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Urinary Alpha-1-Microglobulin Excretion as an Early Predictor for Renal Tubular Malfunction in Diabetic Patients

Thesis

Submitted in partial fulfillment for requirements of

Master Degree in

Clinical Pathology

By

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بسم الله الرحمن الرحيم

﴿و ما أوتيتم من العلم إلا قليلا﴾

صدق الله العظيم

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Aim of the Work

Introduction

Renal impairment is an important feature of diabetes mellitus causing significant morbidity and mortality, and much effort has been devoted to the detection of this complication at a stage when it is silent and potentially reversible (lapsley et al1993).

The detection of low level albuminuria reliably predicts a high risk of the subsequent development of progressive and fatal renal disease in diabetic patients (Mogensen, 1984). More importantly, it has been shown that improvement of glycaemic control at this stage can reduce the proteinuria and significantly improve the outlook for long term renal function (Feldt-Rasmussen et al, 1986).

The increased albumin excretion is usually considered to be of glomerular origin but there is evidence that tubular malformation may also contribute (Abrass, 1984, Gibb et al, 1989). Also, impaired tubular reabsorption of low molecular mass plasma proteins has been reported in diabetic patients with normal albumin excretion (Holm et al, 1988).

Alpha-1-microglobulin is a glycosylated protein of molecular weight between 26,000-33,000 daltons containing167 aminoacid (Takagi et al, 1981). It has been shown that the measurement of urinary alpha-1-microglobulin can provide information about the renal filtration and reabsorption of low molecular weight protein and in absence of renal

failure a raised urinary alpha-1-microglobulin is an indicator of tubular proteinuria (Yu et al 1983).

Aim of the Work

The aim of the present work is to study urinary excretion of alpha-1-microglobulin in diabetic patients as an early marker of renal tubular malformation in those patients.

Review of Literatures

Review of Literature

Diabetes Mellitus

Diabetes mellitus is a common disorder, and its complications account for over 25% of all new cases of end stage renal failure and over 50% of all lower extremity amputations, also diabetes is a leading cause of blindness (Andreoli et al, 1990).

Classification of Diabetes Mellitus

According to the world health organization (WHO) 1980, two major forms of diabetes were recognized; which they termed insulin dependent diabetes: IDDM (type I diabetes) and NIDDM (type II Diabetes). Their classification system went on to include evidence that diabetes mellitus was etiologically and clinically heterogeneous group of disorders that share hyperglycemia in common.

On this basis, diabetes mellitus was classified into five distinct types (IDDM, NIDDM, gestational diabetes mellitus, malnutrition-related diabetes and other types). In addition, the 1979 classification included the category of impaired glucose tolerance (IGT), in which plasma glucose levels during an oral glucose tolerance test were above normal but below those defined for defined diabetes.

This previous classification indicated that the disorders grouped together under the term diabetes differ markedly in pathogenesis, natural history, response to therapy, and prevention. In addition,

different genetic and environmental factors can result in forms of diabetes that appear phenotypically similar but may have different etiologies.

An international expert committee, working under the sponsorship of the American diabetes association, was established in MAY 1995 to review the scientific literature since 1979. In 1997 this committee has proposed changes to the national diabetes data group (NDDG)/WHO classification scheme which are listed as:

I. **Type** I diabetes (B-cell destruction, usually leading to absolute insulin deficiency):
   A) Immune mediated.
   B) Idiopathic.

II. **Type** II diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance).

III. **Other specific types:**
   A) Genetic defects of B-cell function:

1. Chromosome 12, HNF 2. 1a (formerly MODY3).
3. Chromosome7, glucokinase (formerly MODY2).
4. Chromosome 20, HNF-4a (formerly MODY I).
5. Mitochondrial DNA. 6. Others.