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Diabetes Leading to Kidney Disease: A Review

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ABSTRACT: Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia, and it represents a significant risk factor for the development and progression of kidney disease. This review aims to elucidate the complex interplay between diabetes and kidney disease, highlighting the underlying mechanisms, risk factors, diagnostic approaches, and therapeutic interventions.

KEYWORDS: Diabetes mellitus, kidney function, hyperglycemia, hemodynamic, renalfunction

INTRODUCTION

Diabetic nephropathy (DN) is a serious complication of diabetes mellitus and remains the leading cause of end-stage renal disease (ESRD) globally[(1)]. It is characterized by progressive kidney damage due to long-standing uncontrolled diabetes[(2)].
DKD, also known as diabetic nephropathy, is a microvascular complication of diabetes that affects the kidney's structure and function. It develops progressively over time and significantly contributes to morbidity and mortality among individuals with diabetes. Despite advances in diabetes management, the prevalence of DKD remains high, posing a substantial burden on healthcare systems and society as a whole.

Diabetes is a significant risk factor for the development of kidney disease[(3)]. The chronic hyperglycemia associated with diabetes can lead to damage of the small blood vessels in the kidneys[(2)], ultimately resulting in diabetic nephropathy[(4)]. This condition is characterized by proteinuria, progressive decline in renal function, and an increased risk of cardiovascular events[(5)]. Early detection and management of diabetes, along with tight glycemic control and blood pressure management, are crucial in preventing or delaying the onset and progression of diabetic kidney disease[(6)].

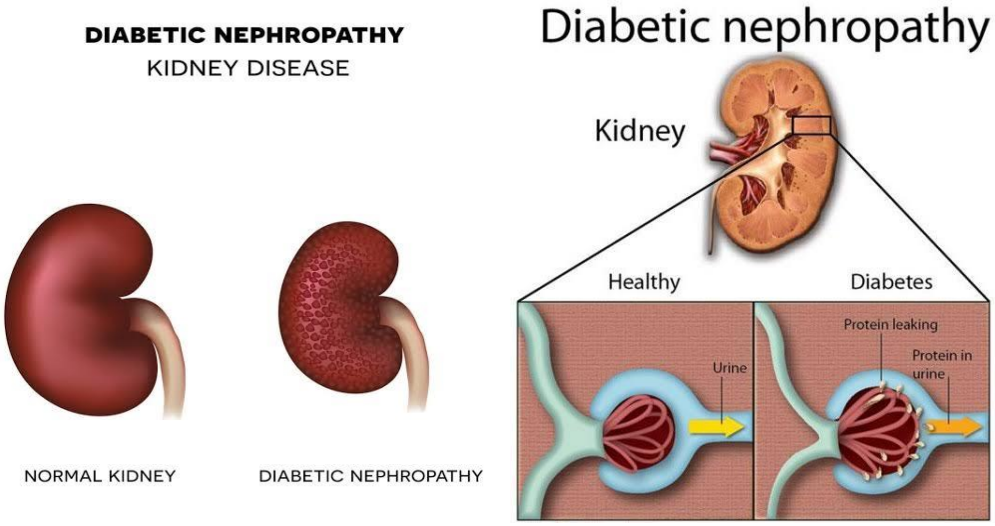

Figure No. 1: Diabetic Nephropathy

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ETIOLOGY

Diabetic neuropathy multifactorial, involving hyperglycemia-induced metabolic and hemodynamic changes[7]. Chronic hyperglycemia leads to the activation of various pathways such as the polyol pathway, advanced glycation end products (AGEs) formation, protein kinase C (PKC) activation, and increased hexosamine pathway flux, ultimately contributing to renal injury[8].

EPIDEMIOLOGY

DN affects approximately 20-40% of individuals with type 1 diabetes and 10-20% of those with type 2 diabetes[9]. Its prevalence is expected to rise due to the increasing incidence of diabetes worldwide[10].

