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Modern approaches to the diagnosis and treatment of heart failure in patients with type 2 diabetes mellitus

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Abstract. The aim of the study was to identify systemic limitations in the approaches to the diagnosis and treatment of chronic heart failure (CHF) in patients with type 2 diabetes mellitus (DM) based on the analysis of pathogenesis, verification methods, and treatment effectiveness. Within the framework of the theoretical approach, clinical-pathophysiological and organisational models of CHF in type 2 DM were reconstructed, followed by the comparison of diagnostic algorithms, therapeutic strategies, and statistical data from various national healthcare systems, along with an expert evaluation of the main pathogenetic mechanisms. During the theoretical analysis, it was established that the formation of CHF in type 2 DM was determined by the combined influence of diabetic cardiomyopathy, microvascular ischaemia, and chronic inflammation. Diagnostic algorithms based on ejection fraction (EF) assessment demonstrated limited sensitivity in patients with preserved systolic function, whose proportion reached 60-70%. The most informative early diagnostic methods were recognised as tissue Doppler echocardiography and the identification of fibrosis and overload biomarkers, including natriuretic peptides, soluble suppression of tumorigenicity-2 receptor, and galectin-3. The greatest evidence-based effectiveness in treating CHF in patients with type 2 DM was demonstrated by the combined use of four classes of drugs – angiotensin-converting enzyme inhibitors, β-adrenergic blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter 2 inhibitors (SGLT2 inhibitors). Significant differences in access to basic drug therapy were identified: up to 78% of patients in the USA received combined treatment, whereas in Kazakhstan – less than 45%. These discrepancies also appeared in mortality levels: 66.2 cases per 100,000 population in the USA, versus a marked increase in this indicator in certain regions of Kazakhstan. The obtained results substantiated the need to adapt clinical protocols considering phenotypic features of the combined pathology, which allows for improved diagnostic accuracy, individualised therapy, and reduced cardiovascular mortality

Keywords: myocardium; metabolic comorbidity; ischaemia; diastolic dysfunction; insulin; ejection fraction

Introduction

Chronic heart failure (CHF) in patients with type 2 diabetes mellitus (DM) formed a stable comorbid phenotype, associated with early development of myocardial dysfunction, atypical clinical manifestations, and a high rate of adverse outcomes. In clinical practice, difficulties in early disease verification were recorded, especially in cases with preserved ejection fraction (EF), which hindered timely initiation of therapy. Universal diagnostic protocols demonstrated limited sensitivity regarding subclinical forms, and therapeutic approaches did not take into account the pathophysiological specificity of metabolic disorders. The absence of adapted diagnostic and treatment schemes amid the progressive spread of type 2 DM increased the epidemiological burden and raised cardiovascular mortality. Collectively, these factors substantiated the need for a systematic theoretical analysis aimed at reconstructing pathogenetic models, assessing the effectiveness of diagnostic strategies, and identifying pharmacotherapeutic deficiencies in this patient category.

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The significance of heart failure in type 2 DM was observed in several Kazakhstani and international sources, which highlighted the scale and clinical-social relevance of this comorbidity. In the study by G. Zhakhina *et al.* [1], data from the national electronic healthcare system were analysed, and an increase in hospitalisations for heart failure was recorded, especially among patients with type 2 DM. However, the study lacked in-depth assessment of risk factors and the dynamics, which limited its prognostic value. O. Jumagazieva *et al.* [2] presented a general picture of mortality and prevalence of CHF, DM, and arterial hypertension within the framework of a chronic disease management programme. The authors stressed the importance of an interdisciplinary approach, but the analysis remained predominantly descriptive. In the Resolution of the Government of the Republic of Kazakhstan No. 945 "On Approval of the Concept of Health Care Development of the Republic of Kazakhstan until 2026" [3], strategic directions for healthcare development until 2026 were defined. Despite its regulatory comprehensiveness, the provisions concerning type 2 DM in the context of heart failure were virtually absent. An attempt to reflect the prevalence and management tactics of CHF was made in the review by Y. Kakhramonovna *et al.* [4], but the lack of methodological rigour diminished the reliability of the conclusions.

The diagnosis of CHF in patients with type 2 DM was complicated by the fact that the clinical picture in the early stages was often nonspecific, and standard algorithms were not adapted to the features of this population. The difficulties in detecting subclinical forms of the disease were associated by A. Ceriello *et al.* [5] with the low sensitivity of traditional methods, especially in the group with diabetic cardiomyopathy. The authors proposed a multiparametric diagnostic model, but its effectiveness had not yet been confirmed by large clinical trials. In the review by J.J. Park [6], special attention was paid to the role of diastolic dysfunction and subtle symptoms as the main causes of diagnostic delays. However, the author relied mainly on retrospective data, which limited the generalisability of the conclusions. The potential of using N-terminal pro-B-type natriuretic peptide (NT-proBNP), suppression of tumorigenicity 2 (ST2), and galectin-3 as early diagnostic markers was considered in the study by V. Castiglione *et al.* [7]. Nevertheless, according to the authors, the specificity of biomarkers in type 2 DM remained insufficiently studied.

Optimising CHF treatment in patients with type 2 DM was a clinically significant task, given the high likelihood of decompensation and limited effectiveness of universal therapeutic schemes in the context of metabolic disorders. In the consensus publication by R. Pop-Busui *et al.* [8], prepared with the participation of the American Diabetes Association (ADA), CHF was regarded as a systemic and underappreciated complication of DM, requiring integration into management algorithms for this population. Despite the comprehensive framing of the problem, the work lacked specific recommendations regarding pharmacotherapy. In the publication by P.A. Heidenreich *et al.* [9], developed by the Joint Committee on Clinical Practice Guidelines under the auspices of the American College of Cardiology (ACC), American Heart Association (AHA), and Heart Failure Society of America (HFSA), a universal medication management scheme for CHF was proposed, taking EF into account. This approach ensured clinical applicability to a broad population but did not provide for separate stratification for the subgroup of patients with type 2 DM, which limited the potential for personalised treatment. A detailed stepwise therapy algorithm for CHF, with emphasis on evidence and safety, was developed by the ACC Solution Set Oversight Committee and substantiated in the publication by T.M. Maddox *et al.* [10]. The authors outlined the principles of phased initiation and titration of major drug classes, but metabolic aspects – including the presence of type 2 DM, insulin resistance, and diabetic cardiomyopathy – were not systematically analysed or used as stratification factors.

