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Correlation Between Diabetic Nephropathy and Diabetic Retinopathy

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Abstract

Diabetic retinopathy (DR) and diabetic nephropathy (DN) are prevalent and substantial microvascular complications of diabetes. This review discusses the associations between DR and DN about epidemiology, pathogenesis, clinical features, and treatment. This review aims to provide a summary reference for future, rigorous research, early detection, and comprehensive treatment of diabetic complications.

Keywords: Diabetes; Diabetic Retinopathy; Diabetic Nephropathy

Abbreviations: IDF: International Diabetes Federation; DR: Diabetic Retinopathy; DN: Diabetic Nephropathy; RAS: Renin-Angiotensin System; DN: Diabetic Nephropathy; RAS: Renin-Angiotensin System; ACR: Albumin - Creatinine Ratio; NDRD: Non - Diabetic Retinopathy Nephropathy; ACEI: Angiotensin - Converting Enzyme; RAS: Renin-Angiotensin System; KKS: Kallikrein-Kinin System; EPC: Endothelial Progenitor Cell

Introduction

The International Diabetes Federation (IDF) Diabetes Atlas 2021 estimates that the global prevalence of diabetes mellitus (DM) among adults aged 20 - 79 years was 10.5% (536.6 million) in 2021 and will increase to 12.2% (783.2 million people) by the year 2045. In 2021, the Diabetes Atlas estimates that the prevalence was greater in urban areas (12.1%) versus rural areas (8.3%), in high - income countries (11.1%) versus low - income countries (5.5%). Overall, the highest relative increase in prevalence from 2021 to 2045 is predicted to be in middle - income countries (21.1%) relative to high - income (12.2%) and low - income countries (11.9%). The global diabetes - related health expenditure was estimated to be $966 billion in 2021 and will be estimated to be $105.4 billion in 2045 [1]. Diabetes is a chronic metabolic disease of global relevance, in which the incidence has continuously risen. Extended periods of prolonged hyperglycemia can affect multiple organs and systems in the body. Specifically, diabetic retinopathy (DR) and diabetic nephropathy (DN), tend to occur together in practice since they have many similarities, from a pathogenesis perspective. Thus, it is significant to study the relationship between them in deeper depth.

Diabetic Retinopathy

In the nineteenth century, the signs of diabetic retinopathy as detected by ophthalmoscopy were described. The common retinal microvascular signs of non-proliferative diabetic retinopathy include microaneurysms, hemorrhages, hard exudates (lipid deposits), cotton wool spots (axoplasmic debris identifiable in the adjacent ganglion cell axon bundles), venous dilation and beading, and intraretinal microvascular abnormalities. The signs of proliferative diabetic retinopathy include neovascularization of the optic disc (NVD) or elsewhere (NVE), pre - retinal hemorrhage or vitreous hemorrhage (Figure 1,2) [2].

Diabetic Nephropathy

Diabetic nephropathy represents the most common cause for end-stage renal disease globally, making it a serious complication of diabetes [3]. Diabetic nephropathy can be organized into glomerular hyperfiltration stage, latent stage, microalbuminuria stage, macroalbuminuria stage, and end-stage renal disease from the perspective of clinical manifestations [4]. The persistent proteinuria is distinguishing clinical manifestations in earlier stages; while some patients may experience an increase in urine foaming, there will be gradual declines in renal function, increased blood pressure, and swelling of the body. The end-stage renal disease will result in a short-term severe loss of renal function which will subsequently lead to uremia, which can only be managed with dialysis or other interventions for life maintenance [5].

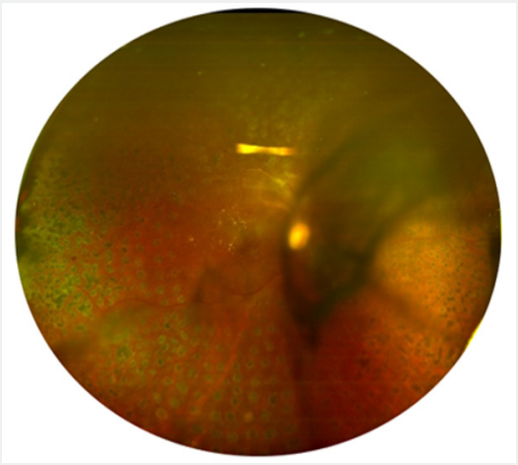

Figure 1: Vitreous hemorrhage in a DR patient (right eye).

