International Scholars Journals

International Journal of Urology and Nephrology ISSN 2756-3855 Vol. 10 (3), pp.001-002, September, 2022. Available online at www.internationalscholarsjournals.com © International Scholars Journals

Perspective

Comprehensive study on the diabetic nephropathy and its treatment

John Francis*

Department of Nephrology, Medical University of Heidelberg, Heidelberg, Germany.

Received: 16-Aug-2022, Manuscript No. IJUN-22-75858; Editor assigned: 18-Aug-2022, PreQC No. IJUN-22-75858 (PQ); Reviewed: 02-Sep-2022, QC No IJUN-22-75858; Revised: 07-Sep-2022, Manuscript No. IJUN-22-75858 (R); Published: 16-Sep-2022

ABOUT THE STUDY

Diabetes mellitus patients who have diabetic nephropathy, also known as diabetic kidney disease, experience a continuous loss of kidney function. The most common causes of End-Stage Renal Disease (ESRD) and Chronic Kidney Disease (CKD) worldwide are diabetic nephropathy. Many kinds of CKD have the triad of protein leakage into the urine (proteinuria or albuminuria), rising blood pressure with hypertension, and finally declining renal function. When the glomeruli are damaged, protein loss in the urine can become substantial, resulting in low serum albumin levels and the so called nephrotic syndrome, which is characterized by broad body edoema. The same is true for the estimated Glomerular Filtration Rate (eGFR), which may gradually go from a normal of over 90 ml/min/1.73 m^2 to less than 15, at which point the patient is considered to have end-stage renal disease. Typically, it develops gradually over years. Long-term poorly managed blood glucose levels are typically when pathophysiologic problems in diabetic nephropathy start. The kidney's nephrons, which function as filters, then undergo a number of alterations. Each adult kidney has between 750,000 to 1.5 million nephrons on average. As nephrons become obsolete, there is initially constriction of the efferent arterioles and dilation of the afferent arterioles, which results in glomerular capillary hypertension and hyperfiltration. However, the adaptation of hyperfiltration paradoxically causes further shear stress-related damage to the delicate glomerular capillaries, which in turn causes further proteinuria, rising blood pressure, and a vicious cycle of further nephron damage and decline in overall renal function. Concurrently, the glomerulus itself undergoes modifications that include thickening of the basement membrane, enlargement of the slit membranes of the podocytes, expansion of the mesangial cell population, and expansion of the mesangial matrix. Kimmelstiel-Wilson nodules are deposits that are produced when this matrix invades the glomerular capillaries. The glomerulus might be gradually consumed by the mesangial cells and matrix, stopping filtration.

Treatment

Treatment aims to slow the development of kidney impairment and manage any associated consequences. Currently, diabetic nephropathy management focuses on four key areas: reduction of cardiovascular risk, management of blood sugar, management of blood pressure, and suppression of the RAAS system.

Cardiovascular risk reduction: Diabetes mellitus patients have a much higher risk of cardiovascular disease, which also puts them at risk for renal failure on its own. As a result, it's critical to actively control cardiovascular risk factors in diabetes mellitus patients, especially in those who have diabetic nephropathy. The basic strategies for controlling cardiovascular disease include quitting smoking, lipid-lowering medications (such as statins), regular exercise, and a nutritious diet. Since it does not need to be dose-adjusted based on GFR, atorvastatin is favoured over other statins in individuals with kidney disease.

Glycemic control: Improved glycemic management has been proven to have a significant impact on the clinical outcomes of individuals with diabetic nephropathy in numerous investigations. Additionally, other DM problems including retinopathy and neuropathy are less common when blood sugar levels are tightly controlled. In individuals with Type 1 DM, insulin is primarily used to maintain glycemic control; in patients with Type 2 DM, hypoglycemic medications and/or insulin are used. Studies found that achieving a target HbA1c concentration of 7% reduced the microvascular consequences of diabetic nephropathy. In most individuals, further lowering of the HbA1c is not advised because it may raise the risk of

*Corresponding author. John Francis, E-mail: Johncis03@yahoo.com.

hypoglycemia episodes and did not correlate with better outcomes.

Blood pressure control: In individuals with diabetic nephropathy, lowering systolic blood pressure to less than 140 mmHg has been shown to be beneficial in numerous randomized clinical trials. Microalbuminuria development is accelerated with high blood pressure, as is the progression of proteinuria and renal function decline. Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers are very beneficial in lowering blood pressure and slowing the progression of nephropathy in diabetic individuals. It has been demonstrated that more aggressive blood pressure control (125–130/80) in diabetes mellitus patients reduces the chance of developing diabetic nephropathy as well as other diabetic complications. Calcium channel blockers or diuretics are good second-line options when some patients might need dual medication to effectively regulate pressure.

AAS inhibition: Several treatments, primarily ACE inhibitors, angiotensin receptor blockers, direct renin inhibitors, and mineralocorticoid antagonists, can be used to achieve inhibition. The most efficient treatment for reducing the progression of diabetic nephropathy at all stages has been shown to be RAAS inhibition. The danger of adverse effects (such as hyperkalemia and acute renal injury) outweighs any potential advantages, even if RAAS blockage using multiple agents may further reduce proteinuria. Therefore, it is advised that individuals with DM who have hypertension, any indication of microalbuminuria, or diabetic nephropathy utilize just one medication.