FREQUENCY OF RETINOPATHY IN PATIENTS WITH DIABETES WITH AND WITHOUT DIABETIC NEPHROPATHY.

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Abstract

Background:- A common pathophysiological mechanism has been suggested as the link between diabetic retinopathy and diabetic nephropathy. However, if patients do not have diabetic retinopathy, it is very challenging to distinguish diabetic nephropathy from other glomerular disorders; the only option to do this is to take renal biopsies.

Study Objective:- To comprehend the current argument concerning the frequency of retinopathy in patients with diabetes with and without diabetic nephropathy.

Material and Methods:- The current study is based on a meta-analysis study design carried out on secondary published research. A midline search of the literature between 2017 and 2023 was conducted. 37 articles with related content were chosen from this search.

Conclusion:- Patients with and without diabetic retinopathy exhibit various clinical and biochemical characteristics of diabetic nephropathy. The relationship between the severity of diabetic retinopathy and the severity of diabetic nephropathy and neuropathy can be used to anticipate the progression of chronic renal disease as well as the neurological repercussions in diabetic patients.

Keywords: - Diabetes, Diabetic Nephropathy, Diabetic Retinopathy, Diabetic Neuropathy.

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Introduction

The clinical indicator of type 2 diabetes mellitus, a chronic endocrine disorder associated with a variety of metabolic issues, is hyperglycemia. The International Diabetes Federation (IDF) recently released the most recent Diabetes Atlas, which estimates that 462 million persons worldwide between the ages of 21 and 78 have diabetes. By 2045, there will be 700 million people worldwide who have diabetes mellitus (DM). Diabetes mellitus is a collective term for a group of typical metabolic disorders with the phenotype of hyperglycemia. A clinical indicator of diabetic nephropathy (DN) is the start of microalbuminuria, which progresses to macroalbuminuria, a steady fall in glomerular filtration rate, and ultimately end-stage renal disease. Although 10–30% of persons with type 1 and type 2 diabetes mellitus experience a decline in renal function before the onset of microalbuminuria or macroalbuminuria, this has recently offered a challenge to the conventional course of DN.

Diabetes frequently has a side effect called diabetic retinopathy (DR). Chronic illness, insufficient glycemic management, and hypertension are the main risk factors. The large variation in risk, however, suggests that additional variables, like as genetic predisposition or glycemic variability, are essential in explaining the susceptibility to DR development. Another important concept is the independent predictive value of DR for microvascular and macrovascular issues. As a result, DR should be included while evaluating the cardiovascular risk of a diabetic person.

A complicated interplay among hereditary and environmental factors leads to the emergence of several distinctive types of diabetes. Retinopathy, nephropathy, and neuropathy are three of diabetes mellitus (DM) main microvascular repercussions. Proliferative diabetic retinopathy and diabetic macular edema, which are more advanced and vision-threatening phases of diabetic retinopathy, move progressively from non-proliferative diabetic retinopathy (NPDR), which is milder. Poor glycemic management and systolic hypertension are risks associated with both diabetic retinopathy and diabetic nephropathy. DM is categorized based on the pathogenic process that generates hyperglycemia. Type 1 diabetes is characterized by a complete or almost complete lack of insulin and is brought on by immune system attacks on the beta cells that produce it. The diverse collection of diseases known as type 2 diabetes are characterized by varying degrees of insulin resistance, decreased insulin secretion, and increased hepatic glucose production.

Depending on the underlying cause of diabetes, factors that increase blood sugar levels include impaired insulin secretion, decreased glucose absorption, and increased glucose synthesis. Both the diabetic and the healthcare system are heavily burdened by the secondary pathophysiologic changes in various organ systems brought on by the metabolic abnormalities associated with DM. It can be challenging to distinguish between diabetic nephropathy and membranous nephropathy in patients with proteinuria when there is no evidence of diabetic retinopathy. This is especially true when there are no facilities for renal biopsies or when a renal biopsy is not an option because of bleeding tendencies. In order to uncover some clinical cues for differentiating diagnoses, we carried out the current investigation contrasting pure diabetic nephropathy with membranous nephropathy without retinopathy (1). To predict DN or DR incidence risk in T2DM patients, the DN or DR incidence risk nomogram considers illness history, TGs, PBG, BMI, BUN, and SBP (2).