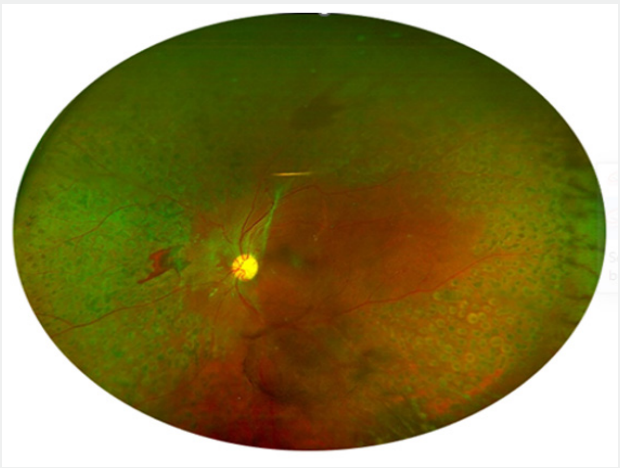

Figure 2: Vitreous hemorrhage in a DR patient (let eye).

Epidemiological Relevance

Research [6] shows that the incidence rates of chronic complications of diabetes from high to low are as follows: Diabetic cardiovascular disease (DPN) (29.09%), diabetic nephropathy (DN) (26.04%), diabetic retinopathy (DR) (24.61%), diabetic cerebrovascular disease (23.15%) and diabetic lower extremity vascular disease (19.32%). The incidence rates for DN and DR are close in rank with only a 1.43% difference.

Correlation Between the Pathogenesis of DN and DR

There is a direct and mutual pathological mechanism between DN and DR, which affects patients [7].

Endothelial Barrier Damage

The major molecular mechanisms that contribute to retinopathy (DR) and nephropathy (DN) when the microvascular endothelial barrier in both the retina and glomerulus are damaged:

8.1.1. Abnormal and sustained blood glucose levels may cause ischaemia/hypoxia in retinal tissue, thus initiating oxidative stress responses and the accumulation of inflammatory mediators, whilst also activating multiple metabolic pathways. These pathological alterations affect the vascular endothelial barrier collectively, leading to a rupture of the endothelial structure and large increases in vascular permeability. Therefore, the above mechanisms are important for the promotion of the pathophysiological process of diabetic retinopathy (DR) and diabetic nephropathy (DN) [8].

8.1.2. Exosomes [9] are abundant and located throughout the retina, kidneys and blood circulation. Increased blood glucose levels can vary considerably in the number and composition of exosomes, which could cause functional defects in the endothelial cells in the retina and kidneys. The high-glucose environment has a significant influence on exosomes generation and secretion and ultimately damage the physiological functions of endothelial cells in specific tissues. Exosomes target tissues are the retina and

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kidneys, and the functional status of the endothelial cells in those organs have a close relationship with blood glucose levels.

8.1.3. Research has shown [10] that circular RNAs (circ RNAs) are plentiful in the retina, kidneys, and circulatory system. These RNAs can behave as competitive endogenous RNAs and regulate the expression levels of mRNAs in retinal and renal endothelial cells by adsorbing miRNAs, which have implications in the pathogenesis of diabetic retinopathy (DR) and diabetic nephropathy (DN).

8.1.4. In the pathological process of diabetes, the endothelial tissues in the retina and kidneys form intercellular signal transmission with the surrounding cells (including renal tubular epithelial cells and pericytes). This interaction mechanism leads to atypical changes in endothelial function and contributes to the development of diabetic retinopathy (DR) and diabetic nephropathy (DN). Continued research on the endothelial barrier function of the retina and kidney could elucidate DR and DN. In this process, biomarkers can be identified, and targets for therapy or new therapies can be harnessed [11].

Renin-angiotensin System (RAS)

Angiotensin II not only causes inflammatory responses and endothelial cell dysfunction via reactive oxygen species generation but also plays a role in the regulation of the extracellular matrix and activates several tissue injuries signaling pathways. Angiotensin II disrupts retinal blood flow and increases the creation of reactive oxygen species and inflammatory mediators in the retina while also stimulating the growth of new retinal blood vessels by increasing vascular endothelial growth factor expression. Thus, renin-angiotensin system (RAS) inhibitors are likely useful for diabetic retinopathy and not just blood pressure control [12].

