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ABOUT COVER

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REVIEW

Emerging risk factors for heart failure in younger populations: A growing public health concern

Razieh Parizad, Akash Batta, Juniali Hatwal, Mohammadreza Taban-sadeghi, Bishav Mohan

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Abstract

Heart failure (HF) is a growing public health concern, with an increasing incidence among younger populations. Traditionally, HF was considered a condition primarily affecting the elderly, but of late, emerging evidence hints at a rapidly rising HF incidence in youth in the past 2 decades. HF in youth has been linked to a complex interaction between emerging risk factors, such as metabolic syndrome, environmental exposures, genetic predispositions, and lifestyle behaviors. This review examines these evolving determinants, including substance abuse, autoimmune diseases, and the long-term cardiovascular effects of coronavirus disease 2019, which disproportionately affect younger individuals. Through a comprehensive analysis, the study highlights the importance of early detection, targeted prevention strategies, and multidisciplinary management approaches to address this alarming trend. Promoting awareness and integrating age-specific interventions could significantly reduce the burden of HF and improve long-term outcomes among younger populations.

Key Words: Heart failure; Metabolic syndrome; Environmental factors; Substance abuse; Psychosocial stressors; Youth; Genetics

Core Tip: Recent research hints at an alarming rise in heart failure cases among younger populations. The same is believed to stem from a complex interaction between various risk factors, including metabolic syndrome, environmental pollutants, unfavorable genetics, and lifestyle behaviors including substance abuse. This review examines these evolving determinants, discussing each in detail and exploring the key therapeutic strategies to disrupt this rapidly rising public health problem.

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INTRODUCTION

Heart failure (HF) is one of the most significant causes of morbidity and mortality worldwide, and it predominantly affects the older people. Recent studies have revealed an alarming rise in HF cases among younger populations[1]. The prevalence and causes of HF vary significantly between developed and developing countries. Approximately 1%-3% of all HF cases occur in individuals under 40 years of age. Notably, between 1999 and 2019, hospitalizations due to HF among young people aged 18-44 years increased by 23% in the United States[2].

In developed countries, the rising prevalence of HF among younger populations is associated with lifestyle factors, such as increasing rates of obesity, hypertension (HTN), and diabetes. Despite improvements in medical care and management of cardiovascular diseases (CVD), the burden of HF continues to rise, especially among younger adults. Hospital admissions for HF in youth have markedly increased in the last two decades, highlighting the urgent need for early detection and intervention[2,3].

HF is a growing public health concern in developing countries, with prevalence rates increasing due to demographic changes, urbanization, and the rising burden of non-communicable diseases (NCDs)[4,5]. In many developing countries, HF affects younger populations compared to developed countries, often due to untreated or poorly managed risk factors such as HTN, diabetes, and rheumatic heart disease (RHD)[6].

In Sub-Saharan Africa, the prevalence of HF is estimated to be between 1%-2%, with RHD and hypertensive heart disease being the primary causes. Patients in this region tend to be younger and often present with more advanced stages of the disease compared to those in developed countries[7]. Similarly, in South Asian countries such as India and Pakistan, HF is predominantly caused by ischemic heart disease, HTN, and diabetes. Patients in these regions frequently experience a higher prevalence of comorbid conditions and worse health outcomes compared to Western populations[8]. Infectious diseases, such as Chagas disease, human immunodeficiency virus (HIV), and tuberculosis contribute significantly to HF in developing countries. Chagas disease, caused by the parasite *Trypanosoma cruzi*, is endemic in Latin America and leads to chronic cardiomyopathy and HF[9]. HIV-associated cardiomyopathy is another important cause of HF in sub-Saharan Africa, where HIV prevalence is high[10].

Many developing countries face significant challenges in providing timely and effective healthcare for HF patients. Limited access to diagnostic tools, essential medications, and specialized care contributes to poor outcomes[11].

Despite the rapid identification of new associations and risk factors, the burden of HF among younger individuals continues to rise. This trend underscores the urgent need for further investigation in this area. The increasing prevalence of HF in younger adults is particularly concerning due to its severe complications and long-term consequences, including frequent hospitalizations, reduced quality of life, and elevated healthcare costs[12].

Most epidemiological studies on HF have focused on its increased prevalence in recent decades, often without distinguishing between older and younger people. Most of these studies have involved cohorts over 40 years of age, resulting in a limited understanding of HF in younger age groups.

The primary objective of this review is to explore the interplay of various risk factors, including emerging ones associated with the rising incidence of HF in younger generations focusing on identifying key contributors such as behavioral, environmental, genetic, and socio-economic influences. Additionally, it emphasizes the importance of age-specific screening, early diagnosis, and individualized treatment strategies tailored to the unique characteristics of HF in youth. Furthermore, it aims to raise awareness among healthcare professionals and the broader community about the growing incidence of HF in younger populations. Ultimately, this work seeks to inform strategies for reducing the burden of HF through targeted prevention, early intervention, and comprehensive management approaches.