The diagnosis and therapy of CHF in type 2 DM under conditions of metabolic comorbidity remained insufficiently developed, indicating structural gaps in existing research. The aim of the present study was to theoretically substantiate the clinical-pathophysiological principles of diagnosing and treating CHF in patients with type 2 DM. The objectives included: characterising the pathogenetic mechanisms of the relationship between type 2 DM and CHF; analysing key diagnostic approaches and the limitations; summarising current therapeutic strategies with an emphasis on the adaptation to the characteristics of this population; comparing indicators and organisational models of the USA and Kazakhstan, and assessing the potential for adapting the American experience to Central Asian countries.

Materials and Methods

The methodological foundation of the study was an interdisciplinary theoretical-analytical approach, which ensured a comprehensive examination of the pathophysiological, diagnostic, and therapeutic aspects of CHF in patients with type 2 DM, as well as a comparison of statistical indicators of the treatment of this condition in the USA and the Republic of Kazakhstan. During the analysis, analytical, inductive-deductive, comparative, and logical-synthetic methods were used. The application of the analytical method provided an in-depth interpretation of the mechanisms of the relationship between type 2 DM and CHF, including the specific features of diabetic cardiomyopathy. Inductive and deductive approaches made it possible to logically structure the argumentation: from the study of epidemiological trends and pathogenesis – to the analysis of diagnostic and therapeutic solutions. The comparative method was used when comparing data from clinical guidelines, biomarkers, instrumental approaches, and

national statistics. The logical-synthetic procedure ensured the integration of multilevel information – from molecular mechanisms to population indicators – into a single theoretical construct.

Within the theoretical model, an expert comparative assessment of the pathogenetic mechanisms of CHF development in patients with type 2 DM was carried out. The principle of conditional significance (from 1 to 10 points) was used, reflecting the frequency of reference to mechanisms in scientific literature, the clinical significance, and pathogenetic role. For statistical comparison, official data on the incidence and mortality from CHF in adult patients with type 2 DM in the Republic of Kazakhstan and the USA were selected. Sources included national reports prepared by the Centres for Disease Control and Prevention [11,12], as well as by the American Heart Association [13,14]. For Kazakhstan, data from the Unified National Electronic Health System and publications describing epidemiological trends in the post-pandemic and forecast periods [15,16] were analysed. The analysis covered the period 2020-2023, as this interval provided the most comparable and updated data.

Statistical comparison was conducted using key epidemiological indicators: prevalence of CHF among adults, mortality rate, hospitalisation frequency, disability-adjusted life years (DALY), and the proportion of patients with type 2 DM among all patients with heart failure. The analysis covered the dynamics of the cardiovascular disease burden in urban and rural populations. This approach made it possible to identify differences in treatment accessibility, medication adherence, and patient routing, as well as to determine directions for optimising the strategy for managing patients with CHF and type 2 DM. From a diagnostic perspective, the study analysed the following methods: clinical assessment of functional class according to the New York Heart Association classification [17]; echocardiography with determination of EF, left ventricular volume, and diastolic function; evaluation of myocardial structural changes (hypertrophy, fibrosis); laboratory diagnostics using NT-proBNP, ST2, galectin-3, and cardio-specific troponins. Particular attention was paid to the role of multi-biomarker panels used for early detection of subclinical heart failure, as well as the specificity of these indicators in the diabetic population. The study considered all key pharmacological classes approved by international guidelines for the treatment of CHF: sodium-glucose cotransporter-2 inhibitors (SGLT2 inhibitors), angiotensin-converting enzyme inhibitors (ACE inhibitors), angiotensin II receptor antagonists, beta-adrenergic blockers, and mineralocorticoid receptor antagonists.

The methodological approach, based on the use of theoretical analysis, systematic comparison, and structural interpretation of statistical and clinical data, ensured a comprehensive consideration of the problem of CHF in patients with type 2 DM. The statistical data were processed using Microsoft Excel (version 2303) and IBM SPSS Statistics (version 29.0), which ensured the reliability of calculations, the ability to visualise, and conduct comparative analysis between samples.

Results and Discussion

Clinical and pathophysiological aspects of the relationship between CHF and type 2 diabetes

In conformity with the general theoretical analysis, chronic heart failure and type 2 DM formed a stable biomedical link in which the pathogenesis of one condition accelerated and aggravated the manifestation of the other. Results from molecular, clinical-epidemiological, and imaging studies showed that the presence of type 2 DM increased the risk of developing CHF by 2-4 times. The course of heart failure in such patients was characterised by early clinical manifestation, reduced survival, and increased therapeutic resistance. Similar data were presented in the study by K. Nakamura *et al.* [18], which emphasised the importance of lipotoxicity and glucose-induced oxidative stress as key determinants of remodelling and early fibrotic transformation of the myocardium. These changes aggravated cardiac dysfunction independently of the severity of coronary atherosclerosis, indicating the independent pathogenetic significance of diabetic cardiomyopathy.

In the available literature, diabetic cardiomyopathy was considered a key link in the relationship between type 2 DM and CHF. Diabetic cardiomyopathy developed under the influence of chronic hyperglycaemia, systemic insulin resistance, microvascular ischaemia, chronic inflammation, and metabolic overload of the myocardium. At the histological level, this condition was characterised by cardiomyocyte hypertrophy, interstitial fibrosis, mitochondrial disorganisation, and reduced left ventricular compliance. In the study by C.X. Ma *et al.* [19], it was described that mitochondrial dysfunction, accompanied by impaired β-oxidation of fatty acids, led to an energy deficit in cardiomyocytes and reduced contractile capacity, especially under ischaemic conditions. The analysis showed that chronic hyperglycaemia was a central trigger of destructive processes in the myocardium. Through activation of the polyol pathway, accumulation of advanced glycation end-products, and generation of reactive oxygen species, it caused calcium imbalance, cardiomyocyte apoptosis, and diastolic dysfunction.

