Editorial

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Editorial Note for Pathophysiology of Diabetic Nephropathy

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Editorial Note

Diabetic Nephropathy remains the leading cause of ESRD in western countries. It is considered as microvascular complication occurring in both Type 1 DM and Type 2 DM. 30%-40% of patients with diabetes are prone to develop Diabetic nephropathy. In clinical practice, some of the individuals suffering from diabetes are not tend to develop diabetic kidney disease, even if their glucose is not strictly controlled. However some individuals with perfect glycemic conditions tend to develop ESRD.

Diabetic Nephropathy also called as “Diabetic kidney disease”, is classically defined as the progressive loss of kidney function and a slow increase in albuminuria. In the earliest stages of diabetic nephropathy there is a noticeable hyper filtration, where the glomerular filtration rate is higher than the normal. Later in these disease there is a decrease in the estimated glomerular filtration rate (eGFR) However this factor cannot be used for early diagnosis, as it is not same in case of all the DM patients. Persistent albuminuria is considered as the sign of diabetic nephropathy. In the initial stages the patient suffers from “microalbuminaria” where small amounts of urine are leaked in the urine. Later it is progressed to a stage known as where the urine albumin excretion is sufficiently high. This change is the result of hypertension, and progressive reductions in kidney function. The progression can be slowed down or stopped by, intensive glycemic control, optimization of blood pressure (BP), and the use of renal protective drugs.

The common pathway involved in the pathogenesis of DN is renal fibrosis. It is the result of many changes in the kidney that includes glucose metabolism, renal hemodynamic changes and ischemia associated with increase of oxidative stress, inflammatory effects as well as circulation of AGE’s. These causes some functional changes in the nephron like glomerular hyper filtration, glomerular hypertension. The structural changes involve deposition of extra-cellular matrix in the mesangium, glomerular basement membrane thickening, tubular atrophy results in glomerulosclerosis and renal fibrosis.

Renal hemodynamic changes: Hyper filtration is the clinical symptom observed in incipient/early diabetic nephropathy. Hyper filtration is the result of both, that is afferent arteriole dilation and efferent arteriole constriction. RAAS system is involved in this mechanism. Renin is the hormone that is released when there is low blood flow to the kidney. Renin performs a cascade of mechanisms in order to maintain optimal blood pressure. Renin is indirectly involved in the vasoconstriction through RAAS system. The function of renin is to act in case of low blood pressure but the hyperglycemic conditions in diabetes results in over activation of RAAS system.

- Dilation of afferent arteriole and constriction of efferent arteriole leads to increased GFR that is hallmark or symptom of early/incipient Diabetic Nephropathy.
- Increased blood pressure state in the glomerulus leads to mesangial expansion. The mesangial cells in response to damage releases cytokines as well as oxygen free radicals that leads to endothelial dysfunction.
- All this results in hypertrophy, accumulation of mesangial matrix and podocyte injury.

Ischemia or cell death: In DKD, oxygen supply is reduced that causes renal medulla hypoxia and renal tubular dysfunction and results in death of the nephron. The residual nephrons present demand for more oxygen supply. Without supply of adequate of adequate oxygen more number of oxygen free radicals are produced and results in renal tissue destruction. In terms of defence, hypoxia inducible factor (HIF) is increased to handle the hypoxic state, but hyperglycemia here disturbs the stability of HIF leading to cellular atrophy.