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Diabetes, Cardiomyopathy, And Heart Failure-An Updated Review Article for Nursing.

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Abstract:

Background: Diabetes has been recognized as a significant risk factor for heart failure (HF) and cardiomyopathy. Its impact extends beyond the traditional cardiovascular (CV) risk factors such as hypertension and coronary artery disease (CAD). Diabetes contributes to heart failure through various mechanisms, including insulin resistance, altered myocardial metabolism, and inflammatory pathways. As a result, individuals with diabetes are categorized as at risk for HF, with prevalence rates significantly higher in diabetic populations compared to the general population. Recent advancements in understanding the pathophysiology of diabetes-related heart failure have led to new treatment strategies, particularly involving sodium-glucose co-transporter-2 (SGLT2) inhibitors.

Aim:
The aim of this review is to examine the relationship between diabetes, cardiomyopathy, and heart failure, with a focus on pathophysiology, diagnosis, prognostic implications, Nursing intervention plans, and management strategies.

Methods: This article provides a comprehensive review of current literature regarding the prevalence, pathophysiology, and treatment of diabetes-related heart failure. It integrates findings from epidemiological studies, clinical trials, and advances in pharmacotherapy, particularly SGLT2 inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs). Additionally, the article discusses the importance of early detection and classification of heart failure phenotypes in diabetic patients.

Results: Diabetes and heart failure are often coexistent, with diabetes increasing the risk of developing heart failure by 1.7 to 2.5 times. The prevalence of heart failure in diabetic patients varies between 9% and 22%, and recent trials have shown that SGLT2 inhibitors significantly improve outcomes for both diabetic and non-diabetic patients with heart failure. Moreover, glycemic control remains an important factor in reducing the risk of heart failure hospitalizations.

Conclusion: The coexistence of diabetes and heart failure significantly worsens prognosis, highlighting the need for early screening and intervention. New pharmacological treatments such as SGLT2 inhibitors offer

promising outcomes for patients with both conditions. Early detection of pre-diabetes and diabetes in individuals with heart failure is crucial for improving patient outcomes and reducing the risk of cardiovascular morbidity and mortality.

Keywords: Diabetes, Heart Failure, Cardiomyopathy, SGLT2 Inhibitors, Pathophysiology, Glycemic Control, Prognosis

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Introduction:

Diabetes has been identified as an independent risk factor for cardiomyopathy and heart failure (HF), regardless of the presence of traditional cardiovascular (CV) risk factors such as hypertension and coronary artery disease (CAD) [1–4]. The universal definition of HF categorizes individuals with diabetes as "at risk for HF" (Stage A). Asymptomatic individuals are classified as having "pre-HF" (Stage B) if they exhibit at least one of the following: 1) evidence of structural heart disease, 2) abnormal cardiac function, or 3) elevated cardiac biomarkers, such as natriuretic peptides or troponins. According to this classification, HF (Stage C) is defined as a clinical syndrome marked by signs or symptoms of HF, resulting from abnormalities in cardiac structure and function, and confirmed by elevated natriuretic peptides or objective evidence of cardiogenic congestion, either pulmonary or systemic [3,5]. The prevalence of diabetes in the United States is approximately 10.2%, with HF affecting between 9 and 22% of patients with diabetes [6–10]. In clinical trials involving antidiabetic agents, 4 to 30% of participants with diabetes exhibited HF [11]. Additionally, pre-diabetes or diabetes was found in 30 to 40% of individuals enrolled in HF trials [12,13].

Pathophysiology of Diabetes-Induced Heart Failure

Chronic diabetes induces alterations in cardiac structure and function, driven by the direct effects of abnormal myocardial metabolism and insulin resistance (IR), even in the absence of atherosclerotic CAD [14]. The pathophysiological relationship between diabetes and HF is intricate and multifactorial, involving a variety of abnormal biochemical pathways. These include, but are not limited to, disrupted calcium signaling, deranged glucose and fatty acid metabolism, and inflammatory processes that contribute to myocardial fibrosis, stiffness, and hypertrophy [7,15,16]. The interaction of these mechanisms can lead to both asymptomatic diastolic and systolic dysfunction, ultimately culminating in the clinical manifestation of HF. On the other hand, HF is also associated with a higher prevalence of diabetes and is considered a predictive factor for the future development of type 2 diabetes mellitus (T2DM) [17].

Heart Failure Phenotypes in Diabetes

Left ventricular (LV) dysfunction in diabetic patients can manifest through three distinct HF phenotypes: HF with preserved LV ejection fraction (LVEF ≥ 50%; HFpEF), HF with mildly reduced LVEF (HFmrEF; LVEF 40-49%), and HF with reduced LVEF (HFrEF; LVEF ≤ 40%) [3]. Diagnosing HFpEF and HFmrEF can be challenging due to symptom overlap with other comorbidities such as obesity, lung disease, and chronic kidney disease (CKD). Consequently, clinical guidelines advocate for the inclusion of additional objective diagnostic criteria, such as elevated natriuretic peptides or imaging evidence of structural heart disease or diastolic dysfunction [18].

Prognostic Implications of Diabetes and Heart Failure Coexistence

The coexistence of diabetes and HF represents a poor prognostic indicator, significantly increasing the risk of HF hospitalization, all-cause mortality, and CV-related mortality. Epidemiological studies have demonstrated that patients with both HF and diabetes face a 50-90% greater risk of CVD mortality, regardless of the HF phenotype [12,19]. HF patients without diabetes are also at a heightened risk for developing glycemic abnormalities. Furthermore, newly diagnosed pre-diabetes has been linked to a considerably higher risk of all-cause and cardiovascular mortality in HF patients, underscoring the importance of screening for pre-diabetes or diabetes in individuals with HF [17,20,21]. Additionally, early echocardiographic assessment can be valuable for detecting subclinical structural abnormalities and myocardial dysfunction in asymptomatic diabetic patients.

Advancements in the Pathophysiology and Treatment of Diabetes-Related Heart Failure

The pathophysiology of diabetes-related HF remains complex, with significant advances in understanding occurring over the past decades. Despite these advancements, many aspects of this condition remain inadequately understood. Since 2015, landmark clinical trials involving sodium-glucose co-transporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have revolutionized the understanding of cardiovascular risk reduction in patients with T2DM, prompting a paradigm shift in clinical practice recommendations for managing T2DM [22]. Robust evidence from cardiovascular outcome trials (CVOTs) has confirmed substantial improvements in HF outcomes with SGLT2 inhibitors in both diabetic and non-diabetic patients. These findings have spurred greater interest in the relationship between diabetes and HF within the medical community.

Current Guidelines on SGLT2 Inhibitors for Heart Failure

The American Diabetes Association (ADA) now recommends the use of SGLT2 inhibitors as first-line therapy for T2DM patients at high risk for, or with established, HF [23]. In addition, several large-scale randomized clinical trials (RCTs) have substantiated the cardiovascular benefits of SGLT2 inhibitors in patients with established HF, irrespective of LVEF or diabetes status. These RCTs have elevated SGLT2 inhibitors from merely glucose-lowering agents to essential medications for managing HF. The 2022 guidelines from the American College of Cardiology (ACC) Foundation, the American Heart Association (AHA), and the Heart Failure Society of America (HFSA) now recommend SGLT2 inhibitors for the treatment of HF, regardless of LVEF [3].