Simultaneously, pronounced insulin resistance developed, disrupting metabolic balance and the myocardium's ability to effectively utilise glucose. This contributed to a metabolic switch in cardiomyocytes towards fatty acid metabolism, increasing oxygen consumption and reducing energy efficiency. This shift towards "metabolic ischaemia" even in the absence of coronary stenosis was confirmed by experimental models described in the study by A. Groenewegen *et al.* [20], and corresponded to the concept of functional hypoperfusion in type 2 DM.

Based on reviews and clinical-experimental data, endothelial dysfunction was identified as the next important pathogenetic factor. In patients with type 2 DM, reduced bioavailability of nitric oxide, increased expression of vascular cell adhesion molecules (VCAM-1 and ICAM-1), and impaired vasomotor endothelial function were observed. This led to microvascular hypoperfusion and arteriole remodelling, especially in the subendocardial layers. In the study by B. Zareini *et al.* [21], it was emphasised that these changes underlay the increased mortality in diabetics with CHF compared to non-diabetic patients, even with a comparable level of coronary lesions.

A number of theoretical sources also focused on the role of chronic low-grade inflammation. In patients with type 2 DM, persistent levels of proinflammatory cytokines were recorded – interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β). These cytokines activated fibroblasts and induced collagen production, promoting the progression of interstitial fibrosis. Such mechanisms were particularly evident in CHF with preserved EF, where diastolic dysfunction was determined by reduced myocardial compliance. Similar pathogenetic characteristics were demonstrated in the study by S. Nazir *et al.* [22], which emphasised that even under standard therapy, the combination of type 2 DM and ischaemic myocardial injury significantly worsened the prognosis. In addition, the results of R.D. Ianos *et al.* [23] confirmed that this patient group exhibited pronounced activation of inflammatory cascades, accompanied by elevated levels of galectin-3 and ST2, which correlated with the severity of diastolic dysfunction and the volume of fibrotic transformation.

Furthermore, literature analysis showed that the systemic effects of type 2 DM on the cardiovascular system were realised through the development of atherosclerosis, endothelial dysfunction, and diabetic microangiopathy. Vascular imaging studies described intimal thickening, reduced arterial elasticity, and impaired coronary autoregulation, leading to functional ischaemia even in the absence of stenosis [21,22]. Diabetic angiopathy affected not only large vessels but also arterioles and capillaries, reducing capillary density and vasodilatory reserve. This formed the substrate for latent ischaemic myocardial damage, especially under overload conditions. Comparable conclusions were presented in reports by the Centers for Disease Control and Prevention [11,12], which emphasised the high prevalence of cardiovascular complications in people with DM, especially those over 50 years of age, and highlighted the link between microvascular disorders and increased mortality. In the global statistical review by S.S. Martin *et al.* [13], data were shown on increased frequency of heart failure and mortality among patients with type 2 DM, particularly in the presence of dyslipidaemia and concomitant arterial hypertension. The described vascular changes were exacerbated by atherogenic dyslipidaemia – hypertriglyceridaemia, reduced levels of high-density lipoproteins, and hyperinsulinaemia, as noted by D.A. Serebrennikova [24] – intensifying tissue hypoperfusion and myocardial remodelling.

The totality of the described pathogenetic mechanisms allows the formation of an integrated conceptual model reflecting the key links in the development of CHF in patients with type 2 DM. Despite the complexity of the interactions, current data allow for a conditional differentiation of the relative contribution of each based on experimental, clinical, and epidemiological observations (Fig. 1).

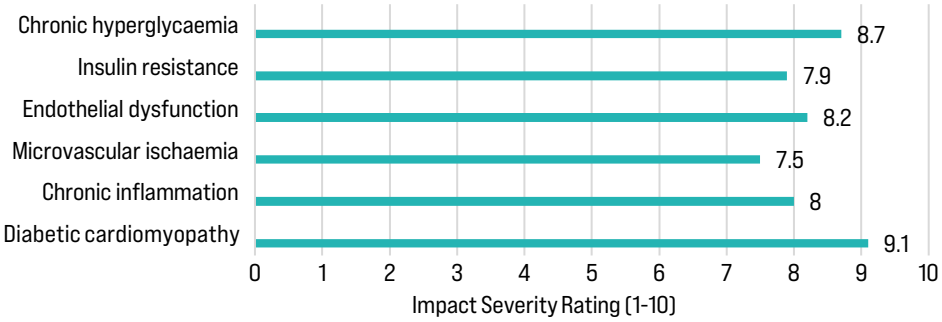

Figure 1. Relative severity of key pathogenetic mechanisms of CHF in type 2 diabetes
Source: created by the author based on data analysis [18-24]

The analysis of the presented data highlighted the systemic and hierarchically organised nature of the pathogenesis of CHF in type 2 DM. Among the six key mechanisms, diabetic cardiomyopathy received the highest score (9.1 points), indicating its integrative nature as a morphofunctional endpoint of metabolic, vascular, and inflammatory disorders. The high scores for chronic hyperglycaemia (8.7), endothelial dysfunction (8.2), and chronic inflammation (8) indicated that the primary triggers of cardiac dysfunction formed long before EF decline and clinical decompensation, playing a decisive role in disrupting the metabolic stability of the myocardium and vascular tone. Insulin resistance (7.9) and microvascular ischaemia (7.5), although less pronounced, acted as critically important modulators, exacerbating energy deficiency and contributing to a

reduced adaptive reserve of the heart. The presented model visualised the functional interaction of pathogenetic links and emphasised the need for multilevel intervention aimed not only at symptom control but also at early correction of underlying disorders, including tissue insulin resistance, low-grade inflammation, and subclinical myocardial damage.