BUN is the main renal excretion and nitrogen-containing waste product of human protein metabolism. BUN will increase if the glomerulus' ability to filter blood is impaired. BUN is used to evaluate the health of the kidneys (2). The onset of diabetes and its consequences are strongly correlated with HbA1c, according to a number of prospective studies. A key tool for assessing glycemic state is HbA1c. The International Expert Committee (IEC) recommended a HbA1c level of 6.5% in order to diagnose diabetes; notably, their recommendation was based on the likelihood that individuals with HbA1c levels of 6.3% or more have a noticeably increased risk of retinopathy in comparison to those whose HbA1c falls below that threshold. Diabetic nephropathy is one prominent microvascular side consequence of DM. Diabetic nephropathy affects fewer than 20% of type 2 diabetics and 20% to 30% of type 1 diabetics in the US. Type 2 diabetes, which is more frequent than type 1 diabetes and accounting for around 60% of patients with diabetic end-stage renal disease, is less common than diabetic nephropathy, although being less common (3).

Many patients with diabetic retinopathy who needed PRP also developed diabetic nephropathy during long-term follow-up. The most important characteristic of advanced nephropathy was higher HbA1c, which may indicate that patients' blood

sugar levels are out of control. Ophthalmologists should carefully examine controlling blood glucose and the development of diabetic nephropathy because these conditions are linked to a higher incidence of ocular problems. It is important to get medical advice right away, especially for male patients with high HbA1c values and extended follow-up after PRP.

The prevalence of non-diabetic renal disease varies significantly across geographic regions and due to various renal biopsy selection criteria. According to recent research analyzing the histology data, about one-third of renal biopsies taken from diabetes patients reveal pure diabetic nephropathy, one-third a nondiabetic state, and another third diabetic nephropathy with a superimposed illness. However, due to the prevalence of aggravating medical comorbidities and the heterogeneity of clinical presentations in this population, it is difficult to differentiate between DN and NDRD in specific patients without the use of a renal biopsy. Based on clinical and laboratory data, it will be determined whether persons with type 2 diabetes are more likely to have NDRD with or without DN than isolated DN (4).

People with type T2DM and kidney disease can use DR to diagnose DN, but DN may not always be present when DR is severe (5). Compared to individuals with DN, those with NDRD demonstrated considerably better proteinuria and renal function after receiving systemic treatments with glucocorticoids, immunosuppressive drugs, cytotoxic agents, blood pressure-lowering medications, and lipids. When compared to patients with NDRD, patients with DN did not significantly improve their renal function, and renal survival was also observed to be poorer in these patients (6). The pathological analysis of renal biopsies is currently the highest standard for DN diagnosis. However, renal biopsy is an invasive procedure with a variety of disadvantages. Notably, it can be challenging to perform a kidney biopsy in a setting run by non-specialists. In this investigation, 213 people with T2DM complicated by CKD had their clinical features and pathological kidney types identified. We also created a model for DN and NDRD differential diagnosis in T2DM patients. This model could be a resource for medical practitioners working in facilities without kidney biopsy technology. A common microvascular adverse effect of T2DM is DR.