METHODOLOGY FOR LITERATURE SELECTION

A systematic approach to literature selection was implemented to enhance the reproducibility and reliability of this review. The following steps were undertaken.

Databases searched and timeframe

The primary databases consulted were PubMed, Scopus, and Web of Science, covering studies published between 2000 and 2024.

Search terms

A combination of keywords and Medical Subject Headings (MeSH) terms was used, including "heart failure," "younger populations," "metabolic syndrome," "substance abuse," and "COVID-19 cardiovascular effects". Boolean operators (AND, OR) were utilized to refine the search strategy.

Inclusion criteria

Studies were included if they met the following criteria: (1) Focused on individuals under 40 years old; (2) Examined risk factors for HF; and (3) Provided original data or systematic reviews relevant to the topic.

Exclusion criteria

Non-English articles, conference abstracts, and studies lacking a clear methodology were systematically excluded.

The initial search yielded 632 articles. After screening for relevance and applying inclusion and exclusion criteria, 89 studies were included in this review. A PRISMA flow diagram summarizing the selection process is provided in Figure 1.

RISK FACTORS FOR HF IN YOUNG INDIVIDUALS

Metabolic syndrome and obesity

The increasing prevalence of metabolic syndrome (MetS) and obesity is a significant factor contributing to HF among younger individuals. Obesity leads to structural and functional alterations in the heart, including left ventricular hypertrophy (LVH) and diastolic dysfunction, both of which are early indicators of HF with preserved ejection fraction (HFpEF)[13].

MetS, influenced by excessive consumption of high-calorie foods, physical inactivity, HTN, hyperglycemia, dyslipidemia, and central obesity, is being diagnosed more frequently in adolescents and young adults, particularly in high-income countries[14]. This growing trend substantially raises the likelihood of HF in this demographic. Additionally, MetS is strongly associated with an elevated risk of type 2 diabetes (T2D), CVD, non-alcoholic fatty liver disease, chronic kidney disease, and various cancers[15]. These comorbidities not only contribute to HFpEF but also play a critical role in the development of coronary artery disease (CAD), a key precursor to HF with reduced ejection fraction (HFrEF).

Recent studies have highlighted a concerning rise in T2D prevalence among younger populations, which significantly contributes to the development of HF. This increase is attributed to factors such as childhood obesity, unhealthy dietary habits, and sedentary lifestyles[16-18]. For instance, data from the Global Burden of Disease Study indicate that the incidence of T2D in adolescents and young adults has risen by approximately 30% over the past decade, particularly in high-income countries where MetS is more[7]. Early-onset T2D is strongly linked to accelerated cardiovascular complications, including LVH and diastolic dysfunction, both of which are precursors to HF[19]. These findings emphasize the urgent need for targeted interventions aimed at reducing obesity and HTN, and addressing the rising burden of diabetes in younger age groups.

A sedentary lifestyle and unhealthy eating habits among young individuals are growing public health concerns. Physical inactivity and reduced functional capacity are strongly associated with adverse cardiovascular outcomes, independent of other risk factors[20].

Environmental factors

Environmental factors are often overlooked in clinical practice but, are increasingly recognized as an important risk factor for various CVDs. Air pollution and exposure to environmental toxins are emerging as critical contributors to HF, especially among young people, given their increased exposure. Oxides of nitrogen (NO_2) and particulate matter smaller (PM2.5) than 2.5 microns are particularly concerning as they can easily reach the lungs and bloodstream. Long-term exposure to these pollutants causes inflammation and oxidative stress, affects endothelial function, and ultimately is closely linked to HF pathophysiology[21,22]. Young individuals living in polluted environments are at a higher risk of developing early-onset HTN and MetS, both of which increase their likelihood of HF in later years. For instance, one study revealed that young age groups exposed to higher PM2.5 concentrations had elevated biomarkers associated with various CVDs, including C-reactive protein, one of the most clinically relevant biomarkers linked to CVD and HF[23,24].

Built environment: Urban planning and access to green spaces are increasingly recognized as critical components of cardiovascular health. Studies have shown that individuals living in areas with limited recreational spaces and high pollution levels are at a higher risk of developing HF[25]. The lack of walkable neighborhoods and exposure to urban heat islands further exacerbate cardiovascular risks by promoting sedentary lifestyles and increasing thermal stress[2]. Additionally, residential proximity to major roadways and industrial zones has been associated with elevated levels of air pollutants, contributing to systemic inflammation and endothelial dysfunction, key precursors to HF[3].

