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ROLE OF HbA1C AND MICROALBUMINURIA AS EARLY MARKERS OF NEPHROPATHY IN TYPE 2 DIABETES MELLITUS

KEYWORDS: Evidence based Dentistry, Cochrane database, meta- analysis, cohort studies

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ABSTRACT

Background: Diabetes mellitus with its accompanying vascular complications is on a rise globally and a similar trend has been observed in Libya too. Early detection, effective monitoring and timely management is important to control this growing problem. Various studies have shown microalbuminuria as an early indicator of nephropathy in diabetes mellitus. The present study is aimed at evaluating microalbuminuria and HbA1C in type 2 diabetes mellitus.

Introduction

While not used for diagnosis, an elevated level of glucose irreversibly bound to hemoglobin (termed glycosylated hemoglobin or HbA1c) of 6.0% or higher (the 2003 revised U.S. standard) is considered abnormal by most labs; HbA1c is primarily used as a treatment-tracking test reflecting average blood glucose levels over the preceding 90 days (approximately). However, some physicians may order this test at the time of diagnosis to track changes over time. The current recommended goal for HbA1c in patients with diabetes is <7.0%, which as defined as (good glycemic control) although some guidelines are stricter (<6.5%). People with diabetes who have HbA1c levels within this range have a significantly lower incidence of complications from diabetes, including retinopathy and diabetic nephropathy, **(Genuth, 2006).**

Kidney Disease

Diabetes can damage the kidneys, which not only can cause them to fail, but can also make them lose their ability to filter out waste products, kidneys are remarkable organs. Inside them are millions of tiny blood vessels that act as filters. Their jobs are to remove waste products from the blood. Sometimes this filtering system breaks down. Diabetes can damage the kidneys and cause them to fail. Failing kidneys lose their ability to filter out waste products, resulting in kidney disease, **(Franz, 2007).**

Materials and methods: Sixty subjects in the age group ranging from 40 to 70 years have been recruited from Seventeenth February Teaching Hospital, Al- Baida for the study, twenty controls with no history of diabetes, twenty recently diagnosed diabetics under treatment and twenty old cases of diabetes mellitus who have suffered an episode of coronary artery disease (CAD) or cerebrovascular accident (CVA). Venous samples were drawn after an overnight fast for glucose, glycosylated hemoglobin, urea and Creatinine and 24 hours urine sample for albumin estimation. The tests were performed using Enzyme Immunoassay for the quantitative determination of Albumin in urine ,(Caduff, 1991) . HEMOGLOBIN A1C was measured by chromatographic-spetrophotometric ION EXCHANGE method, (Bisse and Abraham,1985). Enzymatic determination of urea (Modified Bert helot reaction) (Patton and Crouch, 1977), Glucose was measured by glucose oxidase enzymatice method(ref) and Creatinine was measured by colorimetric method(ref) by using Cobas integra 400 auto analyzer.

Results: Slightly high levels of fasting blood glucose (p=0.03) and glycosylated hemoglobin (p=0.02) but normal urea, Creatinine and 24 hours urinary albumin were observed in diabetics under treatment when compared with controls. However, diabetics with complications showed higher levels of fasting blood glucose (p<0.0001), glycosylated hemoglobin (p<0.0001), serum urea, Creatinine and microalbuminuria. There is positive correlation of glycosylated hemoglobin and albuminuria in the diabetic group with coronary artery and cerebrovascular complications. All the results were summarized in tables 1 to 5

Discussion

The current study showed that kidney function tests (urea and creatinine) concentration in diabetic patients (group II and III) were higher than in normal controls (group I). As the diabetes disease got advanced and complicated, these kidney functions got worse. This is indicated by statistically highly significant increase in the creatinine levels in complicated diabetic patients (group III) when compared to uncomplicated diabetic group (group II).

The current study showed that the incidence of microalbuminuria in diabetic patients (group II and III) was statistically higher than in normal controls (group I). Such difference was absent between complicated diabetic patients (group III) when compared to uncomplicated diabetic group (group II).

At the same time, present study revealed statistically insignificant positive correlations between microalbuminuria and homocysteine concentration in the diabetic groups studied (II and III) as indicated by correlation coefficient studies.

These results were in agreement with Parving et al, 1992, who reported that the prevalence of micro albuminuria was significantly

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higher in NIDDM than non diabetic subjects.

Conclusion: The present study has shown a significant correlation of glycosylated hemoglobin and urinary albumin in diabetes mellitus with vascular complications. Hence measurement of glycosylated hemoglobin along with microalbuminuria is significant as an early marker in predicting nephropathy in uncontrolled type 2 diabetes mellitus with complications

	Control (group I)	Group II (group I Vs. Group II)	Group III (group I Vs. Group III)	(Group II Vs. Group III)
Range	64 – 110	86 – 224	124 - 377	
Mean	90.7	158.95	191.85	
S.D.	± 13.85	± 39.99	± 56.053	
t-test		-7.298	-7.657	-1.877
P-value		< 0.0001	< 0.0001	0.076
significance		H.S	H.S	N.S

H.S. = Highly significant

Table 2: HbA1c% in control and two other studied groups (number = 20 each).

	Control (group I)	Group II (group I Vs. Group II)	Group III (group I Vs. Group III)	(Group II Vs Group III)
Range	4.2 – 6.0	6.3 – 15.8	6.6 - 13	
Mean	5.64	9.23	8.86	
S.D.	± 1.66	± 2.265	± 1.62	
t-test		- 6.720	- 5.459	0.582
P-value		< 0.0001	< 0.0001	0.567
significance		H.S	H.S	N.S

Table 3: Microalbuminuria (mg/L) in control and two other studied groups (number = 20).

	Control (group I)	Group II (group I Vs. Group II)	Group III (group I Vs. Group III)	(Group II Vs. Group III)
Range	6 – 20	9 – 100	8 - 150	
Mean	11.2	21.1	27.95	
S.D.	± 5.29	± 20.55	± 37.07	
t-test		-2.1	-2.006	-0.956
P- value		< 0.001	< 0.001	0.351
significance		Significant	Significant	N.S.

Table 4: Serum urea (mg/dl) in control and two other studied groups (number = 20 each).

	Control (group I)	Group II (group I Vs. Group II)	Group III (group I Vs. Group III)	(Group II Vs. Group III)
Range	24 – 48	27 – 79	24 - 49	
Mean	33.10	41.05	37.20	
S.D.	± 6.21	± 11.29	± 7.36	
t-test		-2.633	-2.333	1.281
P value		< 0.001	< 0.001	0.216
significance		Significant	Significant	N.S.

Table 5:Serum creatinine (mg/dl) in control and two other studied groups (number = 20 each).

	Control (group I)	Group II (group I Vs. Group II)	Group III (group I Vs. Group III)	(Group II Vs. Group III)
Range	0.6 – 1.2	0.6 – 2.1	0.8 – 2.4	
Mean	0.875	0.970	1.110	
S.D.	± 0.171	± 0.414	± 0.359	
t-test		- 0.948	- 2.590	- 1.339
P -value		0.349	0.014	0.197
significance		N.S	significant	N.S.

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