The relationship between CHF and type 2 DM was not limited to clinical correlation but was determined by the complex interaction of cardiometabolic, vascular, and immune mechanisms. These mechanisms formed a specific phenotype of heart failure resistant to standard therapy and required personalised strategies of diagnosis and treatment. Scientific understanding of the pathophysiological foundations of this comorbidity represented an important step towards improving the effectiveness of medical intervention in this high-risk population.

Diagnostic approaches and limitations of CHF verification in patients with type 2 diabetes

The diagnosis of CHF in patients with type 2 DM required consideration of multiple pathophysiological and clinical-diagnostic factors specific to this comorbid group. According to the literature, in patients with type 2 DM, CHF progression occurred in the absence of classic signs of systolic dysfunction, which was associated with the high prevalence of the diastolic form of heart failure. The proportion of CHF with preserved left ventricular EF in this population reached 60-70% of cases, and traditional EF assessment methods (e.g., two-dimensional echocardiography) did not allow a reliable diagnosis at early stages [23,24]. These forms were accompanied by impaired active relaxation of the left ventricle, reduced compliance, and the development of hidden fibrosis, which remained undiagnosed for a long time. Comparable conclusions were presented in the Chinese clinical guidelines by S. Zhang *et al.* [25], which emphasised the necessity of comprehensive assessment of filling parameters, left atrial volume, and longitudinal strain as an essential element of diagnosis in the diabetic population.

Electrocardiography remained a mandatory component of initial diagnosis when CHF was suspected, including in patients with type 2 DM. Special attention was paid to the detection of atrial fibrillation, ischaemic changes, signs of left ventricular hypertrophy, and conduction disorders including left bundle branch block. Although electrocardiography had limited specificity, its findings served as the basis for referring the patient for advanced imaging studies, including echocardiography and biomarker analysis. In the systematic review by C.W. Wong *et al.* [26], it was emphasised that diagnostic errors of CHF in patients with type 2 DM were often associated with the nonspecificity of symptoms and lack of alertness at the primary care level. Echocardiography, as the basic imaging method, demonstrated limited sensitivity under type 2 DM conditions. These indicators enabled verification of diastolic dysfunction at normal EF values. Data from the study conducted by H. Akasaka *et al.* [27] indicated that the use of tissue Doppler imaging with measurement of diastolic relaxation velocities and longitudinal strain enabled the detection of hidden forms of CHF in patients with type 2 DM.

Gadolinium-enhanced magnetic resonance imaging provided an opportunity to assess diffuse fibrosis and extracellular matrix volume, especially in patients with inconclusive echocardiography data or clinical deterioration despite normal EF [25]. According to S. Kulbayeva *et al.* [16], the use of magnetic resonance imaging in the diagnosis of CHF in patients with type 2 DM increased the sensitivity of detecting fibrotic changes, especially in the early stages of decompensation. In particular, in the case of uninformative echocardiography or mismatch between clinical picture and imaging findings, MRI was considered the preferred method of clarification.

Biochemical diagnostics were based on the use of NT-proBNP as a marker of haemodynamic overload. According to ADA recommendations [28], its use was advised for initial screening and treatment monitoring. NT-proBNP elevation was observed even in the absence of pronounced clinical symptoms, in line with the concept of subclinical volume overload. Additional biomarkers of inflammation and fibrogenesis – ST2, Galectin-3, copeptin, FGF21 – were also used, improving the sensitivity of diagnosis in preserved EF [23]. The findings presented by F. Yasmin *et al.* [29] confirmed that the combined use of NT-proBNP, copeptin, fibroblast growth factor 21 (FGF21), Galectin-3, and copeptin increased diagnostic sensitivity in hidden forms of CHF in patients with type 2 DM and reduced exercise tolerance. Such a multibiomarker approach became especially important in the context of DM, in which traditional clinical criteria were often uninformative. This aligned with integrated recommendations by Z. Bloomgarden & Y. Handelsman [30], prepared in collaboration with AHA, ADA, the European Association for the Study of Diabetes, and other organisations, emphasising the need to expand the range of diagnostic markers for CHF in patients with metabolic disorders.

Late diagnosis of CHF in patients with type 2 DM was explained by the influence of diabetic neuropathy, reduced sensitivity to physical activity, and symptom masking due to metabolic adaptation. As noted in the work by R. Indrakumari *et al.* [31], traditional clinical scales, including the New York Heart Association classification, demonstrated low sensitivity in the diabetic population due to autonomic dysfunction and attenuated manifestation of decompensation. Interdisciplinary studies analysed the integration possibilities of digital algorithms based on machine learning methods – logistic regression, decision trees, neural network models – into the diagnostic process structure [29]. These methods demonstrated high predictive

accuracy (AUC from 0.81 to 0.89) in recognising hidden forms of CHF, relying on the synthesis of heterogeneous clinical-laboratory and imaging data, including levels of natriuretic peptides, body mass index, heart rate (HR), and echocardiographic parameters. In the study by O. Akbulgic *et al.* [15], it was demonstrated that the use of big data analysis platforms and telemedicine solutions not only increased the detection of subclinical forms of CHF but also contributed to the individualisation of patient routing in the context of comorbidity-related healthcare overload.

Late diagnosis of CHF in patients with type 2 DM was due to a combination of factors influencing the interpretation of clinical symptoms and the choice of diagnostic tools. It was established that in the presence of diabetic neuropathy and reduced sensitivity to physical activity, classic signs of decompensation, including dyspnoea and fatigue, could be mildly expressed, reducing the effectiveness of clinical stratification. The low informativeness of traditional functional status scales, such as the New York Heart Association classification, in patients with type 2 DM was associated with autonomic regulation disorders and compensatory adaptation of the myocardium to metabolic overload [26]. To improve the accuracy of CHF diagnosis under metabolic comorbidity, alternative algorithms were developed based on digital computational methods. Neural network models are applied in clinical practice to integrate clinical-laboratory data, imaging studies, and anamnesis indicators to automatically identify patients at high risk of CHF with type 2 DM. These systems allow analysis of parameters that go beyond linear dependencies, including hidden interactions between biomarkers, echocardiographic parameters, and demographic characteristics [29]. Predictive modelling methods based on logistic regression, decision trees, and other machine learning algorithms were also used to develop diagnostic tools focused on outpatient monitoring data. Input variables included glucose level, body mass index, HR, blood pressure, and natriuretic peptide concentrations (NT-proBNP), which enabled the creation of accessible and scalable primary screening schemes [31].

