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Case Report

Clinical Outcomes of Vitamin D Repletion in a Patient with Insulin Resistance: A Case Report

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Abstract

Vitamin D is a secosteroid hormone that has traditionally been recognized for its central role in calcium-phosphate homeostasis and skeletal health. In recent years, growing evidence has suggested that vitamin D may also influence a range of extra-skeletal functions, including immune modulation, inflammation regulation, and glucose metabolism. Of particular interest is its potential association with insulin sensitivity and the pathophysiology of insulin resistance.

Keyword: Insulin resistance, Vitamin D, High dose, Obesity, Treatment

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Introduction

Insulin resistance is a metabolic condition characterized by reduced responsiveness of peripheral tissues to circulating insulin, leading to compensatory hyperinsulinemia and an increased risk of developing type 2 diabetes mellitus and other metabolic disorders. Multiple observational studies have demonstrated an association between low serum vitamin D levels and increased prevalence of insulin resistance; however, interventional studies have yielded inconsistent outcomes regarding the therapeutic benefit of vitamin D supplementation.

This case report describes the clinical course of an individual diagnosed with vitamin D deficiency and features suggestive of insulin resistance. The patient presented with biochemical evidence of reduced serum 25-hydroxyvitamin D levels along with metabolic abnormalities consistent with impaired insulin action. Clinical manifestations included features commonly associated

with insulin resistance, such as acanthosis nigricans and weight gain, along with menstrual irregularities suggestive of endocrine imbalance.

A high-dose vitamin D supplementation regimen was initiated following baseline evaluation. The supplementation was administered over a six-month period, with periodic clinical and biochemical monitoring to assess metabolic response and safety outcomes. The primary objective of the intervention was to correct vitamin D deficiency and observe potential downstream effects on insulin sensitivity and associated clinical features.

Over the course of treatment, a progressive increase in serum vitamin D levels was observed, eventually reaching values within the optimal physiological range. This improvement in vitamin D status coincided with notable metabolic and clinical changes. The patient exhibited improved insulin sensitivity, as reflected by better glycemic control indicators and reduced clinical signs of insulin resistance. A gradual regression of acanthosis nigricans was also noted, suggesting improvement in underlying hyperinsulinemic state.

In addition to metabolic improvements, the patient experienced a reduction in body weight over the follow-up period. This weight change may have contributed further to improved insulin responsiveness. Furthermore, menstrual cycles became more regular, indicating possible restoration of endocrine balance, potentially mediated through improved metabolic function and vitamin D-related hormonal interactions.

The observed clinical improvements may be explained through several proposed biological mechanisms. Vitamin D is thought to influence insulin secretion through its action on pancreatic β-cells and may enhance insulin sensitivity by modulating inflammatory pathways and upregulating insulin receptor expression in peripheral tissues. It may also play a role in regulating adipokine secretion, thereby influencing energy metabolism and adiposity.

Despite these favorable observations, it is important to acknowledge that current evidence regarding vitamin D supplementation and insulin resistance remains inconclusive. While some studies suggest beneficial metabolic effects, others have reported minimal or no significant improvement in insulin sensitivity following supplementation. This variability may be attributed to differences in baseline vitamin D status, dosage regimens, study duration, genetic factors, and heterogeneity in study populations.

Conclusion

Therefore, although this case supports a potential association between vitamin D repletion and metabolic improvement, it does not establish a definitive causal relationship. The findings should be interpreted cautiously, and high-quality randomized controlled trials with larger sample sizes are necessary to better define the therapeutic role of vitamin D in the management of insulin resistance and related metabolic disorders.

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