Aggravating role of inflammation in diabetic kidney disease

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Introduction

Many chronic illnesses are currently in pandemic proportions and gradually become a main cause of morbidity and mortality throughout the world. Diabetes mellitus, particularly type 2 diabetes, plays a major role in this hindrance, while diabetic complications being a very crucial public health issue. One of the most important medical concerns of the diabetic patients is diabetic kidney disease. Diabetic kidney disease is the leading cause of end-stage kidney disease worldwide (1,2). Therefore, in an effort to find novel therapeutic approaches that permit the prevention and retardation of diabetic kidney disease, innovative perception into the pathophysiology of diabetic kidney disease is needed. Hemodynamic modifications like hyperfiltration and hyperperfusion imposed by hyperglycemia are believed as the main renal damage factors, however such changes are only one feature of a complex series of pathophysiological modifications related to the presence of glucose metabolism defects. In fact, the most difficult problem in nephrology is the persistent and progressive expansion in patients with end-stage kidney failure worldwide (1-4). The influence of diabetic kidney disease on the increasing population with chronic renal failure and end-stage kidney failure is enormous. It was detected that, three main pathways showing abnormality of intracellular metabolism have been recognized in the expansion of diabetic kidney disease; the creation of advanced glycation end-products and the activation of polyol and PKC (protein kinase C) pathways and finally, intra-glomerular hypertension induced by glomerular hyperfiltration (1-3).

Materials and Methods

This paper intended to discuss on mechanisms of inflammation in diabetic kidney disease. For this paper we searched; Directory of Open Access Journals (DOAJ), Google Scholar, PubMed, EBSCO and Web of Science were searched with key words relevant to diabetic kidney disease, acute renal failure, end-stage renal disease, type 2 diabetes mellitus, inflammation, type 1 diabetes mellitus and diabetic nephropathy.

Diabetic kidney disease

Diabetic kidney disease take place nearly one-third of individuals with type 1 diabetes mellitus and 25% approximately of patients with type 2 of this disease. Additionally, diabetic kidney disease is associated with heart and vessel diseases, and enhances mortality of diabetic individuals. In recent years, the concept of the pathophysiological processes that lead to diabetic kidney disease has particularly improved on a genetic and molecular level (2-5). Recent investigations have shown that renal inflammation is essential in stimulating the development and evolu-
tion of diabetic kidney disease. Inflammation may be a key factor which is triggered by the metabolic, biochemical, and hemodynamic imbalances known to exist in the diabetic nephropathy. In fact, diabetic kidney disease is traditionally believed to be a nonimmune disease, however, many evidences currently signifies that immunologic and inflammatory mechanisms play a substantial role in its development and aggravation (1,4). It was detected that, various cells, consisting macrophages, leukocytes and monocytes, and also other molecules, like adhesion molecules (intercellular adhesion molecule-1 (ICAM-1)), chemokines (monocyte chemoattractant protein-1), various enzymes (cylooxygenase-2, nitric oxide synthase), some growth factors (vascular endothelial growth factor, growth hormone, IGF, TGF-β), and nuclear factors (NF-κB), are interacted in this processes related to diabetic kidney disease (4-6). While, type 2 diabetes mellitus is not an immune disease, however at this time we could envisage that the combined of immunologic process and inflammatory mechanisms both play a crucial role in its starting, development and eventually its progression.

Factors responsible for progression of diabetic kidney disease

It is well-defined the diabetic kidney disease is extended firstly, with the mesangial extracellular matrix accumulation. The progression of diabetic kidney disease contains of three steps; hypertrophy of the glomeruli and hyperfiltration, then inflammation tubulointerstitial region and damage to the glomeruli and accordingly reduction of cell proportion by apoptosis and mesangial extracellular matrix accumulation (3-5).

Inflammation aggravates diabetic kidney disease

Inflammation plays an indispensable role in the expansion of diabetic kidney disease. This process started by increasing chemokine construction, infiltration of various inflammatory cells to the kidney, pro-inflammatory cytokine creation and renal injury. Recent investigations have detected that inflammation, and more specifically pro-inflammatory cytokines, play a basic role in the extension of microvascular diabetic complications. Recent studies have shown that, mesangial, glomerular, tubular epithelial and endothelial, cells can produce pro-inflammatory cytokines. Notably, these molecules have been related to substantial kidney influences. Interleukin-1 intensifies vascular endothelial permeability and has been complicated in the proliferation of mesangial cells and matrix synthesis, and also in the enhancement of intra-glomerular microvascular disturbances (2-5). Various components of the diabetic condition, such as hyperglycemia, renin-angiotensin system and oxidative stress are able to activate the inflammatory process in the kidneys, which leads to renal infiltration by lymphocytes and monocytes which produce injurious molecules, like reactive oxygen species (ROS) and pro-inflammatory cytokines. This leukocyte activity intensifies the inflammatory response and promotes cell damage and initiation of interstitial fibrosis (4-7).

Conclusion

Diabetic kidney disease is a complication that happens in some people with diabetes. It can extend to chronic renal failure in some individuals. Treatment modalities aim to inhibit or delay the development of the disease. Exact understanding of the inflammatory response in diabetic nephropathy is anticipated to recognize new anti-inflammatory approaches for the potential treatment of diabetic kidney disease.

Author's contribution

HN was the single author of the paper.

Conflicts of interest

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

Funding/Support

None.

References

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