Socioeconomic factors: Socioeconomic disparities significantly influence the prevalence of HF in younger populations. Low socioeconomic status is associated with limited access to healthcare, poor nutrition, and higher exposure to environmental pollutants[4]. These factors contribute to the development of MetS, obesity, and other precursors of HF. Financial stress and job insecurity can lead to chronic psychosocial stress, which is an independent risk factor for HF[5]. Furthermore, individuals from lower socioeconomic backgrounds often face barriers to early diagnosis and treatment, resulting in delayed interventions and worse outcomes[6].

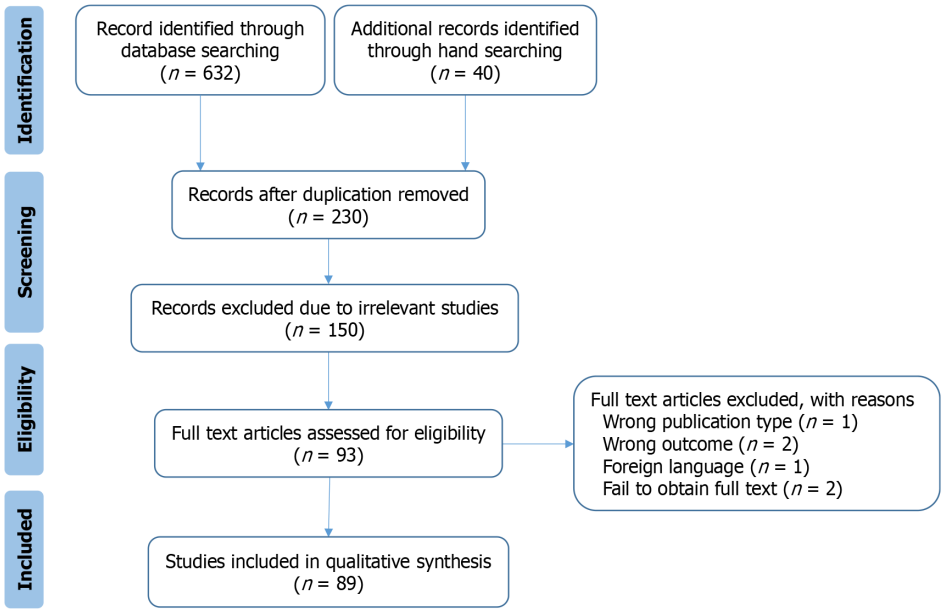

Figure 1 PRISMA flowchart of the literature selection process.

Climate changes: Climate change has emerged as a significant contributor to cardiovascular morbidity, particularly among younger individuals who are more exposed to extreme weather conditions. Heatwaves increase the risk of dehydration, electrolyte imbalances, and acute cardiovascular events, while cold spells elevate blood pressure and myocardial oxygen demand[7]. Climate-induced displacement and migration also often result in overcrowded living conditions, worsening exposure to air pollution and infectious diseases predisposing individuals to HF[8]. Rising global temperatures have also been linked to increased hospitalizations for HF, particularly in vulnerable populations such as those with pre-existing cardiovascular conditions[9].

HF is becoming increasingly prevalent among younger populations, with environmental factors playing a significant role in its development. Prolonged exposure to air pollutants, particularly fine PM2.5 and NO_2, has been linked to systemic inflammation, oxidative stress, and endothelial dysfunction, all of which contribute to CVD and HF. Moreover, exposure to heavy metals such as lead and mercury has been associated with structural changes in the myocardium, increasing the risk of early-onset HF. These environmental risk factors emphasize the need for preventive strategies and public health interventions to reduce exposure and mitigate their long-term impact (Figure 2).

Early-life risk factors and their role in HF development

Low birth weight (LBW) is increasingly recognized as a critical early-life risk factor for CVDs, including HF, particularly among younger populations. LBW is often associated with adverse perinatal circumstances such as maternal undernutrition, stress, and inadequate prenatal care, all of which can impair the growth and development of cardiovascular tissues. These factors contribute to structural and functional abnormalities in the heart, predisposing individuals to conditions like HTN and LVH, both of which are significant precursors to HF[26].

Furthermore, exposure to environmental pollutants during pregnancy has also been shown to exacerbate these effects, underscoring the multifactorial origins of LBW-related cardiovascular risks[27].

Recent studies have highlighted the significant impact of maternal nutritional deficiencies during pregnancy on fetal cardiac development, leading to long-term cardiovascular risks, including HF in adulthood. Maternal nutrition plays a crucial role in determining the risk of NCDs in offspring, such as heart disease, T2Ds, cancer, and chronic obstructive pulmonary diseases[28,29].

Research indicates that exposure to air pollution during pregnancy is associated with an increased risk of LBW, which in turn elevates the risk of CVD in adulthood. A study published in *Environmental Health Perspectives* found that exposure to air pollution, even at low levels, may increase the risk of LBW, particularly for certain segments of the population[30].