CLINICAL MANIFESTATIONS

Early stages of DN are often asymptomatic. However, asthe disease progresses, clinical manifestations become evident. These include persistent proteinuria, hypertension, declining glomerular filtration rate (GFR), and eventually, signs of kidney failure such as fluid retention and uremia[5].

SIGNS AND SYMPTOMS

The signs and symptoms of DN vary depending on the stage of the disease. Patients may present with edema, especially in the lower extremities, due to fluid retention caused by impaired kidney function[6]. Additionally, they may experience fatigue, nausea, vomiting, and pruritus as the kidney function declines[11].

PATHOPHYSIOLOGY

Chronic hyperglycemia in diabetes leads to the activation of multiple pathways, including the polyol pathway, advanced glycation end-products(AGEs) formation, proteinkinase C (PKC) activation, and increased hexosamine pathway flux, contributing to kidney damage.

Hyperglycemia:

Chronic hyperglycemia leads to increased production of advanced glycation end-products (AGEs), which accumulate in the kidneys and contribute to tissue damage[12].

Hemodynamic changes:

Hyperglycaemia causes glomerular hyperfiltration, leading to increased intraglomerular pressure and subsequent damage to the glomerular filtration barrier[13].

Renin – angiotensin - aldosterone system (RAAS) activation: Chronic hyperglycemia activates the RAAS, leading to renal vasoconstriction, inflammation, and fibrosis[2].

Inflammation and oxidative stress:

Hyperglycemia triggers the release of pro-inflammatory cytokines and reactive oxygen species (ROS), leading to endothelialdysfunction, inflammation, and tissue damage[14].

Extracellular matrix accumulation:

Increased production of extracellular matrix proteins, such as collagen and fibronectin, leads to glomerular and tubulointerstitial fibrosis, impairing kidney function[15].

Genetic predisposition:

Certain genetic factors predispose individuals with diabetes todevelop DKD, including polymorphisms in genes involved in glucose metabolism, inflammation, and renal function.

DIAGNOSIS

Diabetic kidney disease involves several tests, including urine albumin – to – creatinine ratio (UACR), estimated glomerular filtration rate (eGFR), and kidney biopsy in some cases[15]. Diabetic nephropathy progresses through various stages, starting with microalbuminuria (a small amount of protein in the urine), followed by macroalbuminuria (a large amount of protein in the urine), and eventually leading

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toend-stage renal disease (ESRD), requiring dialysis or kidney transplantation[2].

TREATMENT

The treatment of diabetic kidney disease typically involves a multifaceted approach aimed at controlling blood sugar levels, managing blood pressure, and addressing other risk factors such as high cholesterol. Common treatments include:

Blood sugar control:

Tight control of blood sugar levels through diet, exercise, oral medications, or insulin therapy can help slow the progression of kidney disease in diabetic[2].

Blood pressure management:

Controlling high blood pressure is crucial in managing diabetic kidney disease. Medications such as angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) are often prescribed to protect the kidneys[3].

Medications to reduce proteinuria:

Drugs like angiotensin receptor blockers (ARBs) or angiotensin-converting enzyme (ACE) inhibitors can help reduce proteinuria (excess protein in the urine), which is a sign of kidney damage[16].

Cholesterol management:

Statin medications may be prescribed to manage cholesterol levels, which can help protect the kidneys in people with diabetes[17].

Lifestyle modifications:

Lifestyle changes such as maintaining a healthy weight, quitting smoking, limiting alcohol intake, and regular exercise can also play a significant role in managing diabetic kidney disease[6].

Regular monitoring:

Close monitoring of kidney function through blood tests and urine tests is essential to assess the progression of kidney disease and adjust treatment as needed. It's important for individuals with diabetes to work closely with their healthcare team to develop a personalized treatment plan tailored to their specific needs and medical history[6].