Systematisation of approaches to diagnosing CHF in patients with type 2 DM allowed key verification directions to be identified. Methods covered clinical, imaging, biochemical, and digital levels, reflecting the need for a comprehensive approach. The diagnostic value varies depending on the form of CHF and the severity of metabolic disorders, requiring the combined application within a unified clinical model (Table 1).

Table 1. Complex of diagnostic approaches to verifying CHF in patients with type 2 diabetes

Direction of diagnostics	Methods and parameters	Features of type 2 diabetes
Clinical assessment	Collection of anamneses, physical examination, New York Heart Association functional class scale	Reduced sensitivity to symptoms due to diabetic neuropathy, obesity and myocardial adaptive overload
Electrocardiography	Detection of ischaemic changes, atrial fibrillation, bundle-branch block, left ventricular hypertrophy	Often gives non-specific data; requires correlation with cardiac imaging
Echocardiography	EF, ratio of early transmittal filling velocity to early diastolic relaxation velocity (E/e′), left atrial volume, left ventricular myocardial mass index, diastolic function parameters	Type 2 DM often preserves normal EF; relaxation and filling pressures are important
Magnetic resonance imaging of the heart	Determination of local and diffuse fibrosis, extracellular matrix volume, micronecrosis zones	Detects myocardial remodelling in CHF with preserved EF, especially when echocardiography is normal
Natriuretic peptides	NT-proBNP	Elevated in volume overload; used for early screening and monitoring of response to treatment
Biomarkers of fibrosis and inflammation	Soluble tumour suppressor receptor ST2, galectin-3, coeptin, FGF21 growth factor	Reflects fibroblast activation, interstitial inflammation and metabolic abnormalities in myocardium in type 2 DM
Tissue Doppler echocardiography	Early (e′) and late (a′) diastolic relaxation velocities, E/e′ ratio, global longitudinal strain (deformation)	Main method for preclinical assessment of diastolic dysfunction in patients with type 2 DM and preserved EF
Artificial intelligence	Neural network models, logistic regression, decision trees	Used for automated risk stratification based on a combination of clinical, laboratory and imaging data

Source: created by the author based on data analysis [25-31]

The structure of the presented diagnostic solutions demonstrated the necessity of alignment between the stages of verification and the nature of clinical uncertainty in type 2 DM, as traditional methods – such as assessment using the New York Heart Association scale and standard echocardiography – had limited sensitivity in latent forms of heart failure. The diagnostic framework was built as a system of interconnected levels – from preliminary clinical suspicion to molecular confirmation, with each method compensating for the limitations of others. This multistep approach made it possible to account for atypical features characteristic of the diabetic population, including muted symptoms, hidden diastolic dysfunction, and subclinical inflammation. It was precisely logical complementarity, rather than the prioritisation of a single approach, that formed the basis of an effective risk stratification model in this category of patients.

Modern approaches to the diagnosis of CHF in patients with type 2 DM have formed a qualitatively new paradigm, based on the combination of morphofunctional monitoring, remodelling biomarkers, and digital assessment algorithms. Exclusive reliance on traditional criteria proved unacceptable in the context of metabolic comorbidity, where the pathogenesis was polymorphic and clinical manifestation often lagged behind structural changes. Adequate diagnosis in such cases required the integration of advanced imaging, multi-biomarker panels, and intelligent tools, which significantly increased the accuracy of early detection and enabled timely initiation of pathogenetically justified therapy.

Current treatment strategies and pharmacotherapeutic deficiencies

Treatment of CHF in patients with type 2 DM requires multilevel intervention, taking into account the pathophysiological characteristics of metabolic comorbidity. Standardised treatment regimens are based on four fundamental pharmacological approaches that act on neurohumoral regulation, myocardial stress, and the rheological properties of the vascular bed. Each approach is implemented through separate drug classes, whose choice depends on the clinical form of CHF, the stage of renal dysfunction, and the functional reserve of the myocardium.

Targeting the renin-angiotensin-aldosterone system remained a fundamental approach in reduced EF (<40%). The use of ACE inhibitors was associated with afterload reduction and a lower risk of remodelling. In cases of ACE inhibitor intolerance (e.g., angioedema), angiotensin II receptor blockers were used, demonstrating comparable efficacy. Both drug classes showed a favourable profile in patients with type 2 DM but required monitoring of potassium levels and glomerular filtration rate (GFR). This aligned with the view of J. Bauersachs [32], who emphasised that modulation of the angiotensin-aldosterone system formed the basis of the so-called "great four" CHF therapy (ACE inhibitors/angiotensin II receptor blockers, β-blockers, mineralocorticoid receptor antagonists, SGLT2 inhibitors).

In patients with type 2 DM and symptomatic CHF with a heart rate ≥70 bpm on sinus rhythm, slowing of heart rate with β-adrenergic blockers was considered appropriate. The use was associated with inhibition of sympathetic activation, improved diastolic filling parameters, and reduced risk of life-threatening arrhythmias. It was established that in this category of patients, β-blockers remained effective even in the presence of obesity and insulin resistance. Regular monitoring of blood pressure and heart rate was required during titration [30]. Similar data were presented by B. Bozkurt [33], who emphasised that β-adrenergic blockers reduced the risk of sudden cardiac death and improved prognosis even in the presence of insulin resistance, confirming the necessity in patients with type 2 DM and CHF. In patients with EF ≤35% and stable electrolyte profile, inclusion of mineralocorticoid receptor antagonists such as spironolactone and eplerenone was indicated. The prescription was based on the need to block the effects of aldosterone, a key mediator of sodium retention and stimulator of myocardial fibrosis.