Childhood obesity is strongly associated with early markers of cardiovascular risk, including pre-HTN, pre-insulin resistance, and dyslipidemia, which contribute to adverse cardiac remodeling such as LVH and diastolic dysfunction[16].

Congenital heart diseases (CHDs) represent another major contributor to HF risk among young individuals.

Technological advancements in neonatal cardiac surgery have significantly improved survival rates for children born with CHD, enabling many to reach adulthood[26]. However, these individuals remain at an elevated risk of developing HF later in life due to a combination of factors, including residual anatomical defects, chronic morphological changes, and long-term complications associated with their condition[31]. For instance, even after successful surgical repair, patients often experience distorted cardiac geometry, conduction abnormalities, and prolonged QRS durations, all of which increase susceptibility to arrhythmias and HF[32,33]. Scarring of heart tissue following repeated surgical interventions can predispose individuals to both atrial and ventricular arrhythmias (VTs), further exacerbating their

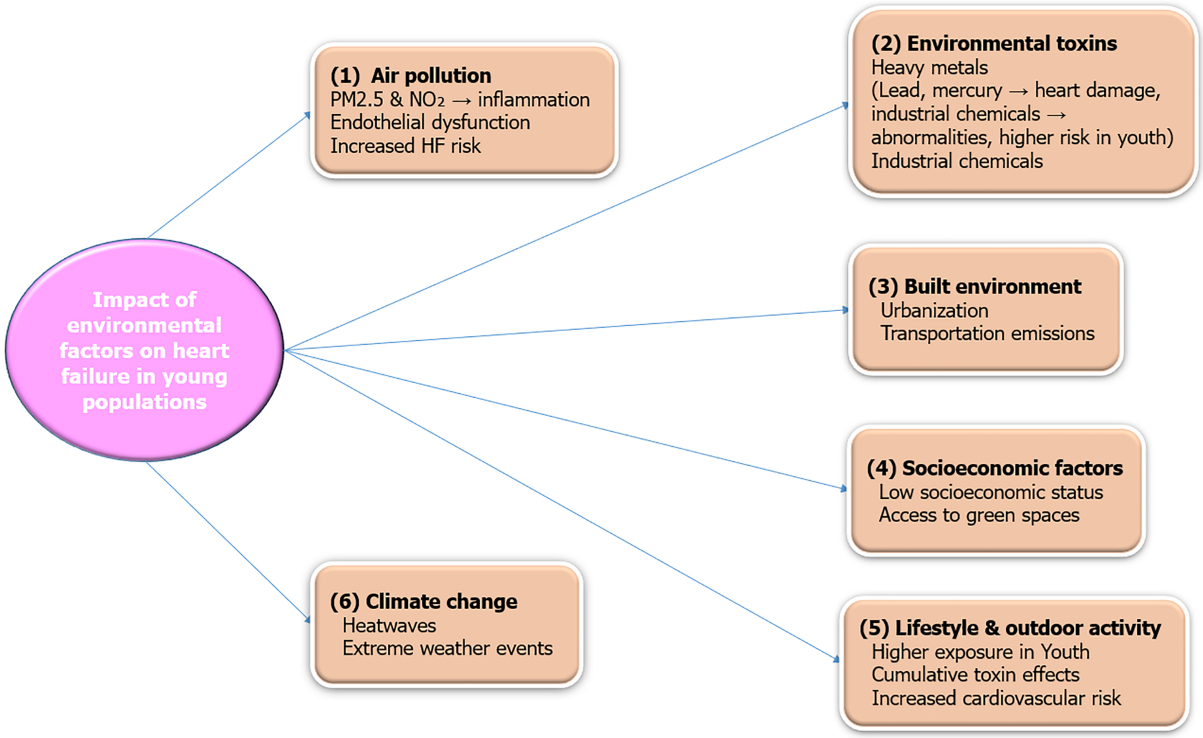

Figure 2 Impact of environmental factors on heart failure in young populations.

cardiovascular risk. Research indicates that myocardial scarring, resulting from surgical procedures, can lead to arrhythmogenic cardiomyopathy, characterized by the loss of ventricular myocardium and subsequent fibrous or fibro-fatty scar tissue replacement. This scarring predisposes individuals to potentially lethal VTs and impairs systolic ventricular function[34].