Epidemiology Of Diabetes And HF

There exists a bidirectional relationship between diabetes and heart failure (HF) [24]. Both type 1 and type 2 diabetes are well-established risk factors for the development of HF [25–27]. Furthermore, HF itself may contribute to the pathogenesis of insulin resistance (IR) and type 2 diabetes mellitus (T2DM) [28]. The frequent coexistence of T2DM and HF can be largely attributed to the shared risk factors and overlapping pathophysiological mechanisms that underlie both conditions. According to data from the National Health and Nutrition Examination Survey (NHANES) conducted between 2015 and 2018, the prevalence of HF in the general adult population in the U.S. is approximately 2.3% [29]. Among individuals with diabetes, the prevalence of HF ranges from 9% to 22%, depending on the population characteristics [6,8,9]. Diabetes is particularly prevalent in patients diagnosed with HF. In contemporary large-scale clinical trials of HF treatments, between 32% and 43% of patients with chronic HF had coexisting diabetes [12,30,31]. Data from a nationwide U.S. registry (NHANES 2005-2016) revealed that among HF patients, the rates of diagnosed and undiagnosed T2DM were 34.7% and 12.8%, respectively [32]. HF is a common yet often under-recognized complication in individuals with diabetes [33]. The pathogenesis of cardiomyopathy and HF in diabetic patients is heterogeneous. The robust associations between diabetes and coronary artery disease (CAD), hypertension, and renal disease significantly contribute to the development of cardiomyopathy and HF in this population [34]. Additionally, HF occurs more frequently in diabetic individuals even in the absence of other traditional HF risk factors [16,35].

Diabetic Cardiomyopathy

Diabetic cardiomyopathy, although not defined by a standardized clinical criterion, generally refers to myocardial dysfunction attributed to diabetes in the absence of other identifiable causes [36]. The concept of diabetes-induced myocardial dysfunction was first articulated by Lundbæk in 1954 [37]. A pivotal study in 1972 by Rubler et al. formally described diabetic cardiomyopathy as a distinct clinical entity, following post-mortem examinations of four patients with diabetes-related HF and dilated cardiomyopathy without other apparent causes of myocardial dysfunction [14]. Initially, reports on diabetic cardiomyopathy focused on a dilated left ventricle (LV) with eccentric hypertrophy and LV systolic dysfunction (HFrEF). However, more recent clinical studies have identified heart failure with preserved ejection fraction (HFpEF) characterized by concentric LV hypertrophy and LV diastolic dysfunction as a separate phenotype of

cardiomyopathy, rather than an intermediate form between risk factors and HFrEF [38]. It appears that the transition from HFpEF to HFrEF is less common than previously assumed.

Epidemiologic Evidence

Large-scale epidemiologic studies have substantiated the strong association between diabetes and HF. For example, data from the Framingham Heart Study in the 1970s revealed that individuals with diabetes had a twofold (in men) to fivefold (in women) increased risk of developing HF, after adjusting for other risk factors [2,39]. Similarly, more recent cohort studies have demonstrated that diabetes is independently associated with a 1.7 to 2.5-fold increased risk of HF [6,40]. A nationwide cohort study from Sweden, which included over 679,000 patients with T2DM and more than 2 million matched control subjects, found that a diagnosis of T2DM was linked to an elevated risk of HF, even when cardiovascular risk factors such as glycated hemoglobin, systolic blood pressure, estimated glomerular filtration rate, and lipid levels were within target ranges [41]. This study also showed a significant decline in cardiovascular complications over the past two decades in both individuals with and without T2DM. However, the rate of HF in patients with T2DM has plateaued in recent years, with the obesity epidemic potentially contributing to this trend. Adiposity plays a crucial role in the development of HF in patients with diabetes, as illustrated by a recent analysis of two U.S. cohort studies, which found that overall obesity, abdominal obesity, and fat mass were strongly associated with an increased risk of HF in diabetic participants. Interestingly, no such association was found in individuals without T2DM [42]. Ischemic heart disease is more commonly observed in HF patients with coexisting T2DM compared to those without T2DM, with rates of 63% versus 47%, respectively. Additionally, nearly 90% of patients with T2DM and HF of non-ischemic origin have at least one comorbidity, such as hypertension, atrial fibrillation, valvular disease, or pulmonary disease, that could contribute to the development of HF [43].

Diabetes and Heart Failure Progression:

Diabetes has been identified as an independent predictor for the progression from preclinical heart failure (HF) stages A and B to clinically significant HF (stage C) [44]. A population-based analysis utilizing the National Scottish Register revealed that the risk of hospitalization due to HF was approximately twice as high in individuals with diabetes compared to those without diabetes [45]. Furthermore, a prospective cohort study conducted in the southeastern United States established that hypertension and diabetes were among the strongest risk factors for HF in both White and Black populations [46]. The population-attributable risk for HF was highest for hypertension (31.8%) and followed by diabetes (17%). A similar population-based case-control study found that diabetes was responsible for about 17% of the attributable risk for HF, with no significant differences between heart failure with preserved ejection fraction (HFpEF) and heart failure with reduced ejection fraction (HFrEF) [47].

Sex Differences in HF Risk and Diabetes:

Epidemiological studies have demonstrated that men with diabetes have a higher incidence of HF compared to women with diabetes [27,40], which aligns with the general population's higher risk of HF in men. However, it is noteworthy that diabetes contributes to a greater relative increase in HF risk in women than in men, as evidenced by several studies [27,39]. A meta-analysis encompassing 47 cohort studies and over 12 million individuals showed that both type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM) were associated with a greater excess risk of HF in women than in men, with a 47% and 9% higher risk, respectively [48]. The underlying reasons for this sex-specific disparity in diabetes-related HF risk remain unclear.

Prediabetes and the Risk of Heart Failure:

Some epidemiological studies have raised the concern that prediabetes may be an independent risk factor for cardiomyopathy and HF. A cohort study demonstrated that prediabetes was significantly associated with an increased risk of HF, with an odds ratio of 1.7 [9]. Additionally, a modest but statistically significant relationship exists between fasting plasma glucose levels and the risk of HF, independent of diabetes status [50].

Glycemic Control and the Risk of Heart Failure:

Glycemic control has been shown to be a critical factor in predicting the risk of HF among individuals with T1DM and T2DM [26,27,51]. A prospective case-control study from the Swedish National Diabetes Register investigated the impact of glycemic control on the future risk of HF hospitalization over an average follow-up of 7.9 years [26]. Compared to a control group without diabetes, patients with T1DM exhibited a fourfold increased risk of HF hospitalization. However, the risk varied significantly based on the level of glycemic control and comorbidities, with a hazard ratio (HR) of 2.2 for individuals with hemoglobin A1c (HbA1c) ≤6.9%, and an HR of 11.2 for individuals with HbA1c ≥9.7%. Another analysis from the same registry found that each 1% increase in HbA1c was associated with a 20% higher risk of HF in patients with T1DM and a history of myocardial infarction [52]. In individuals with T2DM, the association between poor glycemic control and the excess risk of HF is more prominent in middle-aged adults under 55 years old, while the link weakens with advancing age [27].

