Editorial

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Journal of Clinical & Experimental Nephrology
ISSN 2472-5056

2021
Vol.6 No.6: e114

A Brief Note on Diabetic Nephropathy

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Received date: December 8, 2021; **Accepted date:** December 22, 2021; **Published date:** December 29, 2021

Citation: Zakir A (2021) A Brief Note on Diabetic Nephropathy. J Clin Exp Nephrol Vol.6 No.6: e114.

Editorial Note

Diabetic nephropathy, also called as diabetic kidney disease, is the loss of kidney function that occurs over time in diabetes patients. Diabetic nephropathy is a major cause of Chronic Kidney Disease (CKD) and End-Stage Renal Disease (ESRD) all over the world. Protein loss in the urine owing to glomeruli destruction can become vast, resulting in a low serum albumin level, which can lead to generalised body swelling (edoema) and the nephrotic syndrome. Similarly, the estimated Glomerular Filtration Rate (eGFR) may gradually decrease from a normal of over 90 ml/min to less than 15, indicating that the patient has end-stage renal disease. It normally progresses slowly over time.

Diabetic nephropathy's pathophysiologic problems begin with poorly managed blood glucose levels over a long time. Multiple changes in the filtration units of the kidneys, the nephrons, follow. (Each adult kidney contains approximately 750,000-1.5 million nephrons). The efferent arterioles constrict and the afferent arterioles dilate, resulting in glomerular capillary hypertension and hyperfiltration kind of, which gradually transforms to hypofiltration with time. There are also changes within the glomerulus, such as a thickening of the basement membrane, a widening of the podocyte slit membranes, an increase in the number of mesangial cells, and an increase in the amount of mesangial matrix.

The matrix infiltrates the glomerular capillaries, resulting in Kimmelstiel-Wilson nodules. The mesangial cells and matrix can grow and consume the entire glomerulus, thus shutting down filtration. The amount of protein in the urine - proteinuria - and a blood test called serum creatinine can both be used to assess the condition of diabetic nephropathy. The degree of damage to any still-functioning glomeruli is reflected in the amount of proteinuria. The estimated Glomerular Filtration Rate (eGFR), which measures the percentage of glomeruli that are no longer filtering the blood, can be calculated using the serum creatinine measurement.

Treatment with an angiotensin converting enzyme inhibitor or angiotensin receptor blocker, which dilates the arteriole entering the glomerulus and lowers blood pressure within the glomerular capillaries, slowing disease progression. GLP-1 agonists, DPP-4 inhibitors, and SGLT2 inhibitors are three types of diabetes drugs that are hypothesised to decrease the progression of diabetic nephropathy.

Symptoms appear 5 to 10 years after the disease has started. Frequent urination at night, often known as nocturia, is a common initial sign. Tiredness, headaches, a general sensation of illness, nausea, vomiting, frequent daytime urination, a lack of appetite, itchy skin, and leg edoema are among the other symptoms. Proteinuria (protein in the urine), hypertension, and progressive kidney function loss characterize the clinical picture of Diabetic Nephropathy (DN). Because the process can be slow at first, regular monitoring for diabetic nephropathy in patients with diabetes is critical.

The following are the symptoms of for diabetic nephropathy. Blood glucose management is poor. Unrestrained high blood pressure and Type 1 diabetes mellitus is a type of diabetes that develops before the age of 20. A family history of diabetic nephropathy has been established, and specific genes have been linked to the disease. Hyperfiltration, microalbuminuria, nephrotic proteinuria, and progressive chronic kidney disease leading to End-Stage Renal Failure are all clinical stages of diabetic nephropathy (ESRD). The glomerulus, renal tubules, vasculature (afferent and efferent renal arterioles), and interstitium are all damaged components of the kidney. The final common route of DN is renal fibrosis. Multiple factors contribute to fibrosis, including changes in renal hemodynamics, aberrant glucose metabolism linked to oxidative stress, inflammatory processes, and an overactive Renin-Angiotensin-Aldosterone System (RAAS).

Diabetic nephropathy is hypothesised to be caused by a complex combination of hemodynamic and metabolic variables. An elevation in systemic and intraglomerular pressure, as well as over-activation of the RAAS, is hemodynamic factors. Various factors have been demonstrated to stimulate the RAAS, which is one of the most critical channels in diabetic nephropathy pathogenesis, in the presence of diabetes. The sodium-glucose cotransporter 2, which cotransports sodium and glucose back into circulation, is up-regulated in the proximal tubules as a result of the greater load of filtered glucose. The supply of sodium chloride to the macula densa in the distal tubules is reduced as a result, increasing the release of renin and over-activating RAAS. One of the first characteristics of DN is hyperfiltration.