Moreover, specific types of CHDs, such as single ventricle physiology, present unique challenges. Despite advances in surgical techniques, these partially treatable defects often result in chronic hemodynamic stress, leading to progressive ventricular dysfunction and eventual HF[35]. Longitudinal studies have demonstrated that young adults with repaired tetralogy of fallot (TOF) or transposition of the great arteries (TGA) are particularly susceptible to adverse outcomes, including right ventricular (RV) failure and systemic complications. For instance, a longitudinal study by Kochav *et al*[36] evaluated changes in systemic right ventricular remodeling in adult patients with transposition of the great vessels using cardiovascular magnetic resonance imaging. The study highlighted that the systemic right ventricle in these patients is prone to structural and functional alterations, which may lead to significant long-term complications. Beyond the physiological burden, psychosocial factors also play a critical role. The fear of repeated medical interventions and the psychological impact of transitioning from pediatric to adult care can contribute to chronic stress, which has been linked to worsening cardiovascular health[37]. This underscores the need for comprehensive, multidisciplinary care that these patients' physical and emotional well-being[38].

Technological advancements in neonatal surgery have enabled more youngsters with CHD to survive into adulthood. However, this progress comes with increased risks of residual cardiac abnormalities and complications, such as arrhythmias, valvular dysfunction, and progressive ventricular failure[39]. According to Liu *et al*[26], patients with repaired CHDs, such as TOF or TGA, remain at elevated risk for developing HF due to chronic hemodynamic stress and myocardial scarring. Moreover, Fontan procedure survivors face unique challenges, including circulatory inefficiency and liver disease, further predisposing them to HF. The interplay between genetic predispositions and environmental factors, such as air pollution and socioeconomic disparities, also amplifies HF risks in this population[40].

Substance abuse and risk of HF

Substance abuse has been involved in causing HF in young adults in recent years. Various illicit substances and excessive use of legal substances have been demonstrated to have cardiotoxic effects and can contribute to HF. For instance, cocaine and amphetamines have been associated with cardiomyopathy and sudden cardiac death (SCD). Cannabis use has been associated with cardiovascular risks, including tachycardia, HTN, and myocardial infarction (MI). A systematic review by Arenas *et al*[41] found that cocaine use significantly increases the risk of HF and SCD.

The purposes for which cocaine is used may be associated with vasoconstriction of the coronary arteries, myocardial ischemia, and arrhythmias, while its direct cardiotoxicity can result in permanent heart damage. Additionally, the cardiovascular risks of cocaine may be dose-dependent and influenced by co-existing factors such as polysubstance use or pre-existing CVD. For example, a study by Gagnon *et al*[42] found that occasional cocaine users had a lower risk of HF compared to chronic users.

Additionally, the role of cocaine in causing HF may be overstated in some populations, as other substances (*e.g.*, alcohol, opioids) often co-occur and contribute to cardiovascular damage[43].

While cocaine is a well-established risk factor for CVD, the extent of its contribution to HF may vary depending on usage patterns and co-existing risk factors.

A study by Patel *et al*[44] found that cannabis use was associated with a 2–3 times higher risk of MI in young adults. Synthetic cannabinoids, in particular, have been linked to severe cardiovascular complications, including HF[45]. Nevertheless, other studies suggest that the cardiovascular risks of cannabis may be overstated. A systematic review by Sebastian *et al*[46] found limited evidence linking cannabis use to HF, with most studies being observational and prone to confounding. Some researchers argue that the cardiovascular effects of cannabis may depend on the method of consumption (*e.g.*, smoking *vs* edibles) and the presence of co-existing risk factors[47,48].

In contrast, the chronic use of substances containing amphetamine is associated with sympathetic stimulation, leading to hypertensive cardiomyopathy, arrhythmias, and hypertrophy of the heart, all of which render the patient a candidate for developing HF[49].

Except for stimulants, opioids have gained significant attention in the last ten years due to their association with the opioid epidemic and have also been related to cardiovascular complications. Chronic opioid use results in bradycardia, hypotension, and myocardial injury. In addition, opioid-induced hypoxia may trigger ischemia, thereby predisposing individuals to myocardial injury and, over time, eventually leading to HF[50]. Additionally, long-term alcohol consumption and smoking are well-established risk factors for dilated cardiomyopathy (DCM)[51]. The rising use of alcohol and drugs among young people further exacerbates this risk[52].

Other abused drugs, such as amphetamines and synthetic cannabinoids, also carry a risk of severe cardiac complications[53]. Methamphetamine exposure induces oxidative stress, mitochondrial damage, and endothelial dysfunction, all of which predispose individuals to the development of DCM. Synthetic cannabinoids have less literature available, but associations have been found with tachyarrhythmia, MI, and even HF in selected cases[54,55]. While alcohol and tobacco are legal, these represent essential yet already-established risk factors in the causation of DCM[56]. Chronic alcohol consumption leads to a long-term decline in myocardial contractility, eventually resulting in a form of HF known as alcoholic cardiomyopathy. Chronic smoking accelerates atherosclerosis and HTN and, in combination with increased oxidative stress, contributes to the risk of HF in smokers[57].

