decreasing insulin sensitivity in muscle and liver cells (Pirchio et al. 2022) and exacerbating the lipid metabolism disorders (Wang et al. 2023).

Testosterone is important for glucose metabolism, muscle mass, and lipid regulation (Trost 2021). Low testosterone levels in males are often correlated with hyperprolactinemia, which is associated with higher metabolic disease risk on worsening insulin resistance (Szulc 2022). There are studies showing that prolactin-induced hypogonadism deteriorates insulin resistance through reduced androgen-mediated glucose uptake (Flores et al. 2022).

Nevertheless, the relationship between hyperprolactinemia and insulin resistance in overweight men is under research. This study aims to demonstrate whether hyperprolactinemia affects the insulin resistance markers, lipid profile, and hormonal imbalances and thus what are the metabolic consequences of the hyperprolactinemia.

Materials and Methods

Study design and participants. This was a case-control study performed in cooperation with Medical Biochemistry and Molecular Biology Department, Faculty of Medicine, Benha University and Biochemistry Division, Faculty of Science, Menoufia University, Egypt.

A total of 90 overweight male participants, selected from various obesity clinics at Benha University Hospital between February and July 2023, were included in the study. They were divided into two groups: 1) hyperprolactinemia group (n=45): overweight males (100%) (body mass index, BMI >35 kg/m^2) with hyperprolactinemia diagnosed, their age ranged from 23.0 to 39.0 years with a mean of 31.00±2.26 years (mean±SE), BMI index ranged from 35.3 to 45.3 kg/m^2 with a mean of 37.87±1.34 kg/m^2; and 2) control group (n=45): BMI-matched healthy males (100%) having normal prolactin levels, their age ranged from 20.0 to 38.0 years with a mean of 28.29±2.20 years, BMI index ranged from 23.9 to 24.9 kg/m^2 with a mean of 24.40±0.13 kg/m^2.

Inclusion and exclusion criteria. Inclusion criteria were: males at the age from 18 to 40 years, BMI more than 35 kg/m^2, diagnosed with hyperprolactinemia (PRL >25 ng/mL). Exclusion criteria were: patients with pituitary adenomas (prolactinoma), patients in whom prolactin concentration results ranged slightly above the normal range, patients with high prolactin level, but BMI below 35 kg/m^2, individuals with thyroid-stimulating hormone (TSH) abnormalities or primary hypothyroidism, patients with secondary obesity (due to endocrine disorders or medications), and smoking, alcohol, or use of medications with effects on prolactin or glucose metabolism.

The protocol was approved by the Ethical Committee of Medical Research, Faculty of Medicine, Benha University.

Biochemical analysis. Following a 10-h fast, blood samples were obtained by vein puncture into vacutainer tubes centrifuged at 3000 rpm and then kept at –20 °C.

Parameters measured. Prolactin (ng/mL) levels were determined by the procedure described previously by Loraine and Bell (1971), total testosterone (ng/dL) was measured using a commercial ELFA (Enzyme Linked Fluorescent Assay) kit (BIOMERIEUX, SA, Marcy-l'Etoile, France) (Yun et al. 2012). Fasting glucose (mg/dL), fasting insulin (μIU/mL) and lipid profile (mg/dL) were measured using method described by Burtis and Bruns (2014) and commercial kits (SPINREACT, Barcelona, Spain).

Assessment of insulin resistance. The following equations were used to calculate Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), Quantitative Insulin Sensitivity Check Index (QUICKI), Triglyceride-Glucose (TyG) index, and TG/HDL ratio:

$$\text{HOMA} - \text{IR} = \frac{\text{Fasting Insulin (μIU/mL)} \times \text{Fasting Glucose (mg/dL)}}{405}$$

$$\text{QUICKI} = \frac{1}{\log(\text{Fasting Insulin}) + \log(\text{Fasting Glucose})}$$

$$\text{TyG} = \log\left(\frac{(\text{Triglycerides (mg/dL)} \times \text{Fasting Glucose (mg/dL)})}{2}\right)$$

$$\text{TG/HDL} = \frac{\text{Triglycerides (mg/dL)}}{\text{HDL (mg/dL)}}$$

Statistical analysis. The obtained results were tabulated and processed then using IBM using SPSS 20 statistical package to analysis. Paired t-test is an

evaluation tool for testing correlation between two related quantitative variables (Calinski et al. 1981). The interrelationships between prolactin, hormones, lipids, and markers of insulin resistance were measured using Pearson correlation coefficients.