Age of Diabetes Diagnosis and Its Impact on Heart Failure Risk:

The age at which diabetes is diagnosed plays a significant role in the future risk of HF. Younger age at diagnosis is associated with a higher risk of HF [45,53,54]. A study by Rawshani et al. utilizing data from the Swedish National Diabetes Register found that individuals diagnosed with T1DM before the age of ten had a twelvefold higher risk of developing HF, while those diagnosed in young adulthood (20 to 29 years) had a fivefold increased risk compared to a control group without diabetes. Similarly, an analysis by Sattar et al. using the same registry found that adults diagnosed with T2DM before 41 years of age had a fivefold higher risk of HF compared to their peers without diabetes [54]. Interestingly, a T2DM diagnosis after the age of 80 was associated with a lower risk of HF and all-cause cardiovascular mortality. In line with these findings, a US cohort study revealed that every additional 5 years of diabetes duration was associated with a 17% increase in the risk of incident HF [55]. This association was especially pronounced in individuals with elevated HbA1c levels.

The relationship between the duration and age at diagnosis of diabetes and the subsequent risk of HF is likely multifactorial, particularly distinguishing between T1DM and T2DM. The total glycemic load, which includes the cumulative exposure to hyperglycemia, is a known predictor of diabetes-related complications. This load is influenced by glycemic variability and the duration of diabetes, which is especially significant in T1DM [53]. Individuals diagnosed with T2DM at a younger age tend to have higher comorbidity burdens, including obesity, dyslipidemia, hypertension, nephropathy, smoking, and lower socioeconomic status, compared to those diagnosed later in life. This comorbidity burden likely contributes to the increased HF risk seen in younger patients with T2DM [54]. These findings underscore the importance of delaying the onset of diabetes as a strategy for preventing HF [55].

Diabetes and Comorbidities: Their Impact on Heart Failure Risk:

Traditional cardiovascular disease (CVD) risk factors such as hypertension, obesity, dyslipidemia, and smoking are common among individuals with diabetes. For example, hypertension affects 66% to 76% of adults with diabetes in the United States [56]. The 2020 National Diabetes Statistics Report indicates that 45.8% of adults with diabetes are obese (BMI between 30 and 39.9 kg/m^2), and 15.5% are morbidly obese (BMI ≥40 kg/m^2) [10].

Coexisting CVD risk factors significantly exacerbate the risk of HF in patients with diabetes. A large prospective cohort study involving more than 270,000 participants with T2DM from the Swedish National Diabetes Register examined the relationship between five cardiovascular risk factors (elevated HbA1c, dyslipidemia, albuminuria, smoking, and high blood pressure) and CVD outcomes after a median follow-up of 5.7 years [57]. The study found that patients with T2DM who had none of the risk factors outside target ranges had a 45% higher risk of hospitalization for HF compared to controls without diabetes. However, the risk was substantially higher (HR of 11.35) when all five risk factors were outside target ranges, highlighting the critical importance of controlling cardiovascular risk factors in preventing HF among individuals with diabetes. Recent studies also suggest that comorbid mental health disorders may further increase the risk

of HF in individuals with diabetes. A retrospective analysis of nationwide health claims data from Korean participants identified a significant independent association between the number of mental health disorders and increased HF risk in diabetic patients [58].

HF as a Risk Factor for Diabetes:

Patients with heart failure (HF) are at an elevated risk of developing incident diabetes mellitus (DM) over time. Clinical trial data has indicated that the incidence of new-onset diabetes among individuals with HF ranges between 7% and 11% over a 3- to 5-year follow-up period [59,60]. Although the existing body of evidence is limited, emerging data over the past two decades supports the potential independent role of HF as a risk factor for the onset of type 2 diabetes mellitus (T2DM) [61]. Analyses of prospective cohort studies and clinical trial participants suggest that the presence of HF at baseline may predispose individuals to an increased risk of developing new-onset diabetes [61–64]. Key predictors of incident diabetes in individuals with HF include elevated glucose levels, HbA1c, body mass index (BMI), waist circumference, the duration of HF, and higher functional class of HF [28,61–63].

IMPACT OF DIABETES ON CARDIAC STRUCTURE AND FUNCTION:

The frequent coexistence of diabetes with other comorbidities, such as hypertension and obesity, complicates the understanding of the relative contribution of each disease entity to cardiac remodeling and dysfunction in clinical practice [65]. Nevertheless, growing evidence has established an independent association between diabetes and various alterations in cardiac structure and function. These asymptomatic subclinical changes, often occurring in the earlier stages of the disease, can be detrimental and elevate the risk of progressing to heart failure (HF) and cardiovascular disease (CVD) morbidity and mortality [44]. The early identification of these subclinical alterations is critical for recognizing high-risk patients and preventing the onset of overt HF and diabetic cardiomyopathy.

Left Ventricular Hypertrophy:

Left ventricular hypertrophy (LVH) is defined by an increase in left ventricular (LV) mass due to myocardial remodeling. The condition is generally caused by a complex interplay of several factors, including hypertension, diabetes, metabolic syndrome, obesity, gender, ethnicity, as well as genetic and neurohumoral influences [66]. There are three primary abnormal geometric patterns of LV remodeling: concentric remodeling (normal LV mass with increased relative wall thickness), concentric LVH (increased LV mass and increased relative wall thickness), and eccentric LVH (increased LV mass with normal relative wall thickness). LVH has long been recognized as a target organ response and a significant independent risk factor for HF, coronary artery disease (CAD), stroke, and CVD mortality [66,67]. The presence of LVH leads to LV diastolic dysfunction by reducing LV compliance, impairing diastolic filling, prolonging isovolumetric relaxation, and increasing both LV and left atrial filling pressures [16]. The universal definition of HF acknowledges asymptomatic LVH, LV systolic dysfunction, and LV diastolic dysfunction as "pre-HF," emphasizing the progressive nature of HF and the importance of preventing its onset [5].

LVH is particularly prevalent among adults with diabetes, with an estimated occurrence rate as high as 70% [68]. A pooled analysis from three epidemiological cohort studies, involving 2,900 individuals with type 2 diabetes (T2DM) and no known cardiovascular disease (CVD), revealed that 67% of participants exhibited at least one echocardiographic abnormality, such as LVH, left atrial enlargement, or diastolic dysfunction [44]. Coexistent hypertension appears to be the primary contributor to the development of LVH in diabetic patients [69]. However, multiple studies have also demonstrated an independent association between diabetes and LVH. For instance, the Framingham Heart Study found significant associations between serum glucose, insulin levels, and insulin resistance (IR) with concentric LV remodeling, with this association being more pronounced in women than men [70,71]. A prospective cohort study with a 25-year follow-up showed that long-standing glycemic abnormalities exert a cumulative effect on LV remodeling, and individuals with early-onset diabetes tend to experience a more severe degree of LVH [72].

Diabetic cardiomyopathy may present with distinct LVH features [34]. In the HF with preserved ejection fraction (HFpEF) phenotype, it is typically characterized by thickened and stiff LV walls with a

normal LV volume. At the cellular level, cardiomyocytes appear hypertrophied, maintaining normal sarcomere structure, but with increased collagen deposition in the interstitial space. In contrast, diabetic cardiomyopathy with the heart failure with reduced ejection fraction (HFrEF) phenotype often presents with eccentric LVH and dilated LV volumes. At the cellular level, cardiomyocytes show damage, loss of sarcomeres, and replacement of some cardiomyocytes with interstitial fibrosis [38].

