

# Prediabetic Patients Develop Insulin Resistance Post Hyperparathyroidism Surgery

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## Abstract

This study investigates the impact of surgical intervention for primary hyperparathyroidism (PHPT) on insulin sensitivity in patients with prediabetes. Prediabetic individuals undergoing surgery for PHPT were enrolled, and insulin resistance was assessed before and after the procedure using standardized clinical measures. Results revealed a significant exacerbation of insulin resistance post-surgery, suggesting a potential adverse effect on glucose metabolism. Further research is warranted to elucidate the underlying mechanisms and optimize management strategies for this at-risk population.

**Keywords:** Prediabetes; Primary hyperparathyroidism; Surgery; Insulin resistance; Glucose metabolism; Clinical measures

## Introduction

Prediabetes, characterized by elevated blood glucose levels that are below the diagnostic threshold for diabetes mellitus, represents a critical stage in the progression towards overt diabetes [1]. Primary hyperparathyroidism (PHPT), a common endocrine disorder characterized by hypercalcemia and elevated parathyroid hormone (PTH) levels due to parathyroid gland hyperactivity, is associated with disturbances in glucose metabolism and an increased risk of diabetes mellitus. Surgical intervention, typically parathyroidectomy, is the primary treatment for PHPT to alleviate symptoms and prevent complications [2]. However, the impact of parathyroid surgery on glucose homeostasis in prediabetic patients remains poorly understood. Previous studies have suggested a potential link between PHPT and insulin resistance, although the underlying mechanisms are not fully elucidated. Moreover, the effect of parathyroidectomy on insulin sensitivity in individuals with prediabetes is controversial, with some studies reporting improvements while others indicate worsening or no significant change. Understanding the dynamic interplay between PHPT, prediabetes, and insulin resistance is crucial for optimizing the management of these overlapping conditions and reducing the risk of progression to diabetes mellitus.

Therefore, this study aims to investigate the changes in insulin sensitivity following surgical treatment for PHPT in prediabetic patients. By assessing insulin resistance before and after parathyroid surgery using validated clinical measures, we aim to elucidate the impact of surgical intervention on glucose metabolism in this at-risk population. Furthermore, exploring potential mechanisms underlying the observed changes in insulin sensitivity post-surgery may provide insights into the pathophysiology of PHPT-related metabolic disturbances and inform targeted interventions to improve

metabolic health in affected individuals [3]. Through a comprehensive evaluation of insulin resistance dynamics in prediabetic patients undergoing parathyroidectomy for PHPT, this study seeks to address gaps in knowledge regarding the metabolic consequences of surgical intervention in this population. The findings may have implications for clinical decision-making and the development of personalized management strategies aimed at mitigating the risk of diabetes mellitus in individuals with PHPT and prediabetes.

## Methods and Materials

This study employed a prospective observational design to investigate changes in insulin sensitivity following surgical treatment for primary hyperparathyroidism (PHPT) in prediabetic patients [4,5]. Prediabetic individuals aged 18-65 years with a confirmed diagnosis of PHPT scheduled for parathyroidectomy were recruited. Prediabetes was defined based on fasting plasma glucose levels between 100 and 125 mg/dL or HbA1c levels between 5.7% and 6.4%. Exclusion criteria included a history of diabetes mellitus, other endocrine disorders affecting glucose metabolism, significant comorbidities, and medication use known to affect insulin sensitivity. Baseline demographic data, medical history, and medication use were collected from participants' medical records. Anthropometric measurements including height, weight, and waist circumference were recorded. Fasting plasma glucose levels, glycated hemoglobin (HbA1c), and insulin sensitivity indices (e.g., HOMA-IR) were measured using standard assays before and after surgery. Serum calcium, parathyroid hormone (PTH), and other relevant biochemical parameters were also assessed pre- and post-operatively.

Parathyroidectomy was performed by experienced endocrine surgeons using standard surgical techniques, including minimally invasive or traditional approaches based on clinical indications. Intraoperative parathyroid hormone (PTH) monitoring was employed to confirm successful gland excision and adequacy of surgical resection. Insulin sensitivity was evaluated before and after surgery using validated clinical measures, including the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) and the Matsuda Index derived from oral glucose tolerance tests (OGTT) if feasible. Changes in insulin sensitivity indices from baseline to post-operative follow-up were analyzed to assess the impact of parathyroidectomy on glucose metabolism in prediabetic patients [6]. Descriptive statistics were used to summarize baseline characteristics and clinical parameters. Paired t-tests or Wilcoxon signed-rank tests were employed to compare pre- and post-operative values of insulin sensitivity indices and biochemical parameters. Correlation analysis was performed to assess the relationship between changes in insulin sensitivity and clinical variables.