Results

Baseline characteristics of study participants. Table 1 summarizes the baseline demographic and biochemical characteristics of participants. There was no statistically significant difference ($p>0.05$) in the age of the participants between the hyperprolactinemia group and the control group. Moreover, the individuals within the hyperprolactinemia group had significantly increased BMI, fasting insulin, fasting glucose, and lipid abnormalities compared to the control group (Table 1).

Prolactin levels and insulin resistance markers. Statistically significant differences were observed between groups in the monitored indicators of insulin resistance, namely hyperprolactinemic patients had significantly higher HOMA-IR (3.19±0.16) compared to controls (0.99±0.14) ($p<0.001$) and significantly lower QUICKI (0.32±0.00 vs. 0.55±0.03, $p<0.001$) (Table 1). The magnitude of these p values suggests the robustness of these findings, as hyperprolactinemic patients are very insulin resistant.

Patients with hyperprolactinemia also showed significantly increased TyG index (4.55±0.04 vs. 2.66±0.37, $p<0.001$) and TG/HDL ratio (5.05±0.80 vs. 1.00±0.13, $p<0.001$) compared to controls supporting the presence of insulin resistance (Table 1). Furthermore, there was a strong association between a higher BMI or obesity in males and worsening insulin resistance along with metabolic dysfunction. In addition to glucose metabolism disturbances, elevated prolactin levels, despite their mild effect, progressively associated to increased BMI, worsened lipid profile, and lower testosterone concentrations as illustrated in Table 1.

Lipid profile and metabolic dysfunction in hyperprolactinemia. Patients with hyperprolactinemia had significantly higher levels of triglycerides, LDL, and TG/HDL ratio, and significantly lower levels of HDL compared to the control group (Table 1). Our findings suggest that metabolic dyslipidemia is a major consequence of hyperprolactinemia.

Correlation analysis of metabolic parameters. As shown in Table 2, the results of the Pearson's analysis indicate statistically significant findings.

A positive relationship was found between prolactin and BMI ($r=0.716$, $p<0.01$), LDL ($r=0.811$, $p<0.01$), cholesterol ($r=0.655$, $p<0.05$), HOMA-IR ($r=0.538$, $p<0.05$), TyG index ($r=0.584$, $p<0.01$), and TG/HDL ratio ($r=0.746$, $p<0.01$), and a negative correlation was

Table 1
Baseline characteristics of participants and statistical analysis for two groups

Parameter	Control group (mean±SE)	Hyperprolactinemia group (mean±SE)	p value
Age (years)	28.29±2.20	31.00±2.26	ns
BMI (kg/m^2)	24.40±0.13	37.87±1.34	***
Prolactin (ng/mL)	11.07±1.19	86.59±21.65	**
Total testosterone (ng/dL)	5.60±0.60	2.24±0.44	**
Cholesterol (mg/dL)	147.29±10.13	217.43±10.53	***
Triglycerides (mg/dL)	107.57±9.83	143.29±11.71	*
HDL (mg/dL)	83.86±5.72	34.00±6.53	***
LDL (mg/dL)	72.00±4.82	124.57±20.24	*
Glucose (mg/dL)	79.71±3.53	65.14±2.91	**
Insulin (μIU/mL)	12.16±1.99	20.00±1.27	**
HOMA-IR	0.99±0.14	3.19±0.16	***
TyG index	2.66±0.37	4.55±0.04	***
QUICKI index	0.55±0.03	0.32±0.00	***
TG/HDL index	1.00±0.13	5.05±0.80	***

Abbreviations: BMI – body mass index; HDL – high-density lipoprotein; HOMA-IR – Homeostatic Model Assessment for Insulin Resistance; LDL – low-density lipoprotein; QUICKI – Quantitative Insulin Sensitivity Check Index; TG – triglycerides; TyG – triglyceride-glucose. *$p<0.05$; **$p<0.01$; ***$p<0.001$; ns – not significant.

demonstrated between prolactin and HDL (r=–0.783, p<0.01) and QUICKI (r=–0.643, p<0.01) (Table 2).

