



E-ISSN: 2706-9575
P-ISSN: 2706-9567
IJARM 2020; 2(2): 246-247
Received: 18-02-2020
Accepted: 22-04-2020

Dr. Anil Vijayakumar
Associate Professor,
Department of Medicine,
Azeezia Institute of Medical
Sciences and Research,
Kollam, Kerala, India

Dr. Radha Krishnan MP
Associate Professor,
Department of Psychiatry,
Azeezia Institute of Medical
Sciences and Research,
Kollam, Kerala, India

Dr. Dhanush Krishna
Junior Resident, Department
of Medicine, Azeezia Institute
of Medical Sciences and
Research, Kollam, Kerala,
India

Corresponding Author:
Dr. Dhanush Krishna
Junior Resident, Department
of Medicine, Azeezia Institute
of Medical Sciences and
Research, Kollam, Kerala,
India

A study of association of proteinuria with hba1c in diabetes mellitus

Dr. Anil Vijayakumar, Dr. Radha Krishnan MP and Dr. Dhanush Krishna

DOI: <https://doi.org/10.22271/27069567.2020.v2.i2d.80>

Abstract

Diabetic nephropathy occurs in as many as 30% of insulin dependent diabetes mellitus patients and 25% of noninsulin-dependent diabetes mellitus patients. Diabetic nephropathy is a dreaded disease with progressive and continuous. Deterioration in glomerular function resulting in irreversible renal failure. Diabetic nephropathy is an important cause of morbidity and mortality and is now among the most common cause of end-stage renal disease. However, there is an early phase of diabetic renal disease called incipient diabetic nephropathy. This study is intended to study the association of Proteinuria with HbA1C in Diabetes Mellitus.

Keywords: HbA1C, proteinuria, diabetes mellitus

Introduction

The term *HbA1c* refers to glycated haemoglobin. It develops when haemoglobin, a protein within red blood cells that carries oxygen throughout your body, joins with glucose in the blood, becoming 'glycated'. By measuring glycated haemoglobin (HbA1c), clinicians are able to get an overall picture of what our average blood sugar levels have been over a period of weeks/months. For people with diabetes this is important as the higher the HbA1c, the greater the risk of developing diabetes-related complications. When the body processes sugar, glucose in the bloodstream naturally attaches to haemoglobin. The amount of glucose that combines with this protein is directly proportional to the total amount of sugar that is in your system at that time. Because red blood cells in the human body survive for 8-12 weeks before renewal, measuring glycated haemoglobin (or HbA1c) can be used to reflect average blood glucose levels over that duration, providing a useful longer-term gauge of blood glucose control. If your blood sugar levels have been high in recent weeks, your HbA1c will also be greater. Two large-scale studies – the UK Prospective Diabetes Study (UKPDS) and the Diabetes Control and Complications Trial (DCCT) – demonstrated that improving HbA1c by 1% (or 11 mmol/mol) for people with type 1 diabetes or type 2 diabetes cuts the risk of microvascular complications by 25%.

Microvascular complications include

- Retinopathy
- Neuropathy
- Diabetic nephropathy (kidney disease)

Research has also shown that people with type 2 diabetes who reduce their HbA1c level by 1% are ^[1]:

- 19% less likely to suffer cataracts
- 16% less likely to suffer heart failure
- 43% less likely to suffer amputation or death due to peripheral vascular disease.

This study is intended to study the association of Proteinuria with HbA1C in Diabetes Mellitus.

Aims and Objectives

To study the association of Proteinuria with HbA1C in Diabetes Mellitus.

Materials and Methods

- Design: Cross-sectional design.
- Study population and setting: 100 patients admitted in the general medicine wards.
- The study was done from Nov 2019 to March 2020 in the Department of General Medicine, Azeezia Institute of Medical research and Sciences.

Inclusion criteria

1. Age: 18-80 years.
2. Those who gave written informed consent.

Exclusion criteria

1. Patients unwilling and uncooperative for the study
2. Patients suffering from known kidney pathology.
 - **Sampling method:** Convenience sampling.
 - **Study duration:** 5 months.