In patients with type 2 DM, this drug group additionally provided an antiproteinuric effect and could be used in patients with diabetic nephropathy at GFR >30 ml/min/1.73 m^2. The key innovation in CHF therapy in type 2 DM was the use of SGLT2 inhibitors. The inclusion in treatment algorithms was substantiated by studies showing a reduced risk of hospitalisation with any EF form. Drugs in this group, including empagliflozin, dapagliflozin, and ertugliflozin, did not require titration, demonstrated a favourable safety profile at GFR >20 ml/min/1.73 m^2, and were able to stabilise metabolic indicators without increasing the risk of hypoglycaemia. In cases of persistent tachycardia despite adequate β-blocking therapy, heart rate correction using ivabradine was allowed. This drug acted selectively on the If-channels of the sinus node, reducing heart rate without negative inotropic effect. Studies demonstrated its metabolic neutrality in type 2 DM and improved exercise tolerance in patients with CHF.

All guidelines (AHA, ACC, ADA, ESC) proposed stratified algorithms for drug prescription taking into account the heart failure phenotype, stage of renal insufficiency, arrhythmia risk, and congestion symptoms severity. Combined regimens were adapted for patients with type 2 DM and aimed at simultaneously affecting haemodynamics, myocardial structure, and the vascular bed. Similar principles of systemic stratification were outlined by Z. Bloomgarden & Y. Handelsman [30], who emphasised the importance of integrating recommendations from AACE, ADA, the European Association for the Study of Diabetes, AHA, ACC, and ESC into a unified patient management model for those with metabolic and cardiac risk. The authors stressed the need for multilevel consideration of comorbidities, including diabetic nephropathy, obesity, and cognitive impairment in elderly patients. Despite the availability of structured international guidelines, real-world clinical practice revealed significant deviations from optimal therapeutic regimens. According to multicentre observational data, the proportion of patients with type 2 DM and CHF receiving the full spectrum of recommended drug classes (ACE inhibitors or angiotensin II receptor blockers, β-blockers, MRAs, SGLT2 inhibitors) was less than 30% [32].

Similar barriers were identified by M. Jarjour *et al.* [34], who found that only 18% of patients with type 2 DM and CHF received guideline-concordant therapy. Furthermore, clinical inertia was recorded in 42% of cases, manifested in the absence of therapy initiation or escalation despite indications. The authors emphasised that the key limiting factors were physiological

(hypotension, reduced GFR), physicians' subjective assessment of frailty in older patients, and poor integration of interdisciplinary approaches. In elderly patients, dose titration was often not feasible due to poor exercise tolerance, unstable haemodynamics, and symptomatic hypoglycaemia, further limiting the implementation of therapy optimisation guidelines.

Additionally, cases of clinical inertia were described when patient conditions deteriorated – with no modification of the treatment regimen even in the presence of CHF progression, despite the availability of alternative drug classes. On the patient side, reduced adherence to therapy was observed when using multicomponent regimens, especially in the presence of cognitive dysfunction and polypharmacy. In the Republic of Kazakhstan and other countries in Central Asia and Eastern Europe with limited healthcare funding, insufficient access to SGLT2 inhibitors, GLP-1 receptor agonists, and angiotensin receptor-neprilysin inhibitors (ARNIs) was noted, which limits full implementation of international recommendations [30].

Modern approaches to CHF therapy in patients with type 2 DM were based on combined use of pathogenetically oriented drug classes, taking into account the clinical phenotype. Alongside the proven efficacy of basic regimens, systemic and individual limitations were identified that hindered the full implementation, especially in resource-limited countries. This underscored the need for further adaptation of therapeutic strategies to the specifics of metabolic comorbidity and the healthcare context.

Comparative analysis of statistical indicators of CHF treatment in type 2 DM in the USA and Kazakhstan

The comparative analysis of the epidemiological characteristics of CHF in patients with type 2 DM in the USA and the Republic of Kazakhstan revealed significant differences in disease burden, the structure of medical care, and pharmacotherapeutic availability. At the same time, it should be noted that the presented data from the USA and Kazakhstan partially cover different time intervals within 2020-2023, which may affect the degree of comparability of indicators. The lack of full synchronisation by year of analysis should be considered a methodological limitation, which does not diminish the significance of the identified differences but requires caution in interpreting trends.

According to the data from the US National Centre for Health Statistics, in 2022 more than 6.7 million adults were registered with a CHF diagnosis, of whom about 40% had concomitant type 2 DM [11]. The overall prevalence rate of CHF among the adult population in the USA during 2020-2023 ranged from 2-2.1% (an average of about 6.9 million people), while the mortality rate from heart failure among patients with type 2 DM reached 66.2 cases per 100,000 population. The rate of repeated hospitalisations due to decompensation within 30 days consistently exceeded 21%, reflecting high clinical instability in this group of patients [12]. According to the AHA report, covering retrospective estimates up to the end of 2023, the specific contribution of CHF in type 2 DM to the total burden of cardiovascular diseases was estimated at 7.3% of all DALYs, which corresponded to more than 2 million years of life lost adjusted for disability [13].

In the Republic of Kazakhstan, according to the analysis of the Unified National Electronic Health System, by 2022 the diagnosed prevalence of CHF among the adult population reached 2.2% (22,088 cases per million), whereas during 2020-2023 this indicator was on average assessed at 0.85%, significantly lower than in the USA. This was considered a reflection of underdiagnosis of latent forms of the disease, primarily CHF with preserved EF, and limited coverage by screening programmes [22]. The proportion of patients with type 2 DM in the structure of all CHF cases reached 38.4%, comparable to US data. Mortality from CHF in type 2 DM in the northern and eastern regions of Kazakhstan during the specified period reached 82.7 cases per 100,000, exceeding similar indicators in the USA [16]. Although the DALY rate for CHF in type 2 DM in Kazakhstan was not directly calculated, predictive models for Central Asia suggest that the total losses from cardiovascular diseases and DM by 2030 may exceed 4,000 per 100,000 women of reproductive age [15].