Kallikrein-kinin System (KKS)

Bradykinin and its receptors are pro-angiogenic and pro-inflammatory. The plasma kallikrein proteolytic cascade in the cardiovascular system leads to: In patients with diabetic retinopathy, increased carbonic anhydrase increases retinal permeability and edema. Increased carbonic anhydrase increases intraocular pH and activates plasma kallikrein-bradykinin [13]. In DN, inhibiting the KKS pathway has been shown to reduce glomerular and tubular injury [14]. Genetic Factors Diabetic complications exhibit a family aggregation effect. There are several shared genes in the occurrence of lesions of the eye and kidney organ, such as Pax 2, BMP 7, and WT - 1, which are implicated by the loss of functions leading to a continuum of diseases, noting the involvement of the eye and kidney [15]. Genetically predicted diabetic retinopathy exhibited a positive association with diabetic nephropathy (OR = 1.32; P = 3.72E-11), with type 1 diabetes that had kidney complications (OR = 1.96; P = 7.11E-11), and type 2 diabetes that had kidney complications (OR = 1.26, P = 3.58E-04). Similarly, the subtypes and multivariable mendelian randomization (MVMR) suggested the same conclusion [16].

Clinical Correlation Between DN and DR

China Research

The study showed a significant correlation between renal function markers (urinary microalbumin, urine albumin/creatinine ratio (UACR), estimated glomerular filtration rate (eGFR)) with the degree of diabetic retinopathy (DR) based on the ETDRS criteria. The association of UACR with DR was higher than that of eGFR which indicates the presence of DR may be identified earlier by the association with UACR [17]. For instance, with a UACR of ≥300 mg/g, the risk of diabetic retinopathy doubles [17]. Guan Yiming and colleagues conducted a retrospective clinical analysis of the data of 140 patients with diabetic retinopathy (DR) of which 88 developed diabetic nephropathies (DN).

In the DN group the duration of diabetes, body mass index, systolic and diastolic blood pressures, serum urea nitrogen, serum creatinine, low-density lipoprotein, uric acid, and total cholesterol as well as diabetic macular edema and the ratio of non-proliferative diabetic retinopathy (NPDR) to proliferative diabetic retinopathy (PDR) were higher than in other patient group with no DN (all $P < 0.05$) whilst glomerular filtration rate was lower in the DN group ($P < 0.05$) [18]. Li Ying et al. [19] found that the hypertension, diabetic retinopathy (DR), and levels of glycated hemoglobin (HbA1c), creatinine, and 24 - hour urinary protein quantification in the DN group, were statistically larger than in their normal patient group (non - diabetic retinopathy nephropathy (NDRD), $P < 0.05$) whilst hemoglobin and triglycerides levels were smaller than a normal patient group (non - diabetic nephropathy, $P < 0.01$) [19].

Wang Lijuan et al. [20] reported that DR presence and account for 29.1% diabetic nephropathy. They reported that the duration of illness, the levels of serum uric acid, and urinary albumin for the DR group were significantly higher than the same parameters in their NDR group ($P < 0.05$). UACR (≥76.56mg/g) and the duration of diabetes were risk factors for DR occurrence in diabetic kidney disease patients. Urinary albumin had an area under the ROC curve of 0.686 for DR prediction, neither a strong predictor, but statistically significant, and urinary albumin levels in DKD patients were associated with DR occurrence. UACR risk factor for DR occurrence, with predictive effect for DR [20].

Tang Juan et al. [21] reported that with worsening DR, the serum creatinine, blood urea nitrogen, 24 - hour urinary albumin and urinary albumin/creatinine levels of patients progressively increased. The incidence rates of DN in patients with no obvious DR, mild NPDR, moderate NPDR, severe NPDR and PDR were documented to be 1.67%, 8.83%, 16.16%, 22.16% and 30.83% respectively. The duration of T2DM, smoking history, glycosylated hemoglobin, total cholesterol, triglycerides, HDL-C, LDL-C, 24 - hour urinary albumin, serum creatinine, blood urea nitrogen, urinary albumin/creatinine, and glomerular filtration rate were independent risk factors for DR [21].

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Research in Other Countries

Yao et al. [22] did a retrospective controlled study on 88 eyes of 88 patients with type 2 DM in preclinical DR (44 of non-DN (NDN) and 44 of DN). Previous studies described patients with DM but with no signs of DR, as preclinical DR patients. They concluded that in preclinical DR, there is decreased retinal vascular density and thickness in DN patients compared to NDN patients. In terms of microvascular and microstructural damage to the retina, preclinical DR in DN patients may be more advanced than in NDN patients, making the eGFR more useful for assessing retinal microvascular damage [22].

