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MICROALBUNIA AND DIABETIC NEPHROPATHY - A MINI REVIEW

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ABSTRACT

In both the United States and Europe, diabetes is currently the major cause of end-stage renal disease (ESRD). People with diabetes live longer lives, type 2 diabetes is becoming more prevalent, and patients with diabetic ESRD are increasingly being admitted for treatment in ESRD programs that had previously rejected them, all of which contribute to this. Nephropathy affects 20–30% of people with type 1 or type 2 diabetes, but only a much smaller proportion of people with type 2 diabetes develop end-stage renal disease. As a result of the much higher prevalence of type 2 diabetes, these patients account for more than half of all diabetic patients who begin dialysis. Numerous therapies are effective, according to recent studies.

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1. INTRODUCTION

Diabetes is now the main contributor to end-stage renal disease in the US and Europe (ESRD). This is due to the rise in the prevalence of diabetes, particularly type 2, the fact that people with diabetes live longer lives and that patients with diabetic ESRD are now being admitted for treatment in ESRD programs that had previously excluded them [1–3]. Nephropathy affects 20–30% of people with type 1 or 2 diabetes. However, only a significantly smaller percentage of people with type 2 diabetes progress to end-stage renal disease (ESRD)[4,5]. However, these patients make up more than half of all diabetic patients who start dialysis due to the significantly higher prevalence of type 2 diabetes. Recent studies have demonstrated that several therapies can considerably slow the development and progression of diabetic nephropathy. However, these interventions are most effective when started as soon as this problem manifests [6,7]. India, the second-most populated nation in the world, has suffered greatly due to the global diabetes epidemic [8].

2. PREVALENCE OF DIABETES

According to the International Diabetes Federation, almost half of all people with diabetes live in China (98.4 million), India (65.1 million), and the United States (2013). 24.4" million. Diabetes is a complex disease in India, where hereditary and environmental variables mix to cause obesity, linked to improving living standards, steady urban migration, and alterations in lifestyle. Although obesity is a significant diabetes risk factor, little study has been done on this risk factor in India [9,10]. India has greater incidences of diabetes than Western nations while having lower rates of overweight and obesity, suggesting that diabetes may develop in Indians at a lower BMI than in Europeans [11].

3. POOR GLYCEMIC CONTROL AND DIABETICS

The micro and macrovascular alterations associated with diabetes are brought on by poor glycemic control, as seen in the diabetic population in India. They can put diabetic patients at risk for further complications such as diabetic myonecrosis and muscular infarction [12]. Although HbA 1c is the globally recognized gold standard test for identifying uncontrolled diabetic mellitus, it is not widely available in India. Furthermore, there is no "clinical inertia" to start insulin therapy in the clinical or patient communities. Europeans [13,14].

Weight gain, hypoglycemic events, and the fear of insulin injection are the most common insulin-related fears. Early detection and screening for pre-diabetes (especially in pregnant women, children, and adults with a BMI of 25) may result in better societal health outcomes [15]. Continuing medical education programs for primary care physicians may provide the "clinical inertia" required to kick-start program adherence. They may be a significant step toward achieving target glycemic levels and avoiding disease complications. Early insulin initiation, optimal doses of oral hypoglycemic agents, and appropriate lifestyle modification may also have long-term benefits in disease management [16,17]. The three most prevalent worries associated with insulin use are weight gain, hypoglycemia incidents, and insulin injection anxiety. Better societal health outcomes could be attained by early detection and screening for pre-diabetes (particularly in pregnant women, kids, and people with a BMI of 25)[18]. The "clinical inertia" needed to jump-start program adherence may be provided through continuing medical education programs for primary care providers. These programs may also be a key step toward achieving the goal of glycemic levels and preventing disease consequences [19,20]. Early insulin introduction, ideal oral hypoglycemic dosages, and suitable lifestyle changes may all help manage diseases over the long term[21].

4. MICROALBUMINURIA AND DIABETICS

Both elevated amounts in a spot sample (30-300 mg/L) and 24-hour urine samples (between 30-300 mg/24 hours) can be used to identify microalbuminuria. Over two to three months, both must be measured at least twice and ideally three times [22]. Macroalbuminuria, or just "albuminuria," is the term used to describe albumin levels that are higher than the upper limit. Sometimes, the upper limit value is given as one less to denote that the higher value (in this case, 300) is defined as macroalbuminuria (for example, 300 is given as 299) [23].