Comparison of CHF mortality rates in patients with type 2 DM in the USA (66.2 per 100,000) and Kazakhstan (82.7 per 100,000) revealed a range of structural, organisational, and therapeutic factors explaining the observed differences. Higher mortality in the Kazakhstani population was explained by limited access to modern pharmacotherapy (particularly SGLT2 inhibitors and ARNIs), poor implementation of systematic screening programmes, and insufficient detection of CHF with preserved EF in patients with oligosymptomatic courses. In contrast, the USA widely used multidisciplinary outpatient approaches, centralised registries, and telemonitoring, which ensured continuity of observation. According to F. Cosentino *et al.* [35], the use of the SGLT2 inhibitor ertugliflozin reliably reduced the risk of CHF hospitalisation by 30% in patients with type 2 DM and established atherosclerotic cardiovascular diseases. These data confirmed the therapeutic significance and feasibility of widespread use of this drug class in the cardiometabolic population. In Kazakhstan, however, the limited introduction of SGLT2 inhibitors into clinical practice was explained by both economic barriers and the absence of local protocols integrating the results of international randomised controlled trials. In the USA, on the contrary, such drugs were actively implemented due to insurance coverage policies and AHA/ACC/ADA recommendations. Additionally, according to A.J. Nelson *et al.* [36], even in the USA a significant gap remained between the evidence base and its implementation: only 55% of patients with

type 2 DM and atherosclerotic cardiovascular diseases received all recommended drug classes. Nevertheless, the same study emphasised that coverage with combination therapy in insured systems significantly exceeded 70%, especially in specialised clinics. This is comparable to data showing 78% coverage in EF cohorts in the USA [11], while in Kazakhstan a similar indicator was less than 45% [17]. Thus, the observed excess mortality in Kazakhstan could be explained not only by differences in drug availability but also by the absence of systematic routing and insufficient implementation of the multidisciplinary approach, which is especially critical for patients with type 2 DM and CHF. The lack of a CHF register for type 2 DM, underdevelopment of telemonitoring, and fragmented follow-up further reduced the potential for long-term disease control.

Based on the identified differences in epidemiological profile, routing structure, and therapy coverage, a step-by-step implementation of elements of the American model of cardiometabolic care adapted to the resource and systemic features of Kazakhstan's healthcare was considered appropriate. First and foremost, diagnostics needed modernisation: the introduction of comprehensive diagnostic circuits with mandatory use of tissue Doppler echocardiography, biomarkers (BNP, NT-proBNP, sST2, Gal-3), and expanded assessment of diastolic function. A review of early detection and screening protocols in individuals with type 2 DM was necessary – especially in high-risk groups. Active application of stratification scales (e.g., H2FPEF score, MAGGIC) and centralised training of primary care specialists was recommended.

The next area was the optimisation of routing and dispensary observation. In the USA, the "cardiometabolic clinics" model proved effective, where patients received interdisciplinary supervision from a cardiologist, endocrinologist, and nurse-mentor. For Kazakhstan, the format of regional multidisciplinary centres at major hospitals with telemonitoring and remote therapy adjustment functions remained potentially applicable. The study by A. Sciacqua *et al.* [37] highlighted that it was the interdisciplinary approach and extended outpatient monitoring of elderly patients with CHF and type 2 DM that reliably reduced repeat hospitalisation rates and improved treatment adherence, especially in the presence of concomitant cognitive and functional disorders. Also important was the formation of a national register of CHF in type 2 DM, linked to electronic health systems, which would allow tracking of therapy coverage dynamics, mortality, and hospitalisation rates in real time. From the pharmacotherapy standpoint, government support was required for reimbursement programmes of new drug classes – SGLT2 inhibitors and ARNIs – as well as the development of a national formulary synchronised with international guidelines (AHA/ACC/HFSA, ESC). Given the high cost of these drugs, it was justified to include these drugs in preferential categories for priority groups, which would improve treatment adherence and reduce the burden of repeat hospitalisations.

Despite certain similarities in the comorbidity structure, the USA and Kazakhstan demonstrated different levels of repeated hospitalisations, mortality, and coverage with effective therapy. These differences were partially explained by disparities in medication availability, care organisation, and healthcare financing systems.

Conclusions

The obtained results confirmed that the pathogenesis of CHF in patients with type 2 DM was caused by the combined influence of metabolic, vascular, and inflammatory disorders, leading to progressive myocardial remodelling. The formation of a specific CHF phenotype in this population was explained not by an isolated factor but by the cumulative effect of chronic hyperglycaemia, insulin resistance, endothelial dysfunction, and intercellular inflammation. Such a multicomponent nature of the lesion required a shift in focus from late compensation to early prevention, aimed at stabilising the cardiometabolic background even before the onset of pronounced systolic dysfunction.

In the diagnostic aspect, it was established that up to 60-70% of patients with type 2 DM had CHF with preserved EF, in which standard echocardiography proved insufficient. Accuracy was improved through a comprehensive assessment of diastolic parameters, biomarkers (natriuretic peptides, sST2, galectin-3), and digital technologies, including neural network models with a risk stratification accuracy of up to 90%. The pharmacotherapy analysis showed that a combination of ACE inhibitors/angiotensin II receptor blockers, β-blockers, MRAs, and SGLT2 inhibitors improved prognosis and reduced hospitalisation rates by 20-25%. However, in Kazakhstan, the coverage of basic therapy was less than 45% compared to 78% in the USA, which was explained by limited drug availability, clinical inertia, and weak primary-level referral systems. A comparative analysis of statistics from 2020-2023 revealed higher mortality from CHF in type 2 DM in Kazakhstan (up to 82.7 per 100,000) compared to the USA (66.2 per 100,000), despite a similar prevalence of DM. Differences in rehospitalisation rates and treatment coverage indicated the importance of systemic factors – insurance coverage, digital monitoring, and standardised referral pathways – in improving outcomes and reducing the epidemiological burden.