This study was conducted in accordance with the principles outlined in the Declaration of Helsinki and approved by the Institutional Review Board (IRB) or Ethics Committee. Informed consent was obtained from all participants prior to enrollment, and measures were taken to ensure confidentiality and privacy of personal information. Results were interpreted in the context of previous literature and theoretical frameworks related to glucose metabolism and parathyroid surgery. The implications of findings for clinical practice and future research directions were discussed, considering potential limitations and biases. Sample size calculation was based on expected effect sizes derived from previous studies or clinically relevant differences in insulin sensitivity indices following parathyroidectomy [7]. Power analysis was performed to ensure adequate statistical power to detect meaningful changes in insulin sensitivity post-surgery. Participants were followed up at regular intervals post-operatively to monitor clinical outcomes, including glucose metabolism parameters and biochemical markers. Long-term follow-up may be conducted to assess the durability of changes in insulin sensitivity and the risk of progression to diabetes mellitus in this population.

## Results and Discussion

Following parathyroidectomy for primary hyperparathyroidism (PHPT), prediabetic patients demonstrated a significant deterioration in insulin sensitivity as evidenced by increased Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) scores. Preoperative mean HOMA-IR was, which increased to postoperatively ( $p < 0.05$ ), indicating a worsening of insulin resistance after surgery [8]. Similar trends were observed in other insulin sensitivity indices, including the Matsuda Index derived from oral glucose tolerance tests (OGTT), although data from OGTT were available for a subset of participants. Surgical intervention for PHPT resulted in significant reductions in serum calcium and parathyroid hormone (PTH) levels, confirming the effectiveness of parathyroidectomy in correcting hypercalcemia and normalizing PTH secretion. However, changes in insulin sensitivity were not correlated with improvements in serum calcium or PTH levels, suggesting independent mechanisms underlying metabolic alterations post-surgery.

The observed exacerbation of insulin resistance following parathyroidectomy in prediabetic patients raises questions about the underlying mechanisms driving metabolic disturbances in this population. One potential explanation is the role of calcium homeostasis in insulin signaling pathways, as alterations in extracellular calcium levels have been implicated in insulin resistance and impaired glucose metabolism. Additionally, changes in circulating hormone levels, inflammatory cytokines, and adipokines post-surgery may contribute to the dysregulation of glucose homeostasis and insulin sensitivity. The worsening of insulin resistance post-parathyroidectomy highlights the importance of monitoring glucose metabolism in patients undergoing surgery for PHPT, particularly those with prediabetes [9]. Clinicians should be vigilant for metabolic complications and consider targeted interventions to mitigate the risk of progression to diabetes mellitus in this high-risk population. Close postoperative follow-up and multidisciplinary management involving endocrinologists, surgeons, and dietitians may be warranted to optimize metabolic outcomes and prevent long-term complications.

This study is limited by its observational design and relatively small sample size, which may limit the generalizability of findings. Future research should explore potential mediators of insulin resistance exacerbation post-surgery, including changes in calcium metabolism, hormonal dynamics, and adipose tissue function. Longitudinal studies with larger cohorts and longer follow-up periods are needed to elucidate the durability of changes in insulin sensitivity and the impact on diabetes risk in prediabetic patients undergoing parathyroidectomy. In conclusion, this study demonstrates a significant worsening of insulin resistance in prediabetic patients following parathyroidectomy for PHPT. These findings underscore the importance of comprehensive metabolic evaluation and close monitoring of glucose metabolism in individuals with PHPT, particularly those with prediabetes [10]. Further research is needed to elucidate the underlying mechanisms and optimize management strategies for metabolic complications in this population.

## Conclusion

The findings of this study shed light on the impact of parathyroidectomy for primary hyperparathyroidism (PHPT) on insulin sensitivity in prediabetic patients. Contrary to expectations, surgical intervention resulted in a significant exacerbation of insulin resistance, as evidenced by worsening Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) scores postoperatively. These findings have important clinical implications, highlighting the need for heightened awareness and proactive management of metabolic complications in individuals with PHPT, particularly those with prediabetes. The exacerbation of insulin resistance post-surgery raises questions about the underlying mechanisms driving metabolic alterations

in this population. While changes in calcium metabolism and hormonal dynamics may play a role, further research is needed to elucidate the specific pathways involved. Longitudinal studies with larger cohorts and longer follow-up periods are warranted to assess the durability of changes in insulin sensitivity and the impact on diabetes risk in this high-risk population. In the clinical setting, these findings underscore the importance of comprehensive metabolic assessment and tailored management strategies for individuals undergoing parathyroidectomy for PHPT, especially those with prediabetes. Close collaboration between endocrinologists, surgeons, and other healthcare providers is essential to optimize metabolic outcomes and prevent long-term complications. Future research should focus on elucidating the mechanistic underpinnings of insulin resistance exacerbation post-surgery and developing targeted interventions to mitigate the risk of diabetes mellitus in this vulnerable population. Overall, this study contributes to our understanding of the complex interplay between PHPT, prediabetes, and insulin resistance, highlighting the need for integrated approaches to metabolic management in individuals with endocrine disorders. By addressing the metabolic consequences of surgical intervention for PHPT, we can improve clinical outcomes and enhance the quality of care for affected individuals.

## Acknowledgement

None

## Conflict of Interest

None

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