Results

Table 1: Age and Sex Distribution

Mean Age	Std. Deviation	Male	Female
58.65 years	±7.76 years	37	63

Table 2: HbA1C association with Proteinuria

HbA1C levels	Proteinuria				Significance
	-	+	++	+++	
<6.5	39	Nil	Nil	Nil	Not Sig
6.5-7	11	04	01	Nil	Not Sig
7-7.5	Nil	09	05	03	Not Sig
>7.5	Nil	03	04	21	P<0.001

Discussion

The knowledge of diabetes dates back to centuries before Christ. Polyuria disease resembling diabetes was described as early as 150 BC in ancient Egyptian records discovered by George Beers. Celsius (30BC-50AD) had recognised the disease. Diabetes, a Greek term, which literally means to 'run thru' or a 'siphon' was initially used by Aretaeus in first century AD for the generic description of a condition causing increased urine output. Roman physicians thought of diabetes as a "wonderful affection", not very frequent among men being melted down of flesh and limbs into urine. The patient never stopped making water, but the flow is incessant as if from an opening of aqueducts Aretaeus, the Cappadocia.^{1,2}

Several major studies among diabetics have been undertaken (Tuft *et al.* 1956, Farqahar *et al.* 1959, Gellman *et al.* 1959, Hatch *et al.* 1961, O Sullivan *et al.*, Thomsal) and proteinuria especially when diffuse changes are considered on the whole advanced clinical diseases accompanied by severe glomerular lesions.³ In 1963, Keen and Chlouervakis developed sensitive and specific radioimmunoassay for detecting human albumin in low concentration, i.e. proteinuria, which indicate earliest stage of diabetic renal disease. Later various other methods were developed for detection of proteinuria.⁴ This means significant increase in Albumin Excretion Rate (AER). Albumin excretion in healthy individuals ranges from 1.5 to 20 mcg/min. with geometric mean in the range of 6.5 mcg/min., these have been termed norm albuminuria. Proteinuria thus defines the wide substantial range of albumin hypersecretion ranging between 20- to 200-mcg/min. (30 to 300 mg/day). Normal persons excrete less

than 30 mg/day.⁵ Proteinuria is not detected by reagent sticks for urinary protein, which generally becomes positive only when proteinuria is greater than 550 mg/day. This degree of leakage is termed macroalbuminuria.⁶ In contrast to microproteinuria, the degree of macroproteinuria shows no relationship with current level of diabetic control. Neither the mean plasma glucose concentration nor glycosylated haemoglobin levels correlated significantly with clearance and excretion rates of different proteins. Long-term correction of hyperglycaemia by an intensified treatment regimens failed to stop or significantly slow the progressive increase in fractional clearance of albumin and IgG in insulin-dependent diabetic subjects with renal failure over a period of 2 years observation.⁷ In IDDM patients with low levels of proteinuria (i.e., AER of 20-30 mcg/min.), no consistent glomerular abnormalities have been found. Above these levels of urinary albumin excretion, however, the fractional volume of mesangium is on average significantly increased and minor reduction in creatinine clearance and rise in blood pressure are observed. Similar findings have been reported in NIDDM patients with proteinuria and proteinuria⁸.

Conclusion

There is a strong association with rising levels of HbA1C and proteinuria.

References

1. Schadewaldt H. History of diabetes mellitus. In: Van Englehardt D, ed. Diabetes, its medical and cultural history. Berlin: Springer Verlag 1989.
2. Crael LP, Bornett D, Levine R. The history of diabetes. Chapter 1. In: Joslins diabetes mellitus, Kahn ER, Weir GC, eds. New Delhi: BI Waverly Pvt Ltd 1994:1-13.
3. Ritz E, Flisa D, Siebers M. Diabetic nephropathy. Chapter-47. In: Massry SG, Glossock RJ, ed. Massry and Glassck's text book of nephrology. 3rd edn. Vol.I. Williams & Wilkins 1995. Jebmh.com Original Article J. Evid. Based Med. Healthc., pISSN- 2349-2562, eISSN- 2349-2570/ Vol. 3/Issue 98/Dec. 08, 2016 Page 5412
4. Keen H, Chlouervakis C. An immunoassay method of estimating urinary albumin at low concentration. Lancet 1963;2:913-916.
5. Mongensen CE, Maurer SM, Kjellstraud CM. Diabetic nephropathy. Chapter-79. In: Schrier RW, Gottschalk WC, eds. Disease of the kidney. 4th edn. Vol. III. Boston: Little Brown 1998.
6. Alvin C. Powers: Diabetes mellitus. Chapter-323. In: Braunwald E, Fauci A, Kasper D, *et al.*, eds. Harrison's principles of internal medicine. 17th edn. New York: McGraw Hill 2008.
7. Van Dijk C, Berl T. Pathogenesis of diabetic nephropathy. Rev Endocr Metab Disord 2004;5:3:237-248.
8. Hosteller TH, Daniels BS. Natural history of renal structural abnormalities in diabetes mellitus. In: Brenner BM, Stein JH, eds. The kidney in diabetes mellitus. Contemporary issues in nephrology. New York: Churchill Livingstone 1989,p51.