A strong inverse relationship was observed between testosterone and BMI (r=–0.682, p<0.01), HOMA-IR (r=–0.844, p<0.01) and TyG (r=–0.666, p<0.01) and a strong positive correlation was found between testosterone and QUICKI (r=0.703, p<0.01) (Table 2).

A positive correlation was demonstrated between HOMA-IR and BMI (r=0.863, p<0.01) as well as cholesterol (r=0.830, p<0.01) (Table 2). QUICKI was directly associated with HOMA-IR (both p<0.01). There were also strong correlations between TyG index and BMI (r=0.781, p<0.01) and TG/HDL (r=0.700, p<0.01) (Table 2) providing additional support that metabolic dysfunction links these diseases.

The statistical significance of these findings demonstrates how prolactin, obesity, testosterone, and lipid dysfunction are related to insulin resistance.

Discussion

This research confirms a specific link between hyperprolactinemia and insulin resistance, specifically for male individuals, who are overweight. Subjects with elevated prolactin levels had increased HOMA-IR scores along with increased TyG index and TG/HDL ratio and decreased QUICKI values, confirming insulin resistance according to established markers (Table 1). These findings were additionally confirmed by correlation analysis, which found that prolactin was positively linked with HOMA-IR (r=0.538) and the TyG index (r=0.584), but negatively with QUICKI (r=–0.643).

As earlier studies have found, chronically high prolactin levels can disrupt insulin receptors and lower the uptake of glucose into cells. Moreover, positive correlations between prolactin and BMI (r=0.716) and LDL (r=0.811) as well as a negative correlation with HDL (r=–0.783) reveal that prolactin is also related to malfunction in lipid metabolism (Table 2). The findings demonstrate how elevated prolactin levels convert into major insulin sensitivity deterioration alongside insulin dysfunction abnormalities that match with previous studies (Parak et al. 2020; Gierach et al. 2022; Sheoran et al. 2023; Wang et al. 2023).

These disturbances seem to have various biological processes responsible for their development. Insulin receptor signaling faces disruption due to hyperprolactinemia, which reduces cellular responsiveness to insulin signals (Leung 2021). Clinical evidence demonstrates that hepatic and muscle tissues manifest decreased glucose uptake levels in patients with prolactin excess thereby leading to insulin resistance.

Table 2
Pearson's correlation analysis for measured parameters

Parameter	Age	BMI	PRL	TT	Chol	TG	HDL	LDL	Glucose	Insulin	HOMA-IR	TyG index	QUIKI index	TG/HDL Index
Age	1	–	–	–	–	–	–	–	–	–	–	–	–	–
BMI	0.285	1	–	–	–	–	–	–	–	–	–	–	–	–
PRL	0.434	0.716**	1	–	–	–	–	–	–	–	–	–	–	–
TT	0.072	–0.682**	–0.499	1	–	–	–	–	–	–	–	–	–	–
Chol	0.263	0.748**	0.655*	–0.584*	1	–	–	–	–	–	–	–	–	–
TG	0.379	0.472	0.234	–0.278	0.472	1	–	–	–	–	–	–	–	–
HDL	–0.506	–0.835**	–0.783**	0.596*	–0.706**	–0.320	1	–	–	–	–	–	–	–
LDL	0.513	0.641*	0.811**	–0.197	0.628*	0.367	–0.655*	1	–	–	–	–	–	–
Glucose	–0.091	–0.686**	–0.460	0.576*	–0.596*	–0.508	0.494	–0.367	1	–	–	–	–	–
Insulin	0.262	0.622*	0.288	–0.586*	0.612*	.540*	–0.658*	0.178	–0.583*	1	–	–	–	–
HOMA–IR	0.029	0.863**	0.538*	–0.844**	0.830**	0.461	–0.732**	0.416	–0.632*	0.685**	1	–	–	–
TyG Index	–0.095	0.781**	0.584*	–0.666**	0.736**	0.433	–0.523	0.516	–0.695**	0.331	0.847**	1	–	–
QUIKI Index	–0.187	–0.863**	–0.643*	0.703**	–0.864**	–0.459	0.740**	–0.522	0.648*	–0.612*	–0.923**	–0.808**	1	–
TG/HDL index	0.330	0.787**	0.746**	–0.583*	0.739**	0.288	–0.877**	0.622*	–0.747**	0.575*	0.737**	0.700**	–0.769**	1