Yan et al. [23] concluded that patients with type 2 diabetes mellitus (DM) complicated by diabetic nephropathy (DN) had a relatively high prevalence of diabetic retinopathy (DR). DM had a significant association with albumin - creatinine ratio (ACR) staging. Therefore, ACR staging could be a risk factor for DR in patients with DN [23]. Liu et al. [24] concluded that there was a significant difference between diabetic retinopathy in type 2 diabetes and diabetic kidney disease (DKD) as DKD showed a higher level of each component of metabolic syndrome and disclosed the existence of DR with higher prevalence in individuals with a lower educational level [24]. In a cross-sectional study with 250 cases, Zhang et al. [25] found that after adjusting for baseline proteinuria, hematuria, eGFR, and interstitial inflammation, the severity of glomerular lesions and DM history more than 10 years were significantly related to the odds of DR.

These results demonstrate that the severity of glomerular lesions is significantly related to DR which is a separate risk factor for renal outcomes in patients with DN. This means that DR can predict renal prognosis in patients with T2DM and DN [25]. Yu Li et al. [26] found in a prospective study that diabetic retinopathy (DR) was associated with increased risk of diabetic nephropathy (DN) for T2DM patients. However, the predictive value of DR for DN in T2DM patients is relatively low. A low predictive value of DR for DN may stem from variations in the follow-up time, mean age, male proportion, and study quality [26]. Muamer Dervišević et al. [27] reported that there was also a significant correlation between duration of diabetes and occurrence of diabetic retinopathy. A correlation between degree of renal failure and changes on fundus has not been confirmed, although more severe renal insufficiency is associated with a greater incidence of DR than those with lesser renal impairment [27].

Jeng et al. [28] concluded that diabetic nephropathy (DN) represents an independent risk factor for the presence and worsening progression of diabetic retinopathy (DR). However, in this study, DN had no significant influence on the development of diabetic macular edema (DME), and that possible associations between these diseases need further investigations [28]. Yukihisa et al. [29] indicated, through multiple logistic regression analysis, that grading of diabetic nephropathy (DN) was associated with occurrence of DR and DME, and that decline of renal function could predict occurrence of DR [29]. Qian Wang et al. [30] reported that DR may be a good predictor of DN. In DN patients, the severity of DR was also related to glomerular damage. The presence of K-W nodules, decreased hemoglobin, and age were independent variables associated with more severe DR [30]. In addition [29], studies have already reported associations between the grading of diabetic nephropathy (DN), the development of diabetic retinopathy (DR), the development of diabetic macular edema, and that decline of renal function may predict occurrence of DR.

In summary, the relevant studies on diabetic nephropathy (DN) and diabetic retinopathy (DR) indicate that DN and DR are independent risk factors for each other, and they can be used as indicators for mutual diagnosis and detection, but the value is relatively low due to practical issues such as samples size. The diagnosis and detection of DN and DR can provide mutual clues to each other. For patients with diabetes, regular fundus examinations can help to find early stages of potential DN risks. For example, if DR is found to be at a relatively severe stage during a fundus examination, timely examination of urinary micro-albumin, renal functions, etc. is required to screen for possible DN. Conversely, when diagnosing and monitoring DN, if at the detection of DN it is determined that the patient has proteinuria, abnormal renal function, etc., fundus examination should be completed as quickly as possible to detect any possible DR and take corresponding treatment measures.

Correlation Between DN and DR Treatment

Control Blood Glucose, Blood Pressure and Blood Lipid [31]

Controlling blood glucose, blood pressure, and blood lipids is important to preventing and treating diabetes - related nephropathy (DN) and diabetes - related retinopathy (DR). Both DN and DR are microvascular complications of diabetes, and return of blood glucose to normal levels can mitigate hyperglycemia - induced damage to the retinal and renal micro vessels and delay progression of DR and DN. Drugs that inhibit angiotensin - converting enzyme (ACEI) or block angiotensin Ⅱ receptors (ARB) have significant effects in regulating blood pressure, and they also change abnormal hemodynamics in the kidneys and retina, reduce proteinuria, prevent or delay progression of glomerulosclerosis and retinopathy, and modulate the impacts associated with the RAS system. Statin drugs lower blood lipids, reduce blood viscosity, and have pleiotropic effects (anti - oxidation and anti - inflammation).