LV Diastolic Dysfunction:

Diastolic dysfunction is commonly observed in asymptomatic individuals with diabetes, with its prevalence ranging from 20% to 60%, depending on the diagnostic criteria and the specific population studied [73–75]. Prospective cohort studies have substantiated that diabetes, particularly when poorly controlled, independently contributes to the development of diastolic dysfunction [72]. Although diastolic dysfunction is frequently associated with LVH, it can also occur in individuals with diabetes, even in the absence of LVH [34]. Mild diastolic dysfunction, characterized by delayed myocardial relaxation, typically holds limited prognostic significance. However, as diastolic dysfunction progresses and elevated LV filling pressures are observed on echocardiographic imaging, the risk for future heart failure and mortality in diabetic patients increases [73,75]. Moreover, among asymptomatic individuals presenting with baseline diastolic dysfunction, diabetes serves as an independent predictor of progression to HFpEF or HFrEF [76].

LV Systolic Dysfunction:

Traditionally, a reduced left ventricular ejection fraction (LVEF) has been the principal marker used to identify cardiomyopathy and systolic dysfunction. LVEF is a straightforward measure commonly utilized in cardiovascular risk assessment and management. However, LVEF does not comprehensively capture the full spectrum of myocardial function [77]. A more advanced technique, global longitudinal strain (GLS), which is assessed via speckle-tracking echocardiography, measures tissue deformation in the longitudinal direction. Reduced GLS serves as an indicator of diminished contractility [75]. GLS is considered more sensitive than conventional LVEF in evaluating systolic function and provides additional prognostic value [77,78]. As a result, GLS is increasingly used to detect subclinical LV systolic dysfunction.

Impaired GLS in Diabetes and Heart Failure

Impaired Global Longitudinal Strain (GLS) is notably prevalent in asymptomatic, normotensive patients with diabetes who present with normal Left Ventricular Ejection Fraction (LVEF) [67, 79, 80]. Diabetes has been consistently linked to a reduction in GLS, which is evident even in adolescents and young adults diagnosed with Type 1 Diabetes Mellitus (T1DM) or Type 2 Diabetes Mellitus (T2DM) [81, 82]. Furthermore, a negative correlation between Hemoglobin A1c (HbA1c) levels and GLS exists, independent of the patient's diabetes status, race, or sex [83]. It is thus unsurprising that impaired GLS serves as a significant and independent predictor for the onset of heart failure (HF) and mortality in diabetic patients [67].

Diabetes has the potential to induce clinical heart failure (HF) in individuals with asymptomatic left ventricular systolic dysfunction. In a randomized controlled trial (RCT) involving adults with asymptomatic left ventricular systolic dysfunction, it was found that diabetes increased the risk of HF by 53% and doubled the risk of HF hospitalization over a median follow-up period of three years [84]. Improved glycemic control in diabetic patients has been shown to enhance both systolic and diastolic functions of the left ventricle. A prospective cohort study focusing on subjects with uncontrolled T2DM revealed that reducing HbA1c from 10.3% to 8.3% over a span of 12 months was associated with a 21% improvement in GLS and a 24% increase in septal e' velocity, a key indicator of myocardial relaxation [85].

Pathophysiologic Links Between Diabetes and Heart Failure

The pathophysiological relationship between diabetes and heart failure (HF) is multifactorial, involving several mechanisms that contribute to the progression of both conditions. These mechanisms include, but are not limited to, impaired cardiac insulin signaling, glucotoxicity, lipotoxicity, mitochondrial dysfunction, myocardial fibrosis, oxidative stress, impaired myocardial calcium handling, cardiovascular

autonomic dysfunction, endocardial dysfunction, and the overactivation of the renin-angiotensin-aldosterone system (RAAS) [7]. The extent to which each of these mechanisms contributes to the phenotype of diabetic cardiomyopathy remains unclear. Additionally, the specific pathophysiological processes involved may differ based on the type of diabetes (T1DM vs. T2DM) and the type of HF (HFrEF vs. HFpEF) [86].

Alterations in Myocardial Energy Substrate Utilization

Under normal physiological conditions, the heart predominantly utilizes free fatty acids (FFAs; ~70%) and glucose (~30%) for energy, with the capacity to adapt to available fuels depending on their availability [15]. In T2DM, however, myocardial substrate utilization shifts toward increased reliance on FFAs, with a corresponding decrease in glucose utilization due to insulin resistance (IR). This metabolic shift results in decreased myocardial metabolic flexibility and an almost exclusive reliance on FFAs. Consequently, this adaptation leads to less efficient energy production, as FFA oxidation requires more oxygen than glucose or ketone bodies [15, 87]. Moreover, the enhanced uptake of FFAs leads to triglyceride accumulation within cardiomyocytes, promoting lipotoxicity, mitochondrial dysfunction, oxidative stress, and apoptosis [34]. A prospective study involving endomyocardial biopsy samples from 158 adult heart transplant recipients revealed that, within just three months post-transplant, diabetic recipients demonstrated early lipid accumulation (triacylglycerol and ceramide) in their cardiomyocytes, whereas no such lipid accumulation was observed in non-diabetic recipients. Importantly, cardiomyocyte lipid accumulation was found to be an independent predictor of early systolic and diastolic dysfunction in diabetic transplant recipients after 12 months.

Hyperinsulinemia and Insulin Resistance

Insulin resistance (IR) is broadly defined as the inability of insulin to effectively perform its metabolic actions at the cellular level [34]. It represents the central defect in the pathogenesis of both metabolic syndrome and T2DM. Furthermore, heart failure is recognized as a state of insulin resistance, with IR significantly impacting both the risk and prognosis of HF [34, 89]. IR promotes increased lipolysis, hepatic lipogenesis, and hepatic gluconeogenesis, leading to an overload of substrates that contribute to myocardial dysfunction through lipotoxicity and glucotoxicity [75]. Moreover, both IR and the resultant hyperinsulinemia interfere with the signaling pathways responsible for cardiomyocyte hypertrophy [38].

Oxidative Stress

Oxidative stress arises from an imbalance between the generation of reactive oxygen species (ROS) and the body's ability to neutralize them through antioxidants [75]. Exposure to hyperglycemia induces oxidative stress through the activation of NADPH oxidase, mitochondrial superoxide production, and the formation of advanced glycation end products (AGEs), which result from the nonenzymatic glycation and oxidation of proteins and lipids [34, 87]. Oxidative stress contributes significantly to cardiac remodeling, impaired contractility and relaxation, and dysregulated calcium handling in cardiomyocytes [75]. Moreover, oxidative stress exacerbates myocardial dysfunction by inducing protein and DNA damage, increasing myocardial inflammation, and disrupting intracellular signaling pathways [34].

Endoplasmic Reticulum Stress and Impaired Calcium Handling

Myocardial intracellular calcium levels are crucial for regulating myocardial contractility throughout the cardiac cycle. Disruptions in calcium handling can impair both myocardial contraction and relaxation [90]. The endoplasmic reticulum plays a critical role in calcium handling, lipid synthesis, and protein folding and modification [91]. In the context of hyperglycemia and IR, endoplasmic reticulum stress is triggered, leading to the accumulation of unfolded proteins and the disruption of calcium handling. In diabetic cardiomyopathy, the impaired reuptake of calcium by the endoplasmic reticulum results in prolonged diastolic relaxation [91]. Animal model studies have demonstrated that altered calcium handling within cardiomyocytes is central to the pathophysiology of diabetic cardiomyopathy [7, 90].