The adverse effects of substance abuse are exacerbated by the increasing incidence of polysubstance use, particularly the combination of drugs with psychoactive properties. For instance, the concurrent use of alcohol and cocaine significantly impacts the cardiovascular system, while opioid and benzodiazepine co-ingestion frequently occurs in overdose cases.

Substance abuse has emerged as a significant contributor to HF among young populations, with distinct patterns of use and cardiotoxic effects observed in this demographic. Cocaine, for instance, is particularly prevalent among young adults and has been strongly associated with cardiomyopathy, arrhythmias, and SCD. Recent evidence highlights that chronic cocaine use in younger individuals leads to oxidative stress, endothelial dysfunction, and myocardial remodeling, all of which predispose them to HF at an earlier age compared to non-users[42]. Notably, the dose-dependent nature of cocaine's cardiotoxicity is exacerbated in young populations due to higher rates of binge use and polysubstance combinations, such as concurrent alcohol consumption, which further amplifies cardiovascular risks[43].

In addition to cocaine, synthetic cannabinoids have gained attention as a growing threat to cardiovascular health in young adults. These substances are linked to severe complications, including tachyarrhythmias, MI, and acute HF, even in otherwise healthy individuals[45]. A study demonstrated that young users of synthetic cannabinoids exhibited elevated biomarkers of cardiac injury, such as troponin levels, underscoring their potential to cause irreversible myocardial damage[44]. Furthermore, the psychosocial stressors often accompanying substance abuse, such as academic pressure or socioeconomic instability, disproportionately affect younger populations and exacerbate the progression to HF. Addressing these risks through targeted public health interventions and educational programs is critical to mitigating the rising burden of substance-induced HF in this vulnerable group[58]. This combination suppresses respiratory and cardiovascular function, potentially resulting in SCD or HF[59].

The impact of various substances of abuse on cardiovascular health is illustrated in Table 1.

The rising prevalence of substance abuse among young individuals highlights the urgent need for targeted public health interventions and educational programs to mitigate these risks.

AUTOIMMUNE DISEASES AND THEIR IMPACT ON HF RISK IN YOUNG WOMEN

Autoimmune diseases, such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA), disproportionately affect young women and are significant contributors to HF risk. These conditions can lead to myocarditis and pericarditis, inflammatory diseases of the heart that heighten the risk of HF[60,61]. Additionally, autoimmune disorders are strongly linked to CAD and acute coronary syndrome (ACS), which are associated with a higher risk of complications and worse prognosis compared to the general population experiencing the same episode. Individuals with autoimmune diseases are more likely to have a reduced ejection fraction and a greater propensity to develop HF following an ACS[62]. Furthermore, the use of corticosteroids for treating autoimmune diseases has deleterious cardiovascular effects, further increasing this risk[61].

The primary treatments for autoimmune diseases include immunotherapy medications such as corticosteroids, tumor necrosis factor-alpha (TNF-α) inhibitors, and Janus kinase (JAK) inhibitors, all of which have serious cardiovascular side effects. The adverse cardiovascular effects of corticosteroids include alterations in lipid profiles, dyslipidemia, HTN, and

Table 1 An overview of different substances of abuse and their effects on the cardiovascular system, including their associations with heart failure

Substance	Mechanism of action	Cardiovascular effects	Association with HF	Ref.
Cocaine	Blocks dopamine, norepinephrine, and serotonin reuptake	Vasoconstriction, tachycardia, arrhythmias, and hypotension	Associated with cardiomyopathy, MI, arrhythmias, and increased HF risk	[30, 46]
Amphetamines	Stimulates release of norepinephrine and dopamine	Tachycardia, hypertension, cardiomyopathy	Linked to hypertensive heart disease and increased HF risk	[47, 48]
Alcohol	CNS depressant altering cardiac function	Chronic use leads to alcoholic cardiomyopathy, hypertension, arrhythmias	High association with dilated cardiomyopathy leading to HF	[30, 49]
Opioids	Binds to opioid receptors, decreases pain, induces euphoria	Bradycardia, hypotension, respiratory depression	Chronic use may lead to myocardial injury and increase HF risk	[48, 50]
Nicotine	Increases heart rate and blood pressure	Endothelial dysfunction, tachyarrhythmias, hypertension	Increases HF risk through oxidative stress and inflammation	[49, 51]
Marijuana	Activates cannabinoid receptors	Tachycardia, hypotension, changes in vascular tone	Limited evidence; potential role in MI and arrhythmias	[50, 52]
Methamphetamine	Elevates dopamine release	Cardiovascular strain, hypertension	High risk of cardiomyopathy and HF due to structural remodeling	[47, 52]
Synthetic cannabinoids	Potent synthetic THC, stronger than natural cannabinoids	Tachyarrhythmias, MI, blood pressure changes	Severe cardiovascular complications including HF	[50, 52]
Ecstasy (MDMA)	Increases serotonin, norepinephrine, and dopamine levels	Hypertension, tachycardia, hyperthermia	Acute HF risks during dehydration and hyperthermia episodes	[51, 52]
Benzodiazepines	CNS inhibition causing sedation	Hypotension, respiratory depression	Limited association with HF; can exacerbate existing conditions	[48, 51]

CNS: Central nervous system; DCM: Dilated cardiomyopathy; HF: Heart failure; TCH: Tetrahydrocannabinol; MI: Myocardial infraction.

hyperglycemia, all of which are recognized risk factors for CVD[63]. Prolonged corticosteroid therapy leads to arterial wall stiffening and atherosclerosis, consequently increasing the risk of MI and stroke[63].