When collecting urine ACR samples to diagnose microalbuminuria, caution must be used. Taking a sample first thing in the morning [24]. At least 24 hours before the test, vigorous exercise should be avoided. The microbial albumin urine test should be repeated three to six months following the initial positive result. Finally, individuals with excessive or inadequate muscle mass perform poorly on the test. provided as 299)[24,25].

Changes in the muscle's creatinine levels are to blame for this. Normal albumin excretion is less than 30 mg/day (20 mcg/min); persistent albumin excretion between 30 and 300 mg/day (20 to 200 mcg/min) is referred to as moderately increased albuminuria (the new term for what was formerly known as "microalbuminuria") [26] and, in diabetic patients (especially type 1 diabetes), may be suggestive of early diabetic nephropathy, unless other renal disease is present [27–29]. Excretion rates of more than 300 mg/day (200 mcg/min) are considered to be severe albuminuria (the new terminology for what was formerly called "macroalbuminuria" and which is also called overt proteinuria, clinical renal disease, or dipstick positive proteinuria) [26,30,31].

The risk of developing overt diabetic nephropathy is most likely closely correlated with albumin excretion rates at all levels, even though these cut-offs for moderately and significantly elevated albuminuria aid in assessing the risk of nephropathy progression [23,32,33]. Proteinuria is the most typical sign of diabetic nephropathy. 31 ' 39 The initial symptom of this is microbial albuminuria. Urinary protein excretion typically occurs at a rate of 50 mg per day. Less than 20 mg of albumin is excreted on average each day [34,35]. Different proteinuria onset and progression rates are seen in diabetic kidney disease[36,37].

5. ROLE OF MICROALBUMINURIA

Microalbuminuria (20–200 g/min) initially appears and is sporadic. An albuminuria of 30–300 mg daily corresponds to a microalbuminuria of 20–200 g/min. Microalbuminuria 1, which persists is a sign of diabetic nephropathy. Stable renal function and slowed nephropathy progression may be achieved with adequate blood pressure and glucose control [38]. Using ACE inhibitors or ARB drugs

can help to stabilize or reverse microalbuminuria. The transition from normal to microalbuminuria appears to be halted by maintaining normal blood glucose levels[39,40].
Renal impairment is likely to worsen after clinical proteinuria appears (dipstick positive, >500 mg/L). (The total amount of proteins in the urine, or proteinuria, is more than the amount of albumin in the urine, or albuminuria. Albuminuria of 300 mg/L is nearly equal to proteinuria of 500 mg/L.) [41]. Renal function declines more quickly due to hypertension. Since ischaemic tissue damage contributes to most late complications of diabetes (retinopathy, neuropathy, heart disease, and peripheral arterial disease), treatment with human recombinant erythropoietin should be beneficial. Still, there is currently insufficient evidence to support this hypothesis [22,42,43].

The risk of developing a diabetic eye disease, heart disease, or a stroke is increased, according to a study, if you also have anemia. Patients with diabetes plus anemia are likelier to pass away at a younger age than diabetes patients alone[44]. Fortunately, anemia is treatable, and benefits, including improved energy, level of activity, and quality of life, are available. Correction of anemia lowers morbidity and mortality while improving energy, vitality, and overall quality of life. According to studies, treating anemia may delay the onset of various diabetes consequences, such as kidney, eye, and nerve damage. The course of treatment will depend on what causes the anemia [45]. It could be advised to take vitamin and iron supplements. Kidney disease-related anemia may require the width of the red blood cell distribution (RDW) to measure the variability of circulating erythrocyte size derived from standard automated complete blood counts. Red blood cell distribution width (RDW) has been reported to be a risk marker of morbidity and mortality for cardiovascular disease in various study populations (CVD) [46].

6. CONCLUSION

RDW has been proposed as a marker of both macrovascular and microvascular problems in diabetes. Numerous investigations on the connection between RDW and diabetic complications have been undertaken. 36' 38 According to certain research, people with diabetes with higher RDW readings have a higher chance of developing CVD and nephropathy. RDW may be a helpful clinical measure for problems associated with diabetes unaffected by conventional risk factors or disease duration. However, little study has been done on the connection between RDW and complications caused by diabetes. To ascertain whether there is a connection between RDW, nephropathy, neuropathy, and peripheral arterial disease (PAD) in type 2 diabetics, a study was conducted. In patients with type 2 diabetes, this study identified no connection between RDW, neuropathy, and PAD. More crucially, in a type 2 diabetes group, RDW was found to be substantially related to diabetic nephropathy.

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COMPETING INTEREST

The authors declare no conflict of interest.

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