A limitation of this study was the absence of primary clinical data and the inability to perform statistical processing of empirical samples. A promising direction for further research may be the development of stratified patient management models for CHF and type 2 DM based on real registry data in countries with different healthcare systems.

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Conflict of Interest

None.

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2-типтеги кант диабети менен ооругандардын жүрөк жетишсиздигин диагностикалоого жана дарылоого заманбап ыкмалар

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Аннотация. Иштин максаты патогенезин, текшерүү ыкмаларын жана терапиянын эффективдүүлүгүн талдоонун негизинде 2-типтеги кант диабети (КД) менен ооруган бейтаптарда өнөкөт жүрөк жетишсиздигинин (ӨЖЖ) диагностикасына жана дарылоосуна карата системалуу чектөөлөрдү аныктоо болгон. Теоретикалык мамиленин алкагында 2 типтеги КДКнын клиникалык, патофизиологиялык жана уюштуруучулук моделдери диагностикалык алгоритмдерди, терапиялык стратегияларды жана саламаттыкты сактоонун ар кандай улуттук системаларынын статистикалык маалыматтарын кийинки салыштыруу менен реконструкцияланган жана алдыңкы патогенетикалык механизмдерге эксперттик баа берилген. Теориялык талдоо 2-тип кант диабетинде ӨЖЖ өнүгүшү диабеттик кардиомиопатиянын, микроваскулярдык ишемиялардын жана өнөкөт сезгенүүнүн биргелешкен таасири менен аныкталганын аныктады. Эжекциялык фракцияны (ЭФ) баалоого негизделген диагностикалык алгоритмдер жыйрылышы функциясы сакталган пациенттерде чектелген сезгичтикти көрсөттү, алардын үлүшү 60-70 % га жетти. Эрте диагностиканын эң маалыматтуу ыкмалары болуп ткандардын доплер эхокардиографиясы жана фиброздун жана ашыкча жүктөмдүн биомаркерлерин аныктоо, анын ичинде натриуретикалык пептиддер, шишиктин эрүүчү рецепторлорун басуу-2 жана галектин-3 таанылды. 2-типтеги кант диабети менен ооруган бейтаптарда ӨЖЖ дарылоодо эң чоң далилдүү эффективдүүлүк төрт класстагы дары-дармектерди айкалыштыруу менен көрсөтүлдү: ангиотензинди айландыруучу фермент ингибиторлору, β-блокаторлор, минералокортикоиддик рецепторлордун антагонисттери жана натрий-глюкозанын ингибиторлору (SLT2 типтеги ингибиторлору). Негизги дары-дармек терапиясын камтууда олуттуу айырмачылыктар аныкталды: АКШда пациенттердин 78 %ке чейинкиси комбинацияланган терапияны алышкан, ал эми Казакстанда 45 %тен аз болгон. Бул карама-каршылыктар өлүмдүн көрсөткүчүндө да чагылдырылган: АКШда 100 000 калкка 66,2 учур, Казакстандын айрым аймактарында бул көрсөткүчтүн олуттуу өсүшү. Алынган натыйжалар диагноздун тактыгын жогорулатууга, терапияны индивидуалдаштырууга жана жүрөк-кан тамыр ооруларынан өлүмдү кыскартууга мүмкүндүк берген комбинирленген патологиянын фенотиптик өзгөчөлүктөрүн эске алуу менен клиникалык протоколдорду адаптациялоо зарылдыгын негиздеди

Негизги сөздөр: миокард; зат алмашуунун коштолгон оорулары; ишемия; диастоликалык дисфункция; инсулин; чыгаруу фракциясы

Современные подходы к диагностике и лечению сердечной недостаточности у пациентов с сахарным диабетом 2 типа

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Аннотация. Целью работы являлось выявление системных ограничений в подходах к диагностике и лечению хронической сердечной недостаточности (ХСН) у пациентов с сахарным диабетом (СД) 2 типа на основе анализа патогенеза, методов верификации и эффективности терапии. В рамках теоретического подхода были реконструированы клинико-патофизиологические и организационные модели ХСН при СД второго типа с последующим сопоставлением диагностических алгоритмов, терапевтических стратегий и статистических данных из различных национальных систем здравоохранения, а также была выполнена экспертная оценка ведущих патогенетических механизмов. В ходе теоретического анализа было установлено, что формирование ХСН при СД 2 типа определялось совокупным влиянием диабетической кардиомиопатии, микрососудистой ишемии и хронического воспаления. Диагностические алгоритмы, основанные на оценке фракции выброса (ФВ), демонстрировали ограниченную чувствительность у пациентов с сохранённой сократительной функцией, доля которых достигала 60-70 %. Наиболее информативными методами ранней диагностики были признаны тканевая допплерэхокардиография и определение биомаркеров фиброза и перегрузки, включая натрийуретические пептиды, растворимый рецептор suppression of tumorigenicity-2 и галектин-3. Наибольшую доказательную эффективность в лечении ХСН у пациентов с СД 2 типа демонстрировало комбинированное применение четырёх классов препаратов – ингибиторов ангиотензинпревращающего фермента, β-адреноблокаторов, антагонистов минералокортикоидных рецепторов и ингибиторов натрий-глюкозного котранспортёра 2 типа (иНГЛТ-2). Были выявлены значительные различия в охвате базовой лекарственной терапией: до 78 % пациентов в США получали комбинированное лечение, тогда как в Казахстане – менее 45 %. Эти расхождения также проявлялись в уровне смертности: 66.2 случая на 100,000 населения в США против выраженного роста данного показателя в отдельных регионах Казахстана. Полученные результаты обосновывали необходимость адаптации клинических протоколов с учётом фенотипических особенностей сочетанной патологии, что позволяло повысить точность диагностики, индивидуализировать терапию и снизить сердечно-сосудистую смертность

Ключевые слова: миокард; метаболическая коморбидность; ишемия; диастолическая дисфункция; инсулин; фракция выброса