Abbreviations: BMI – body mass index; Chol – cholesterol; Glu – glucose; HDL – high-density lipoprotein; HOMA-IR – Homeostatic Model Assessment for Insulin Resistance; Ins – insulin; LDL – low-density lipoprotein; PRL – prolactin; QUICKI – Quantitative Insulin Sensitivity Check Index; TG – triglycerides; TT – total testosterone; TyG – triglyceride-glucose. *p<0.05; **p<0.01 (2-tailed).

Prolactin acts as a factor that damages β-cells in the pancreas leading to impaired insulin production and interrupted insulin release (Faron-Gorecka et al. 2023). Multiple conditions induced by prolactin have combined to generate observed changes in the insulin resistance profiles during this research.

Testosterone deficiency serves as an important factor that enhances insulin resistance among males who have elevated prolactin levels. The hyperprolactinemia group experienced much higher rates of low testosterone compared to the control group ($p<0.001$). An interesting point observed was a negative correlation between testosterone level and markers of insulin resistance, like HOMA-IR ($r=-0.844$) and TyG index ($r=-0.666$). We found a positive relationship between testosterone and insulin sensitivity, as seen by the association with QUICKI ($r=0.703$) (Table 2). It appears that men with high prolactin and low testosterone levels may develop increased insulin resistance, since prolactin likely lowers testosterone production by the hypothalamic-pituitary-gonadal axis. Testosterone controls glucose regulation and insulin sensitivity as well as preserving muscle mass (Szulc 2022). According to Trost (2021), the disruption of hypothalamic-pituitary-gonadal axis from hyperprolactinemia leads to decreased testosterone production, which potentially worsens metabolic abnormalities.

Research conducted by Flores et al. (2022) have supported the correlation between reduced testosterone levels and insulin sensitivity problems, which increases the likelihood of metabolic syndrome development. Testosterone depletion functions in partnership with prolactin-related metabolic issues to strengthen the insulin resistance markers detected within this study group.

Participants with hyperprolactinemia showed substantial lipid disturbance through elevated triglycerides and higher LDL combined with decreased HDL along with their high TG/HDL ratio. Previous research supports these dyslipidemic patterns because prolactin disrupts hepatic lipid metabolism alongside adipose tissue functionality (Han et al. 2021; Pirchio et al. 2022; Kabootari et al. 2023; Korta et al. 2024; Zaidalkilani et al. 2024).

The TG/HDL ratio and TyG index were higher in hyperprolactinemic individuals and had a positive relationship with prolactin, BMI, and LDL.