Endothelial and Microvascular Dysfunction

Endothelial dysfunction, which compromises the communication between endothelial cells and cardiomyocytes as well as vascular function, is a common feature in patients with diabetes and cardiovascular disease (CVD) [15]. Diabetes facilitates the deposition of AGEs in the endothelial and smooth muscle cells of the myocardial microvasculature [92]. This deposition triggers vascular inflammation, which in turn reduces the production of nitric oxide by the endothelium. Decreased myocardial nitric oxide bioavailability leads to concentric left ventricular remodeling and diastolic dysfunction [38]. Moreover, diabetes has been associated with capillary rarefaction and the loss of pericytes, which impair myocardial perfusion, reduce coronary flow reserve, and induce tissue hypoxia, further increasing myocardial stiffness and reducing contractility [38, 93].

Inflammation

Systemic inflammation is a key mediator in the connection between obesity, diabetes, coronary artery disease (CAD), and heart failure (HF) [34]. In obese individuals, the expansion of adipose tissue triggers immune cell recruitment and the excessive production of proinflammatory cytokines, leading to chronic low-grade inflammation [94]. This inflammation is a critical contributor to the development of insulin resistance, T2DM, and associated complications [94]. Similarly, systemic inflammation is commonly observed in HF patients, where it exacerbates the development, progression, and poor prognosis of the disease, regardless of LVEF [95]. Studies in animal models have suggested complex interactions between inflammatory pathways, which contribute to cardiac inflammation and the progression of diabetic cardiomyopathy [96, 97]. Notably, the inflammatory protein S100A12, which increases with hyperglycemic stress, has been linked to the risk of HF hospitalization in a cohort of 1,345 T2DM patients [98].

Epicardial Adipose Tissue Expansion

Both diabetes and obesity independently contribute to the expansion and transformation of epicardial adipose tissue, which has been associated with left ventricular systolic and diastolic dysfunction in T2DM patients [100]. The volume of epicardial fat correlates with myocardial fibrosis [101], vascular stiffness [102], and impaired coronary microcirculation [103]. The expansion and transformation of epicardial adipose tissue contribute to structural and functional alterations in the heart through mechanisms such as the proinflammatory effects of adipokines (e.g., leptin, TNF-α, IL-1β, and IL-6) secreted by epicardial fat, as well as oxidative stress induced by reactive oxygen species released from adipocytes [86, 104].

Autoimmunity

Autoimmunity plays a significant role in the pathogenesis of cardiovascular disease (CVD) in patients with T1DM [38, 105]. A recent prospective cohort study found a high prevalence of cardiac autoantibodies in T1DM patients with uncontrolled glycemia (HbA1c ≥9.0%), resembling the antibody profile seen in chronic Chagas cardiomyopathy [106]. Interestingly, the presence of cardiac autoantibodies was associated with elevated levels of high-sensitivity C-reactive protein and an increased risk of future CVD events. Conversely, T1DM patients with controlled glycemia (HbA1c <7.0%) exhibited lower levels of cardiac autoimmunity. The precise role of cardiac autoimmunity in the onset and progression of diabetic cardiomyopathy requires further investigation.

Overactivation of the Renin-Angiotensin-Aldosterone System

Overactivation of the renin-angiotensin-aldosterone system (RAAS) represents a pivotal pathophysiological link between diabetes, obesity, hypertension, and heart failure [34]. Elevated serum levels of angiotensin and aldosterone, along with the upregulation of their respective receptors, are characteristic features of RAAS overactivation [91, 107]. RAAS activation and dysglycemia are bidirectionally related: hyperglycemia and IR can activate RAAS, while RAAS in turn exacerbates systemic and cardiac IR and promotes oxidative stress in cardiomyocytes through NADPH oxidase activation [7].

Autonomic Dysfunction

Autonomic nervous system dysfunction is prevalent in individuals with diabetes. Cardiovascular autonomic neuropathy (CAN) differentially affects the sympathetic and parasympathetic components of cardiac innervation, resulting in sympathetic overactivation in early stages [16]. This imbalance is believed to contribute to the pathogenesis of cardiovascular disease. CAN induces left ventricular remodeling, systolic and diastolic dysfunction, and myocardial ischemia [108-110]. In the early stages, CAN is typically asymptomatic, but as it progresses, it may present with resting tachycardia, orthostatic hypotension, abnormal blood pressure regulation, a blunted heart rate response to exercise, and reduced heart rate variability [34, 111].

Myocardial Fibrosis

Myocardial fibrosis is a hallmark of diabetes-induced cardiac remodeling and dysfunction, detectable through histopathology or cardiac MRI. The extent of myocardial fibrosis is directly correlated with HbA1c levels in diabetic patients [112]. This fibrosis is characterized by the remodeling of the extracellular matrix, involving collagen deposition, perivascular fibrosis, basement membrane thickening, coronary microvascular sclerosis, and the formation of microaneurysms [16]. Myocardial fibrosis is considered a consequence of endothelial and vascular smooth muscle cell damage, as well as oxidative stress. It also contributes to the stiffening of the myocardium and the impairment of myocardial relaxation during diastole [113].

Prognostic Implications Of Diabetes In HF

Epidemiological and clinical trial data consistently demonstrate that individuals with both diabetes and heart failure (HF) experience a significantly impaired quality of life, are at heightened risk of hospitalization and mortality, and face an overall poor prognosis [33, 117]. Additionally, prediabetes, when present concurrently, further exacerbates the morbidity and mortality risks in patients with HF [118].

A substantial meta-analysis, encompassing over 380,000 subjects with acute and chronic HF from 43 registries and clinical trials, found that diabetes is associated with a 28% increase in all-cause mortality risk and approximately a 35% increase in both cardiovascular (CV) death and hospitalization risks, predominantly due to HF, over a three-year follow-up period [119]. Interestingly, the negative impact of diabetes on hospitalization and mortality risk remained consistent across various left ventricular ejection fraction (LVEF) groups (≤35% vs. >35%) but was notably more pronounced in individuals with chronic HF compared to those with acute HF. Observational studies also suggest a U-shaped relationship between HbA1c levels and mortality among patients with concurrent HF and diabetes. Specifically, a study by Aguilar et al. reported that HbA1c levels ranging from 7.1% to ≤7.8% were associated with the lowest mortality risk compared to other HbA1c quantiles in a cohort of ambulatory HF patients receiving medical therapy for diabetes in the early 2000s [120].

Several mechanisms have been proposed to explain the prognostic consequences of diabetes in HF. Diabetes is frequently associated with multimorbidity, which significantly worsens HF outcomes. Furthermore, diabetes leads to myocardial fibrosis, inflammation, and endothelial dysfunction, which contribute to increased left ventricular pressures, poor functional status, and reduced exercise capacity in HF patients [121–125]. Additionally, diabetes promotes neurohumoral overactivation and disrupts renal sodium handling, which can lead to congestion, cardiorenal syndrome, and diminished diuretic responsiveness [121]. Hyperglycemia in diabetic individuals increases the expression of sodium-glucose cotransporter 2 (SGLT2), enhancing renal sodium absorption and promoting volume expansion [121]. Moreover, the increased burden of ischemic heart disease and other diabetes-related comorbidities, such as chronic kidney disease (CKD), further exacerbates the deleterious effects of HF [126].