Anti-TNF-α therapies, commonly used to manage autoimmune diseases such as RA and psoriasis, have been shown to influence patients' lipid profiles. A meta-analysis study (2023) indicated that these treatments might temporarily elevate high-density lipoprotein levels in psoriasis patients, with significant increases observed within the first three months of therapy. However, changes in triglyceride (TG) levels varied over time, showing significant increases between three to six months of treatment. These findings underscore the importance of regular lipid monitoring in patients undergoing anti-TNF-α therapy[64]. Another study highlighted that while anti-TNF-α treatments were associated with significant increases in total cholesterol and TG levels, the addition of statins helped reduce low-density lipoprotein levels. This suggests that combining statin therapy with anti-TNF-α treatments may mitigate potential atherogenic risks associated with lipid profile alterations[65]. While anti-TNF-α therapies effectively reduce inflammation in autoimmune conditions, their impact on lipid profiles necessitates careful management to address potential cardiovascular risks.

However, some studies suggest that these drugs may confer endothelial protection[66]. JAK inhibitors, the most recent class of immunomodulatory drugs, specifically target inflammatory pathways. Nevertheless, they have been associated with an increased risk of adverse cardiovascular events, including thromboembolic complications such as deep vein thrombosis and pulmonary embolism. Additionally, specific JAK inhibitors may exacerbate conventional CVD risk factors, such as HTN and dyslipidemia[67,68].

The cardiovascular side effects of these immunotherapies underscore the importance of cardiovascular screening to effectively manage CVD risk factors in patients undergoing long-term immunosuppressive treatment[69,70].

The complex interplay between autoimmune diseases, immunotherapy treatments, and cardiovascular complications is shown in Figure 3, which highlights the increased risk of HF in young women due to conditions such as myocarditis, pericarditis, and CAD, as well as the adverse effects of therapies like corticosteroids, anti-TNF-α inhibitors, and JAK inhibitors.

Genetic factors in HF prevalence among young individuals

Genetics plays a crucial role in the prevalence of HF among younger individuals. Familial cardiomyopathies, such as hypertrophic cardiomyopathy (HCM) and arrhythmogenic RV dysplasia (ARVD), significantly contribute to the early onset of HF in genetically predisposed individuals[71]. Advances in genetic testing have facilitated the identification of at-risk individuals; however, its clinical application in younger populations remains limited[26].

Historically, genetics was regarded primarily as a predisposing factor for HF in young groups age. Conditions such as HCM and ARVD exemplify familial cardiomyopathies that lead to early-onset HF in patients with specific gene mutations, reinforcing the role of genetic factors[72]. Recent epidemiological evidence suggests that the incidence of genetic cardiomyopathies, particularly HCM, is higher than previously estimated, affecting approximately 0.2% of the