Klotho Protein

α -Klotho (Klotho) is a protein that has garnered attention for its role as an anti-aging protein, and it has been shown to ameliorate the onset of diseases of aging. Klotho can modify several pathways in the body such as anti-inflammation and oxidative stress, anti-fibrosis, endothelial protection, prevention of vascular calcification, regulation of metabolism, maintenance of calcium and

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phosphate homeostasis, and regulation of cell fates via the regulation of autophagy, apoptosis, and pyroptosis pathways. Klotho has been demonstrated to be capable of intervening in diabetic nephropathy (DN) via multiple pathways and the pathological mechanisms of DR and DN share several similarities. Accordingly, Klotho has the promise to be used as a method of intervention in DR. Additionally, Klotho could be targeted by a recognized drug for DN intervention [32].

Ferroptosis

Ferroptosis is a relatively new type of cell death that has been proven to be related to the occurrence and development of various diabetic complications. The disorder of cellular iron metabolism directly causes ferroptosis and abnormal iron metabolism is associated with diabetes. Selective inhibition of ferroptosis has been demonstrated to significantly improve renal function and produce a protective effect in retinal models, both animal and cell models; although, to date, there have been no clinical trials utilizing ferroptosis specific inhibitors for the treatment of diabetic nephropathy (DN) and diabetic retinopathy (DR). While the biological functions and molecular mechanisms of ferroptosis have not yet been fully characterized, it has emerged as a hot topic in diabetic complication research [33]. Addressing ferroptosis may be the key to the prevention and treatment of DN and DR in the future.

MiRNA

MiRNAs exhibit multiple targets and are associated with different pathogenic pathways of various diseases. Moreover, specific genes may be regulated by several miRNAs. The resulting changes in the expression of specific miRNAs lead to the dysregulation of various molecular mechanisms and signaling pathways, resulting in diabetic microvascular complications, including diabetic retinopathy (DR), and diabetic nephropathy (DN). MiRNAs play an important role in angiogenesis resulting in DR. In addition, the resulting dysregulation levels of different miRNAs are the basis of various pathophysiological mechanisms resulting in DR through their effects on infections, oxidative stress, endothelial cell apoptosis, and endothelial progenitor cell (EPCs) function. There are economic advantages of miRNA treatment due to their selective regulation in anti-miRNA treatments through antisense inhibition that has been shown to regulate the prognosis of various diseases. Thus, targeting miRNAs miRNA modification may be successful and effective treatment modalities for DR and DN [34].

Calcium Dobesilate Capsules Combined with Compound Xueshuantong Capsules Therapy

Research has shown [35] that after a three - month intervention in the experimental group it had significantly lower rates of 24 - hour urinary albumin excretion, serum creatinine, and blood urea nitrogen than the control group. The comparison of differences in statistical analyses between the experimental and control group was of significance ($P < 0.05$). The comparison before and after treatments resulted in a significant decreasing trend in both experimental and control groups for every measure, the range of retinal hemorrhage, diameter of hemangiomas, thickness of the macular area, and gray - scale value of the visual field had predictable statistical differences of significance ($P < 0.05$).

Significantly improved means were exhibited for the experimental group for every measurement that was statistically significant for all of the above comparisons with controls ($P < 0.05$). Research has confirmed that there is significant clinical efficacy in the combined use of calcium dobesilate and Compound Xueshuantong with people with diabetic nephropathy complicated by diabetic retinopathy and the procedure is safe and reliable, which should be significantly promoted in the clinical setting.

Conclusions and Perspectives

Diabetic retinopathy (DR) and diabetic nephropathy (DN) are closely correlated in numerous fields - epidemiology, pathophysiological mechanisms, genetic considerations, clinical features and treatments. The ability to understand the correlation between both diabetic complications improves our understanding of diabetic microvascular complications as a whole and provides a theoretical basis for early diagnosis, follow-up and comprehensive treatment. Further, in-depth research into the underlying basic and clinical medicine continuing should uncover more mysteries of the association between DR and DN and develop more specific and effective therapies, especially using substances such as klotho protein, ferroptosis, miRNA etc. This should improve patient outcomes, lessen the impact of serious adverse events such as blindness, renal failure and management of diabetic patients to improve quality of life with fewer complications. Moving forward, further verification of causal relationships and areas of interest, such as genetic markers and imaging approaches, is needed.

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