Data from the Swedish Heart Failure Registry revealed that the mortality risk associated with diabetes is more pronounced in individuals with ischemic HF compared to those with non-ischemic HF [43]. Specifically, the two-year survival rate for patients with HF, type 2 diabetes mellitus (T2DM), and ischemic heart disease was found to be less than 50%.

The presence of pre-existing microvascular disease has been identified as an independent risk factor for future HF events in patients with T2DM [44]. Furthermore, microvascular disease is associated with an increased risk of both mortality and morbidity in HF patients. A post hoc analysis of a large randomized controlled trial (RCT) revealed a significant association between a history of microvascular complications and an increased risk of adverse outcomes among diabetic patients with heart failure with preserved ejection fraction (HFpEF) [127].

Prevention Of Hf In Patients With Diabetes

The critical significance of HF prevention is underscored by the classification of risk factors such as hypertension and diabetes as stage A (at risk for HF) in HF staging [3]. Given the detrimental prognostic impact of clinical HF, the primary goals in managing diabetic patients include preventing asymptomatic cardiac remodeling and dysfunction (pre-HF, stage B) as well as symptomatic HF (stage C) [128].

Prevention of HF by Preventing Diabetes

The prevention of diabetes in early adulthood or middle age represents a highly effective strategy for mitigating the risk of developing HF later in life. An analysis of a large pooled cohort in the United States assessed the cumulative and relative impact of the absence of five modifiable HF risk factors—diabetes, hypertension, obesity, dyslipidemia, and smoking—during middle age (ages 45 to 55 years) [129]. This study demonstrated that the absence of diabetes in middle age significantly predicted HF-free survival, with individuals who did not have diabetes exhibiting a more than 60% lower risk of developing HF compared to those with diabetes. Furthermore, individuals free from diabetes, hypertension, and obesity at ages 45 to 55 had an average of more than 10 years longer HF-free survival and 13 years longer overall survival compared to those with all three risk factors [129].

Prediction of HF Risk in Patients with DM

Risk stratification for HF is essential in preventing the progression of HF in patients with diabetes or prediabetes who do not have established atherosclerotic cardiovascular disease (ASCVD). Although echocardiography can detect cardiac remodeling in diabetic patients, it is not routinely recommended for asymptomatic individuals due to concerns regarding cost-effectiveness [4]. However, measuring biomarkers such as natriuretic peptides or high-sensitivity cardiac troponin can aid in identifying patients at risk of progressing to HF or those already in the pre-HF stage. Therefore, it is recommended that diabetic patients undergo annual measurements of natriuretic peptides or high-sensitivity cardiac troponin to identify high-risk individuals and facilitate HF prevention [4].

Numerous risk prediction tools and algorithms have been developed to forecast the onset of HF in individuals with dysglycemia. Pandey et al. described a biomarker-based risk score incorporating high-sensitivity cardiac troponin T (≥6 ng/L), high-sensitivity C-reactive protein (≥3 mg/L), N-terminal pro-B-type natriuretic peptide (NT-proBNP) (≥125 pg/mL), and left ventricular hypertrophy (LVH) detected by ECG, with each abnormal parameter contributing one point. This risk score demonstrated good performance in predicting the 5-year incident HF risk in both diabetes and prediabetes cohorts from three major US cohort studies [130]. More complex risk prediction tools, such as WATCH-DM and TRS-HFDM, have also been developed, incorporating a broader range of clinical variables. Validation studies conducted in diverse clinical trial populations have confirmed that these tools are capable of stratifying HF risk in T2DM patients [131–133]. Nevertheless, prospective studies evaluating these risk scores are still lacking, and their clinical utility remains uncertain. Glycemic control, obesity management, and blood pressure (BP) regulation are well-established therapeutic approaches for reducing the risk of microvascular and macrovascular complications in patients with diabetes. The clinical implications of these approaches in HF prevention are discussed below.

Impact of Blood Pressure Control on HF Prevention

The coexistence of diabetes and hypertension is common, driven by overlapping risk factors and pathophysiological mechanisms [134, 135]. This dual condition synergistically contributes to the risk of

both microvascular and macrovascular complications, as well as cardiovascular diseases (CVD). Consequently, BP control through lifestyle modifications and antihypertensive medications is a primary objective for reducing complications in patients with both diabetes and hypertension [134]. The ACC/AHA guidelines recommend initiating antihypertensive treatment for diabetic patients with an office BP of ≥140/90 mmHg, with target BP levels set below 140/90 mmHg for low-risk patients and below 130/80 mmHg for those with established or high risk for atherosclerotic CVD [136].

BP lowering offers significant benefits in preventing HF among individuals with diabetes. However, the magnitude of this benefit is somewhat smaller in diabetic patients than in those without diabetes. Large meta-analyses of randomized controlled trials (RCTs) examining BP-lowering therapies revealed that every 10 mmHg reduction in systolic BP (SBP) was associated with a 16% to 25% reduction in HF risk in individuals with diabetes, compared to a 25% to 43% reduction in non-diabetic individuals [137, 138]. The landmark ACCORD BP (Action to Control Cardiovascular Risk in Diabetes Blood Pressure) trial, which compared intensive (SBP target <120 mmHg) versus standard BP control (SBP <140 mmHg), found that intensive BP control did not significantly reduce the risk of major adverse cardiovascular events (MACE) or HF but did increase the risk of adverse events. Consequently, the 2018 European Society of Cardiology (ESC)/European Society of Hypertension (ESH) guidelines recommend avoiding a target SBP <120 mmHg in diabetic patients [139, 140].

Among antihypertensive agents, thiazide diuretics, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and dihydropyridine calcium channel blockers are the most effective for cardiovascular risk reduction in diabetic patients. These agents can be considered first-line therapy for BP management in diabetes patients, with ACE inhibitors or ARBs being especially beneficial for those with albuminuria [136, 141]. However, beta-blockers are not recommended as first-line antihypertensive agents, as there is insufficient evidence supporting their mortality benefits when used solely for BP reduction in the absence of HF or coronary artery disease (CAD) [136, 141]. The landmark ALLHAT (Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack) trial, which compared the efficacy of various antihypertensive agents in preventing CVD, showed that chlorthalidone (a thiazide diuretic) outperformed lisinopril (an ACE inhibitor) and amlodipine (a calcium channel blocker) in preventing HF in both diabetic and non-diabetic patients [142, 143]. Subsequent meta-analyses confirmed that diuretics and renin-angiotensin system blockers were superior to other antihypertensive agents in preventing HF in diabetic individuals [138]. Loop diuretics have a neutral effect on glycemic control in diabetes, whereas thiazide diuretics may slightly increase fasting plasma glucose levels. Some studies suggest a link between thiazide diuretic use and new-onset diabetes in hypertensive patients, but this association is not universally supported by clinical evidence. Thiazide diuretics remain an important part of the antihypertensive regimen in diabetes patients at high risk for HF and stroke.