It is well known that people with T2DM who frequently experience DR also develop DN, and that those who do not frequently experience DR have a higher likelihood of developing NDRD. The importance of proteinuria in the development of DN was initially underlined by Mogensen and Christensen in the 1970s. They also understood that the development of microalbuminuria is a distinct and early sign of DN. Later research revealed that proteinuria rises concurrently with kidney damage in type 1 diabetic nephropathy (DN) and that 10-15% of T2DM patients experience end-stage renal disease (ESRD) and nephropathy-level proteinuria after being diagnosed with diabetes. The general consensus is that DN is difficult to reverse, and continued treatment focuses on halting the spread of the illness. Several NDRDs, however, such as minimal change disease, immunoglobulin A (IgA) nephropathy, and membranous nephropathy, are typically manageable and even curable. The differential diagnosis is essential because the treatment and prognosis for DN and NDRD are quite distinct. Due to the prognostic and therapeutic implications, any concomitant glomerular illnesses or other renal disorders that coexist with DN must be identified, diagnosed, and treated.

The presence of DR indicates that the diabetic milieu has already harmed the microcirculation, making it a valid biomarker of the negative effects of diabetes in a particular person. Actually, one of the most significant signs for designing a tailored prescription is this. DR and complications of diabetes, such as micro- and macrovascular diseases and events, are clearly linked, according to recent systematic research. The most important side effect of diabetic neuropathy is diabetic foot syndrome. Diabetic foot syndrome is the most common reason for hospitalization among all DM complications. Diabetic neuropathy increases the risk of limb amputation by 1.7 times compared to DM patients without neuropathy; motor neuropathy-induced foot deformities also increase the risk of amputations by 12 times; and diabetic foot ulcers increase the risk of amputations by 36 times.

Discussion

Significant risk factors or predictors of DR include nephropathy, uncontrolled diabetes, HbA1C level, hypertension, age, dyslipidemia, and length of DM, with an increasing incidence and progression in severity throughout the course of the study's 3-year screening interval. As a result, it is highly recommended that you reduce the risk factors that could prevent or slow the development of DR. Diabetic retinopathy is the most common optical

fundus disease among diabetics. Fasting blood glucose, serum triglycerides, total cholesterol, hypertension, and HbA1c all individually increase the risk of developing diabetic retinopathy. Male, diabetic nephropathy, sex, age, the duration of diabetes, diabetic neuropathy, diabetic foot ulcers, and foot amputation are additional risk factors for diabetic retinopathy. DR is a severe microvascular complication of DM that begins asymptomatically, develops gradually, and worsens with time. Primary prevention is strongly suggested in order to lessen the risk factors that will delay the onset and progression of DR(7).

It is important to recall that in the representative cohort; more than 50% of type 2 diabetes patients had the condition, which is easily detectable with a symptom questionnaire and semi-quantitative testing. In groups with good glycemic control, normal blood pressure, and no dyslipidemia, neuropathy is less common, so addressing modifiable risk factors can reduce the likelihood that neuropathy will manifest (8). Additionally, identifying diabetes patients who are at risk for cognitive impairment—a recently identified side effect of the type 2 diabetic populations—might be made easier with the help of the assessment of retinal neurodegeneration. When examining a diabetic person, the identification of DR has treatment implications. In this respect, DR could deteriorate after a sharp drop in blood sugar (9).

DR monitoring is essential for the diagnosis of DN in persons with type 2 diabetes and renal impairment. Although PDR performs significantly worse than DR in separating DN from NDRD and has an excessively high specificity for the diagnosis of DN, there may not be a comparable correlation between the severity of DR and the presence of DN in type 2 diabetes. To more precisely identify patients with DN, a noninvasive, low-cost approach of enhancing retinopathy detection may be a possibility (10). The most prevalent optical fundus condition among diabetics is diabetic retinopathy. Additionally, blood triglycerides, total cholesterol, and HbA1c each independently increase the risk of developing diabetic retinopathy. Additional risk factors for diabetic retinopathy include age, male sex, hypertension, fasting blood glucose, diabetic neuropathy, diabetic nephropathy, the duration of diabetes, diabetic foot ulcers, and foot amputation(11).

Conclusion

The future progression of chronic kidney disease and the neurological consequences in diabetic patients can both be predicted using the link between the severity of DR and the severity of diabetic nephropathy and diabetic neuropathy.

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