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Perspective

Complications and Pathophysiology of Diabetic Kidney Disease

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DESCRIPTION

In the medical literature, the words "Diabetic kidney disease" (DKD) and "Diabetic Nephropathy" (DN) have both been used frequently to refer to kidney disease brought on by diabetes, but their precise meanings have not yet been agreed upon. The Kidney Disease Outcomes Quality Initiative (KDOQI) clinical practice guidelines proposed in 2007 to designate DKD as a cause cell of kidney damage due to diabetes and to use the term "diabetic glomerulopathy" to refer to biopsy-proven kidney disease caused by diabetes. This modification reflects the diversity of DKD's clinical course and manifestation. Particularly in type 1 diabetes, DN was traditionally seen as a progression of stages.

Glomerular hypertrophy and hyper filtration, which are indicated by increase in kidney size and Glomerular Filtration Rate, make up the first phase (GFR).

Pathophysiology

DKD has a complicated and multifaceted etiology that includes both metabolic and hemodynamic variables. These cause dysregulated autophagy, oxidative stress, hypoxia, activation of intracellular signaling pathways, and epigenetic alterations, which lead to fibrosis and inflammation of the kidneys. Recent research has shown that two classes of anti-diabetic medications, sodium-glucose co-transporter 2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists, protect Reno in addition to decreasing blood sugar levels. There are also other more medicinal compounds being created and tested in clinical studies.

The development and progression of diabetic kidney disease (DKD) are influenced by numerous pathophysiological disturbances; from a therapeutic perspective, this multifactorial pathogenic process suggests that the disease will need to be treated with a combination of various medications. The main causes of DKD development are hyperglycemia and hypertension, although once the disease has already started, even strict glycemic and/or hypertension control may not be able to stop or delay the disease's course.

A powerful vasoconstrictor peptide called ET-1(Endothelin Receptors) is mostly released by endothelial cells. It functions by way of ET-A and ET-B receptors. Patients with diabetes have higher circulating levels of ET-1, which may play a role in the pathogenesis of DKD. An ET-A receptor antagonist was shown in a preclinical investigation to decrease albuminuria and improve glomerulosclerosis in streptozotocin-induced diabetic rats. Avosentan was added to typical Renin-Angiotensin-Aldosterone System (RAAS) blocking in a clinical research for short-term proof-of-concept, and this reduced albuminuria in DKD patients. A phase 3 clinical trial that followed, however A Study of Cardiovascular Events in Diabetes (ASCEND), was prematurely stopped due to an increase in cardiovascular events brought on by fluid overload.

Complications

Diabetic nephropathy complications can appear gradually over months or years. They may consist of:

- Fluid retention, which can cause fluid build-up in the lungs, high blood pressure, and swelling of the arms and legs (pulmonary edema)
- An increase in blood potassium levels (hyperkalemia)
- Cardiovascular disease, which can result in a stroke, is a condition of the heart and blood vessels.
- Injury to the blood vessels in the tissue at the rear of the eye that is light-sensitive (diabetic retinopathy)
- Less red blood cells are available to carry oxygen (anemia)
- Diarrhoea, erectile dysfunction, foot ulcers, and other issues resulting from nerve and blood vessel damage
- Bone and mineral abnormalities brought on by the kidneys' inability to keep the blood's calcium and phosphorus levels in balance.
- Obstetrical issues that put both the mother and the foetus at risk.
- End-stage renal disease, which causes irreversible kidney damage and eventually requires dialysis or a kidney transplant to survive.

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