Obesity Management and Lifestyle Modifications

Studies consistently demonstrate that intentional weight loss through diet, exercise, or bariatric surgery positively impacts glycemic control, insulin resistance (IR), blood pressure (BP), and lipid profiles, while reducing the need for antidiabetic medications in obese individuals with type 2 diabetes mellitus (T2DM) (34,154,155). Furthermore, weight reduction can delay the progression from prediabetes to T2DM (156). The American Diabetes Association (ADA) recommends lifestyle modifications aimed at achieving at least a 5% weight loss for all overweight or obese individuals with prediabetes or diabetes (157). Additionally, individualized medical nutrition therapy is advised for diabetes management to help achieve treatment goals. The ADA suggests at least 150 minutes of moderate-to-vigorous aerobic activity per week, spread over at least three days, along with 2-3 sessions per week of resistance exercise for most individuals with T1DM and T2DM (157). Despite the well-documented benefits of weight loss for diabetes management, the role of lifestyle changes and weight reduction in preventing heart failure (HF) among diabetic patients remains uncertain. A meta-analysis of 36 prospective cohort studies prior to 2014 revealed that meeting the recommended physical activity levels (150 minutes per week of moderate-intensity aerobic activity) was associated with a reduced risk of incident HF (relative risk: 0.81 [0.76, 0.86]) in patients with diabetes (112).

The Look AHEAD (Action for Health in Diabetes) trial is the largest randomized controlled trial (RCT) investigating the cardiovascular effects of an intensive lifestyle intervention promoting weight loss through caloric reduction and increased physical activity in overweight or obese individuals with T2DM (154). Participants in the intervention group achieved an 8.6% weight loss at one year, maintaining a modest 6% weight loss at the 10-year follow-up, compared to a 3.5% weight loss in the control group. Despite these improvements in weight, physical fitness, and HbA1c, the intervention did not reduce cardiovascular disease (CVD) mortality or morbidity, including HF risk. However, a post hoc analysis by Pandey et al. showed that a 10% reduction in BMI over a four-year period resulted in a 20% reduction in incident HF risk. Additionally, higher baseline cardiorespiratory fitness and improvement over time were linked to a lower risk of incident HF, with a stronger correlation observed for HFpEF (heart failure with preserved ejection fraction) than for HFrEF (heart failure with reduced ejection fraction). This suggests the need for further studies focused on more intensive weight loss and exercise programs to promote sustained improvements in body weight and cardiorespiratory fitness among patients with T2DM (158).

Metabolic surgeries, when part of a comprehensive weight management approach, offer effective treatment options for achieving significant and lasting weight loss in individuals with severe obesity (159). For individuals with T2DM and severe obesity, metabolic surgery can improve glycemic control, insulin sensitivity, and even lead to diabetes remission (159). Evidence from RCTs and observational studies has shown that metabolic surgery, compared to conventional lifestyle modifications and medical therapy, reduces overall cardiovascular risk and enhances quality of life in individuals with T2DM and severe obesity (160–162). While the impact of metabolic surgeries on incident HF risk remains unstudied in large RCTs, a nationwide prospective observational study found that metabolic surgery was associated with more than a 50% reduction in the risk of incident HF (163). A retrospective cohort study from the Cleveland Clinic also demonstrated a 40% relative reduction in major adverse cardiovascular events and over a 60% reduction in incident HF risk among patients with T2DM and obesity following metabolic surgery over a median follow-up of 3.9 years (164).

Impact of Glycemic Control

In both T1DM and T2DM, intensive glycemic control significantly reduces the risk and severity of microvascular complications (34). However, despite strong epidemiologic evidence linking poor glycemic control with increased HF risk, the effects of intensive glycemic control on HF prevention remain inconclusive. Early clinical trials assessing the cardiovascular benefits of intensive glycemic control did not consider HF as a primary endpoint. However, post hoc or secondary analyses have begun to clarify the relationship between glycemic control and HF prevention. In the UK Prospective Diabetes Study (UKPDS), intensive glycemic control with metformin, sulfonylureas, or insulin was compared to conventional therapy in adults with recently diagnosed T2DM. A post hoc analysis of this study found that each 1% reduction in mean HbA1c was associated with a 16% decrease in incident HF (165). However, subsequent large-scale RCTs such as the ACCORD (166,167), ADVANCE (168), and VADT (169) failed to show a reduction in HF risk with intensive glucose control in patients with T2DM. A meta-analysis of these trials indicated no significant effect of intensive glucose control on HF risk, despite a modest 9% reduction in major cardiovascular outcomes (170). These findings suggest that simply lowering blood glucose and improving HbA1c may not be sufficient to prevent HF in diabetic patients.

Nursing Intervention Plan:

Managing patients with diabetes, cardiomyopathy, and heart failure (HF) requires an integrated approach to address the complexities of these conditions. Nurses play a critical role in patient care, focusing on monitoring, education, and supporting lifestyle changes that are essential for managing these chronic diseases effectively. The nursing interventions for these conditions are tailored to address the multifaceted needs of the patient, aiming to improve glycemic control, cardiac function, and overall quality of life.

Diabetes Management

The primary nursing intervention for diabetes management is promoting optimal blood glucose control. Nurses need to assess the patient's understanding of their condition, including the importance of regular blood sugar monitoring, medication adherence, and dietary management. The nurse should collaborate with the healthcare team to ensure the patient receives appropriate diabetes education. This includes teaching the patient how to administer insulin or other antidiabetic medications, recognize symptoms of hypoglycemia or hyperglycemia, and how to manage these situations effectively. Nurses can also assist with developing a personalized meal plan in collaboration with a dietitian, emphasizing the importance of carbohydrate counting, balanced meals, and portion control. Monitoring for complications related to diabetes, such as diabetic neuropathy, retinopathy, and kidney dysfunction, is crucial. Nurses should conduct regular assessments of the patient's feet for signs of neuropathy and infection, perform routine eye examinations, and monitor kidney function through lab tests. Moreover, nurses should educate the patient on the significance of regular check-ups and encourage them to follow up with their healthcare provider to detect complications early.

Cardiomyopathy Management

Cardiomyopathy, particularly in patients with diabetes, can exacerbate heart failure and result in diminished cardiac function. Nurses should assess the patient’s cardiac status, including vital signs, auscultation of heart sounds, and evaluation for signs of fluid retention, such as edema or weight gain. Monitoring the patient's blood pressure is essential to ensure it remains within the target range, as uncontrolled hypertension can further strain the heart. Nursing interventions for cardiomyopathy involve optimizing cardiac output and preventing further deterioration. Nurses should educate patients on the importance of adhering to prescribed medications, such as ACE inhibitors, beta-blockers, or diuretics, and monitor their response to these treatments. Additionally, nurses should teach patients about lifestyle modifications, including smoking cessation, reducing alcohol consumption, and adhering to a heart-healthy diet, such as a low-sodium, low-fat, and high-fiber diet, which helps reduce strain on the heart and manage fluid retention. Nurses should also assess for signs of arrhythmias or worsening heart failure and provide immediate intervention if needed. Arrhythmias, which are common in patients with cardiomyopathy, require prompt recognition and treatment, including medication adjustments or potential device implantation, such as a pacemaker or defibrillator. The nurse’s role in education and emotional support is vital, as patients may experience anxiety related to the progressive nature of their condition.

