INTRODUCTION

Glucose Homeostasis refers to the maintenance of a stable glucose level within the body by balancing insulin and glucagon. In clinical practise, Thyroid hormones influence the Glycolysis and liver function and vice versa. In 1927, Coller and Huggins investigated association of hyperthyroidism can worsen the condition of diabetes [1-20]. Diabetes mellitus and Thyroid dysfunction mutually have a deep underlying relation. Studies have evidenced that complex intertwines chemical, genetic and secretory malfunctions brings ambiguous pathway mechanism [21-50].

EPIEDEMOLOGY

According to reports, there is a greatest prevalence of thyroid disorder is in pre-menopausal women. The ratio of thyroid disorder in women to men is 4:1 constitutes 31.4% in female and 6.9% in male. In the late 1970s, Whickham survey conducted within the north of European country disclosed a prevalence of 6.6% of thyroid disorders within the adult general population. Within the NHANES III study, a survey of 17,353 subjects representing the American people, glandular diseases was found in 4.6% and thyrotoxicosis in 1.3% of subjects. The latter additionally ascertained an enhanced frequency of thyroid dysfunction with advancing better prevalence of thyroid disorders in women compared to men and in diabetic subjects compared to non-diabetic [51-70].

EFFECTS OF NON-INSULIN DEPENDENT DIABETES MELLITUS ON THYROID DISORDERS

Alterations in hormones of thyroid are delineated in patients with Non-insulin dependent DM. Glandular disease is usually related to worsening glycemic management and exaggerated hypoglycemic agent needs. There’s underlying exaggerated liver gluconeogenesis, fast abdominal glucose absorption, and possibly exaggerated hypoglycemic agent resistance [71-80].
EFFECTS OF THYROID HORMONES ON BLOOD GLUCOSE REGULATION

By many mechanisms, Thyroid hormones have an effect on Blood glucose regulation. Throughout glandular disease, the half-life of Endocrine hormone is reduced, resulting in an increased rate of degradation and thereby enhancing the unleashing of biologically inactive insulin precursors. There is another mechanism explaining the link between thyrotoxicosis and symptoms that increase the sugar level, mediated by the surplus thyroid hormones [81-90].

Most of the genes concerned in carbohydrate metabolism are regulated by an active thyroid hormone Triiodothyronine, by exerting its action via binding to the Triiodothyronine receptor. These Triiodothyronine receptors were derived from two separate genes which encode the major T3-binding isoforms TRα1, TRβ1, TRβ2 and TRβ3. The TRα1 is predominantly concerned within the metabolic effects of Endocrine gland [91-100].

It is known fact that diabetic patients with thyrotoxicosis face the results of glycemic management and glandular disorder shown to precipitate diabetic ketoacidosis in persons with high blood sugar levels [15-25].

CONCLUSION

There is a vivid interdependent interaction between diabetes and endocrine disorders. Failure to recognize the abnormalities in thyroid hormone level in diabetes may be a primary cause of poor management. A scientific approach to thyroid testing in diabetic patients is favourable; but there’s no definitive points relating to screening for the thyroid malfunctioning in diabetic patients.

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