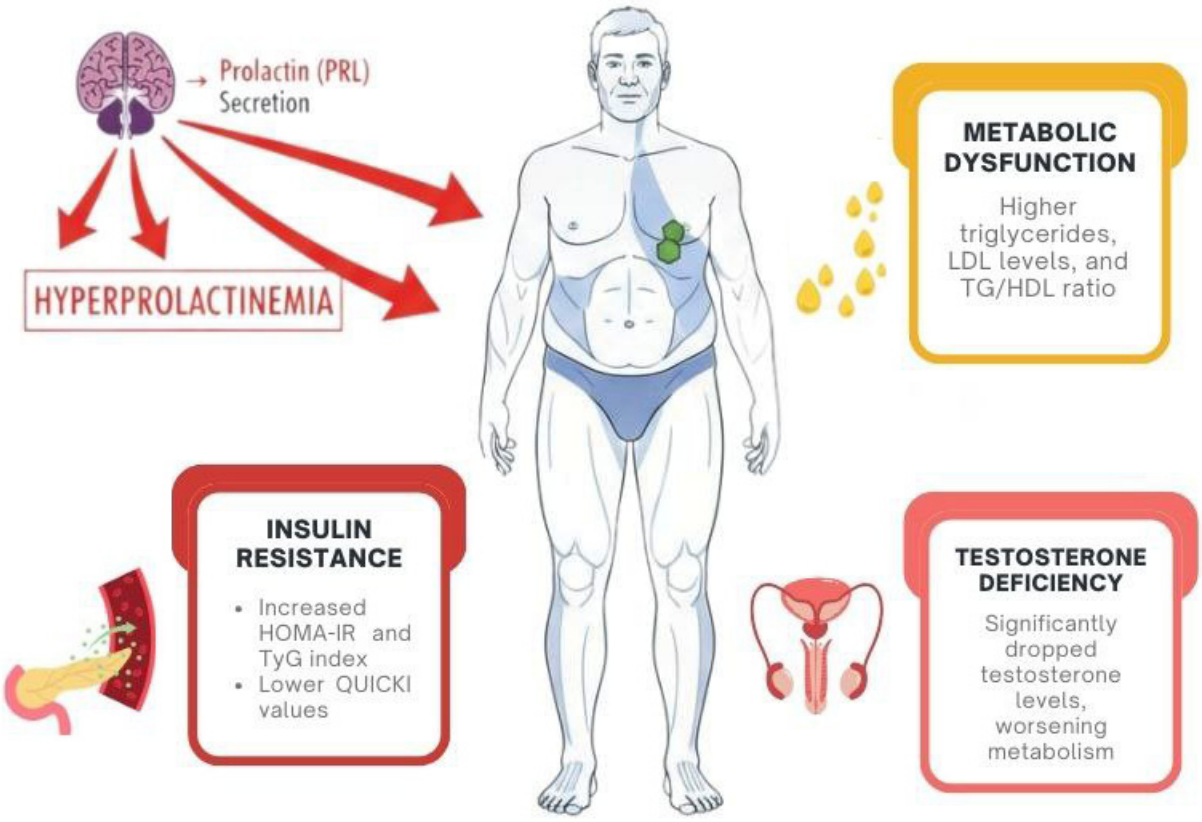

Figure 1. Graphical representation of the impact of hyperprolactinemia on insulin resistance markers in overweight males.

Correlations revealed that BMI and prolactin were strongly associated with TG/HDL ratio (r=0.787 and r=0.746, respectively), indicating that dyslipidemia may lead to insulin resistance and occur through hormonal dysfunctions. Moreover, the QUICKI and HOMA-IR indexes were strongly correlated in opposition (r=–0.923) showing they properly represent different aspects of insulin function (Table 2).

Furthermore, the co-occurrence of obesity in these individuals exacerbates the situation. Individuals with higher body mass indices showed worse insulin resistance results combined with more disrupted lipids along with reduced testosterone levels in their bloodstream. These patients face an increased risk of developing metabolic syndrome and cardiovascular complications due to the combined impact between high prolactin levels and dyslipidemia and hormonal imbalance problems. Metabolic testing along with prompt intervention must start early for overweight males with hyperprolactinemia as the data shows systemic dysfunction can progress when left untreated.

Conclusion

This study presents strong evidence that overweight men with hyperprolactinemia have insulin resistance, metabolic dysfunction, and testosterone deficiency. Elevated prolactin levels correlated with a) increased HOMA-IR and TyG index (increased insulin resistance), b) lower QUICKI values also indicate reduced insulin sensitivity, c) higher triglycerides and LDL levels, TyG index and TG/HDL ratio, and d) significantly dropped testosterone levels, worsening the metabolism (Figure 1).

Based on these findings, an early intervention approach to address the metabolic risks should incorporate prolactin lowering therapies, lifestyle modifications, and testosterone replacement therapies. Longitudinal studies studying the effects of hyperprolactinemia treatment on metabolic health should be carried out in the future.

Conflicts of interest: *The authors declare no conflicts of interest.*

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