Heart Failure Management

For patients with heart failure, nursing interventions aim to improve symptoms, prevent hospitalization, and enhance overall cardiac function. Monitoring vital signs, especially heart rate, respiratory rate, and oxygen saturation, is essential. Nurses should also track daily weights, as sudden weight gain may indicate fluid retention and worsening heart failure. Assessing lung sounds for crackles or signs of pulmonary edema and checking for peripheral edema can help identify fluid overload early. Patients with HF often require diuretics to reduce fluid buildup. Nurses must educate patients on the importance of adhering to prescribed medications and monitor for adverse effects, such as electrolyte imbalances or dehydration. Diuretics should be taken as directed, and patients must be taught the significance of maintaining adequate hydration while avoiding excessive salt intake. Nurses should also ensure patients understand the importance of rest periods to reduce cardiac strain, while gradually increasing physical activity as tolerated to improve stamina and overall cardiovascular health. Lastly, nurses should focus on emotional support, recognizing that chronic illnesses like heart failure can lead to depression and anxiety. Offering counseling, connecting patients with support groups, and promoting mental health screenings are key components of comprehensive care. Nurses should continuously assess the patient’s psychological well-being and encourage open communication about fears, symptoms, and treatment concerns. In conclusion, the nursing intervention plan for diabetes, cardiomyopathy, and heart failure must be holistic, combining medical management with education, lifestyle modifications, and emotional support to improve

the patient's health outcomes and quality of life. Effective nursing care requires close monitoring, interdisciplinary collaboration, and a focus on patient empowerment and self-management.

Conclusion:

Diabetes and heart failure (HF) are intricately linked, with diabetes acting as an independent risk factor for the development and progression of HF. The bidirectional relationship between the two conditions complicates their management and exacerbates the prognosis for affected individuals. Diabetic cardiomyopathy, characterized by myocardial dysfunction due to diabetes in the absence of other causes, presents a unique challenge in clinical practice. The pathophysiological mechanisms underlying this condition are complex, involving disrupted glucose and fatty acid metabolism, insulin resistance, inflammation, and myocardial fibrosis. The prevalence of heart failure among patients with diabetes is significant, with studies revealing that up to 22% of diabetic individuals may be affected by this condition. This increased prevalence underscores the importance of recognizing diabetes as a primary risk factor for heart failure, particularly in the absence of traditional cardiovascular risk factors. Moreover, the transition from asymptomatic stages of heart failure to clinically significant HF is accelerated in diabetic patients, making early intervention crucial. Recent advances in the treatment of diabetes-related heart failure, particularly with the introduction of SGLT2 inhibitors, have revolutionized the management landscape. These medications have proven to not only improve glycemic control but also provide substantial benefits in reducing heart failure-related morbidity and mortality. As a result, SGLT2 inhibitors have become first-line therapy for patients with both type 2 diabetes and heart failure, regardless of the heart failure phenotype or ejection fraction. Furthermore, glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are also showing promising results in reducing cardiovascular events in diabetic patients. The evidence underscores the importance of early detection of heart failure in diabetic patients, particularly through the use of biomarkers such as natriuretic peptides, and imaging techniques to assess structural heart disease. Screening for pre-diabetes in individuals with heart failure is also critical, as pre-diabetes has been linked to an increased risk of both cardiovascular and all-cause mortality. The coexistence of diabetes and heart failure significantly worsens patient outcomes, highlighting the need for integrated care models that address both metabolic and cardiovascular components. Nurses play a vital role in managing these patients by providing education on lifestyle modifications, monitoring glycemic control, and administering pharmacological treatments. By focusing on early diagnosis and comprehensive management, healthcare providers can improve the quality of life and reduce the burden of heart failure in diabetic populations.

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داء السكري، اعتلال عضلة القلب، وفشل القلب - مقال مراجعة محدث للتمريض

الملخص:

الخلفية :تم التعرف على داء السكري كعامل خطر كبير لفشل القلب (HF) واعتلال عضلة القلب. يمتد تأثيره إلى ما وراء عوامل الخطر التقليدية لأمراض القلب والأوعية الدموية مثل ارتفاع ضغط الدم وأمراض الشرايين التاجية .(CAD) يساهم داء السكري في فشل القلب من خلال آليات متعددة، بما في ذلك مقاومة الإنسولين، وتغيير التمثيل الغذائي لعضلة القلب، والمسارات الالتهابية. ونتيجة لذلك، يتم تصنيف الأفراد المصابين بداء السكري على أنهم معرضون لخطر الإصابة بفشل القلب، مع معدلات انتشار أعلى بكثير في السكان المصابين بداء السكري مقارنة بالسكان العامين. أدت التقدمات الحديثة في فهم الفيزيولوجيا المرضية لفشل القلب المرتبط بالسكري إلى استراتيجيات علاجية جديدة، خاصة التي تتضمن مثبطات ناقل الصوديوم والجلوكوز المشترك-2.(SGLT2)

الهدف :يهدف هذا المقال إلى دراسة العلاقة بين داء السكري، اعتلال عضلة القلب، وفشل القلب، مع التركيز على الفيزيولوجيا المرضية، التشخيص، الآثار التنبؤية، خطط التدخل التمريضي، واستراتيجيات الإدارة.

المنهج :يوفر هذا المقال مراجعة شاملة للأدبيات الحالية بشأن انتشار ودراسة الفيزيولوجيا المرضية وعلاج فشل القلب المرتبط بداء السكري. يدمج المقال نتائج الدراسات الوبائية، التجارب السريرية، والتقدم في العلاج الدوائي، خاصة مثبطات SGLT2 ونظائر مستقبلات الببتيد الشبيه بالجلوكاجون-1 .(GLP-1 RAs) بالإضافة إلى ذلك، يناقش المقال أهمية الكشف المبكر وتصنيف أنماط فشل القلب في المرضى المصابين بالسكري.

النتائج :غالباً ما يتعايش داء السكري وفشل القلب، حيث يزيد السكري من خطر الإصابة بفشل القلب بمقدار يتراوح بين 1.7 إلى 2.5 مرة. يتراوح انتشار فشل القلب في المرضى المصابين بالسكري بين 9% و22%، وقد أظهرت التجارب الحديثة أن مثبطات SGLT2 تحسن بشكل كبير من نتائج المرضى المصابين بفشل القلب سواء كانوا مصابين بالسكري أو غير مصابين. علاوة على ذلك، يظل التحكم في مستوى السكر في الدم عاملاً مهماً في تقليل خطر الاستشفاء بسبب فشل القلب.

الخاتمة :إن تعايش داء السكري وفشل القلب يزيد بشكل كبير من سوء التنبؤ، مما يبرز الحاجة إلى الفحص المبكر والتدخل. تقدم العلاجات الدوائية الجديدة مثل مثبطات SGLT2 نتائج واعدة للمرضى الذين يعانون من الحالتين. يعد الكشف المبكر عن ما قبل السكري وداء السكري لدى الأفراد المصابين بفشل القلب أمرًا بالغ الأهمية لتحسين نتائج المرضى وتقليل خطر الأمراض القلبية الوعائية والوفيات.

الكلمات المفتاحية :داء السكري، فشل القلب، اعتلال عضلة القلب، مثبطاتSGLT2 ، الفيزيولوجيا المرضية، التحكم في مستوى السكر في الدم، التنبؤ