INTRODUCTION

Diabetes mellitus is characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both. When fully expressed, diabetes is characterized by fasting hyperglycemia, but the disease can also be recognized during less overt stages, most usually by the presence of glucose intolerance. The effects of diabetes mellitus include long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, heart, and blood vessels. (**Peter & William ., 2005).**

Diabetes may present with characteristic symptoms such as thirst,polyuria, blurring of vision, weight loss, and polyphagia, and in its most severe forms, with ketoacidosis or nonketotic hypersomolarity, which in the absence of effective treatment, leads to stupor, coma, and death. Often symptoms are not severe or may even be absent. Hyperglycemia sufficient to cause pathologic functional changes may quite often be present for long time before the diagnosis is made. Consequently diabetes often is discovered because of abnormal results from a routine blood or urine glucose test or because of the presence of a complication.**(American Diabetes Association., 2002)**.

In some instances diabetes may be apparent only intermittently; as, for example, with glucose intolerance in pregnancy or gestational diabetes, which may remit after parturition. **(American Diabetes Association .,2002).**

In some individuals the like-lihood of developing diabetes may be recognized even before any abnormalities of glucose tolerance are apparent. (**George et al ., 2004**) During the evolution of type I diabetes, for example, immunologic disturbances such as islet cell or other antibodies are present, and these .nay precede clinically apparent disease by months or

even years (**Almind et al ., 2001)** . In some families it is possible to recognize certain gene mutations that are strongly associated with certain forms of diabetes, such as variations in the glucokinase gene or hepatic nuclear factor genes that cause youth or early adult-onset diabetes. These genetic abnormalities are detectable at any time. **(WHO Study Group., 2005)**.

Although a number of specific causes of diabetes mellitus have been identified, the etiology and pathogenesis of the more common types are less clearly understood. The majority of cases of diabetes fall into two broad etiopathogenetic categories, called type 1 and type 2 diabetes (**Gavin et al .,199**7), but the extent of heterogeneity among these types remains uncertain. Because of the increasing number of forms of diabetes for which a specific etiology can be recognized, the current clinical classification, proposed by the American Diabetes Association (ADA) in 1997 and adopted by the World Health Organization (WHO) in 1999 and that supersedes the previously internationally recognized 1985 WHO classification**. (O'Sullivan et al .,1999)** now classifies diabetes according to both clinical stages and etiologic types. The clinical staging reflects that diabetes progresses through several stages during its natural history and that individual subject may move from one stage to another in either direction.

The thyroid gland maintains the level of metabolism in the tissues that is optimal for their normal function. Thyroid hormone stimulates the o_2 consumption of most of the cells in the body, regulates lipid and carbohydrate metabolism, and is also necessary for normal growth and maturation (**Ekholm, 1989).** The main function of the gland is to synthesize and secrete the thyroid hormones, namely,

thyroxine (T_4), triiodothyronine (T_3) and calcitonine **(Ingbar & woeber, 1981).** The thyroid gland plays an important role in the normal metabolic rate and that is why a precise control system is operating to provide the right amounts of thyroid hormones at different conditions, both suprathyroid and autoregulatory mechanisms are involved in this control system **(Morley, 1981).**

The secretion of thyrotropin (TSH), which is the major modulator of thyroid function, is regulated at the level of the pituitary thyrotroph by the antagonistic effects of thyroid hormones and the TSH releasing hormone(TRH), the former inhibits and the latter stimulates the synthesis and secretion of (TSH). Therefore, excess thyroid hormone leads to decreased secretion of (TSH), and thyroid hormone insufficiency is associated with (TSH) hypersecretion, TSH stimulates all steps of thyroid hormone synthesis and secretion **(Morley, 1981).**

insulin and thyroid hormones influence each other actions assumes significance since diabetes mellitus and thyroid disease are the two common endocrine disorders in adult population. Poorly controlled diabetes results in a low T3 state and a loss of TSH response to TRH. Regardless of glycemic control there is an absence of nocturnal THS peak. There is an increased incidence of dysthyroid optic neuropathy in patients with Graves disease and coexistent diabetes. Also, the visual prognosis after treatment is poor. 50% of thyrotoxic patients who were previously euglycemic exhibit variable glucose intolerance while frank diabetes occurs in 2-3% cases. In diabetic patients presence of thyrotoxicosis deteriorates the glycemic control. Diabetic patients with hypothyroidism may suffer from recurrent episodes of hypoglycemia. Type 1 diabetes mellitus patients

have a high prevalence of thyroid dysfunction with nearly one third of all newly detected patients showing thyroid autoimmunity. Also, Type 1 diabetes mellitus and hypothyroidism co-exist in downs syndrome as well as congenital rubella. In autoimmune polyglandular syndrome Type 2, autoimmune thyroid disease and immune mediated diabetes are among the components. **(Schlienger et al .,2006**)

Diabetes mellitus and thyroid diseases are the two common endocrine -opathies seen in the adult population. With insulin and thyroid hormones being intimate involved in cellular metabolism and thus excess or deficit of either of these hormones could result in the functional derangement of the other. This article will review how diabetes affects thyroid function in euthyroid individuals, how it affects pre-existing thyroid disorders, conversely how thyroid diseases clinical situations where both the diseases could coexist. (**Fernandez Castaner et al .,2000**).