CONCLUSION

The conclusion that can be drawn from diabetes leading to kidney disease is that individuals with diabetes are at a higher risk of developing kidney complications, known as diabetic nephropathy. This underscores the importance of managing diabetes effectively to reduce the risk of kidney complications through lifestyle changes, medication adherence, and regular medical monitoring. Early detection and intervention are crucial in preventing or slowing the progression of kidney disease in diabetic patients**.**

REFERENCES

1. Ritz E, Zeng XX, Rychlik I. Clinical manifestation and natural history of diabetic nephropathy. Contrib. Nephrol. 2011; 170: 19-27. Doi: 10.1159/000324936.
2. Alicic RZ, Rooney MT, Tuttle KR. Diabetic Kidney Disease: Challenges, Progress, and Possibilities. Clin J Am Soc Nephrol. 2017;12(12):2032-2045. Doi: 10.2215/CJN.11491116.
3. American Diabetes Association. (2021). Standards of Medical Care in Diabetes –2021. Diabetes Care, 44(Supplement 1), S1-S232.
4. KDOQI. (2020). KDOQI Clinical Practice Guideline for Diabetes and CKD: 2020 Update. American Journal of Kidney Diseases, 76(1), S1-S91.
5. De Boer, I. H., & Rue, T. C. (2021). Diabetic Nephropathy and Cardiovascular Disease. Current Diabetes Reports, 21(7), 28.
6. Tuttle, K. R., Bakris, G. L., Bilous, R. W., Chiang, J. L., de Boer, I. H., Goldstein-Fuchs, J., ... & Molitch, M. E. (2014). Diabetic kidney disease: a report from an ADA Consensus Conference. Diabetes Care, 37(10), 2864-2883.
7. Kanasaki K, Kitada M, Koya D. Pathophysiology of Diabetic Nephropathy: Experimental Findings and Therapeutic Implications. J Diabetes Res. 2017;2017:6078136. Doi: 10.1155/2017/6078136.

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8. Fioretto P, Mauer M. Histopathology of diabetic nephropathy. Semin Nephrol. 2007;27(2):195-207. Doi: 10.1016/j.semnephrol.2007.01.008.
9. Molitch ME, DeFronzo RA, Franz MJ, et al. Nephropathy in diabetes. Diabetes Care. 2004;27 Supply 1:S79-83. Doi: 10.2337/diacare.27.2007.s79.
10. Thomas MC, Brownlee M, Susztak K, et al. Diabetic kidney disease. Nat Rev Dis Primers. 2015;1:15018. Doi: 10.1038/nrdp.2015.18.
11. Kramer HJ, Nguyen QD, Curhan G, Hsu CY. Renal insufficiency in the absence ofalbuminuria and retinopathy among adults with type 2 diabetes mellitus. JAMA. 2003; 289(24): 3273-3277. Doi: 10.1001/jama.289.24.3273.
12. Kanasaki, Keizo, and George L. King. "The role of advanced glycation end- products in the progression of diabetic nephropathy." Experimental diabetesresearch 2011 (2011).
13. Fioretto, Paola, et al. "Molecular mechanisms and clinical pathophysiology ofdiabetic nephropathy." Journal of the American Society of Nephrology 13.10 (2002): 2614-2625.
14. Sharma, K., et al. "Diabetic kidney disease: the case for transforming growthfactor-β as a key mediator." Diabetes 44.10 (1995): 1139-1146.
15. Gnudi, Luigi, et al. "Mechanisms of tissue injury in diabetic nephropathy." Journalof the American Society of Nephrology 27.4 (2016): 822-829.
16. Heerspink, H. J. L., & Perkins, B. A. (2019). Glucose Targets for Diabetic Kidney Disease: Time to Adjust Treatment Paradigms? Diabetes Care, 42(6), 989–992.
17. Colhoun, H. M., & Marcovecchio, M. L. (2019). Biomarkers of diabetic kidney disease. Diabetologia, 62(6), 1015–1025. Alicic, R. Z., Rooney, M. T., & Tuttle, K. R.(2017).

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