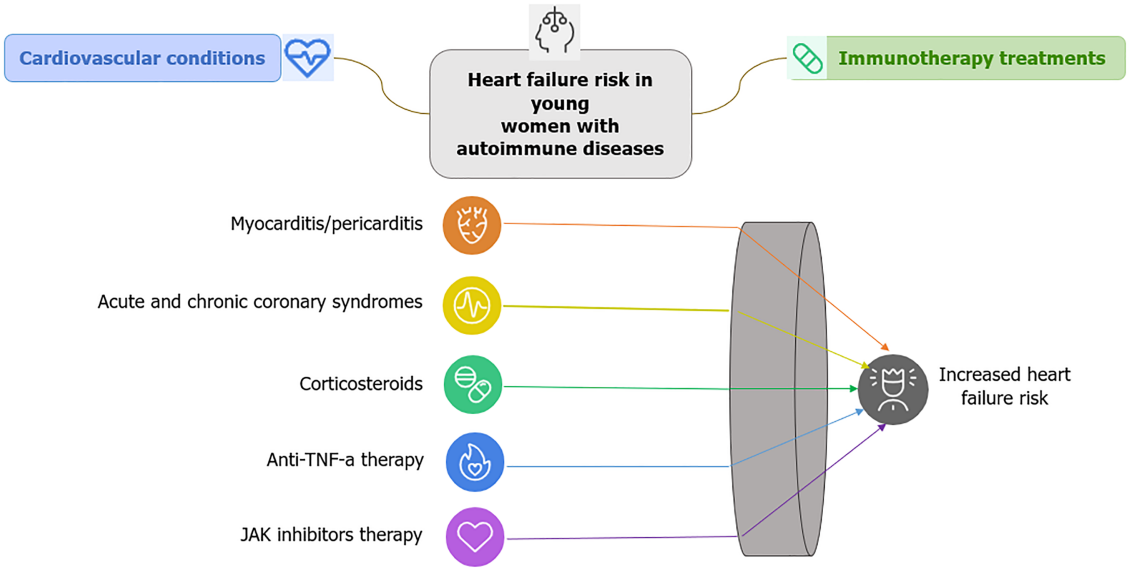

Figure 3 Autoimmune diseases and their impact on heart failure risk in young women.

global population. These conditions tend to present with more severe symptoms in younger individuals due to structural and functional cardiac abnormalities, increasing the risk of SCD and early HF onset compared to other HF types[73].

Recent studies have shown that mutations in the *MY*, *MYBPC3*, *PKP2*, *DSP*, *TTN*, and *LMNA* play a significant role in causing structural and functional myocardial abnormalities, which increase the risk of early-onset HF and SCD. HCM is one of the most common genetic causes of HF, especially in younger individuals. It is primarily caused by gene mutations that encode sarcomeric proteins, such as *MYH7* and *MYBPC3*[74,75].

These mutations lead to abnormal thickening of the left ventricular (LV) wall, diastolic dysfunction, and an increased risk of arrhythmias and SCD[76].

Mutations in the *MYBPC3* gene are also common, accounting for 20%-30% of HCM cases. These mutations typically result in a later onset of symptoms but still carry a significant risk of HF and arrhythmias[73].

Genetic testing for *MYH7* and *MYBPC3* mutations is recommended for individuals with a family history of HCM or unexplained cardiac hypertrophy. Early identification of these mutations allows targeted interventions, such as implantable cardioverter-defibrillators (ICDs), to prevent SCD[77].

ARVD is a genetic cardiomyopathy characterized by fibrofatty replacement of the RV myocardium, leading to arrhythmias and HF. It is commonly caused by mutations in desmosomal genes, such as *PKP2* and *DSP*, which are crucial for maintaining the structural integrity of cardiac tissue. These mutations disrupt the normal functioning of desmosomes, resulting in structural instability and increased susceptibility to arrhythmias and progressive HF[78].

PKP2 mutations are the most common cause of ARVD, accounting for approximately 40%-50% of cases. These mutations disrupt cell-to-cell adhesion, resulting in myocardial damage and arrhythmias[79].

DSP mutations, while less common, are associated with a more severe phenotype, including biventricular involvement and a higher risk of HF[80].

Genetic testing for *PKP2* and *DSP* mutations is recommended for individuals with a family history of ARVD or unexplained arrhythmias. Early diagnosis allows for the initiation of antiarrhythmic therapy and ICD implantation to prevent SCD[81].

DCM is characterized by LV dilation and systolic dysfunction, often leading to HF. It can be caused by mutations in genes encoding cytoskeletal, sarcomeric, and nuclear envelope proteins, such as *TTN* and *LMNA*. Mutations in the *TTN* gene, most notably truncating variants, are the most common genetic cause of DCM, accounting for approximately 20%-25% of cases. These mutations disrupt the structural integrity of the sarcomere, leading to progressive ventricular dilation and HF[82].

Mutations in the *LMNA* gene are associated with a more severe phenotype, including early-onset HF, conduction system disease, and a high risk of SCD[83].

Genetic testing for *TTN* and *LMNA* mutations is recommended for individuals with a family history of DCM or unexplained HF. Early identification of these mutations allows for the initiation of guidelines, directed medical therapy and consideration of ICD implantation[84].

Other genetic cardiomyopathies, such as LV non-compaction (LVNC) and restrictive cardiomyopathy (RCM), are also associated with an increased risk of HF. These conditions are often caused by mutations in genes such as *MYH7*, *TNNT2*, and *TNNI3*[84].

TNNT2 and *TNNI3* mutations: Mutations in *TNNT2* and *TNNI3* are associated with RCM and LVNC, leading to diastolic dysfunction and HF. Although rare, these mutations carry a high risk of adverse outcomes[85].

Genetic testing for *TNNT2* and *TNNI3* mutations should be considered in individuals with unexplained HF or a family history of cardiomyopathy. Early diagnosis allows for tailored management strategies, including heart transplantation in severe cases[86].

Advancements in genetic testing methods have significantly improved over time. Next-generation sequencing, for instance, has facilitated the identification of pathogenic mutations underlying HCM, arrhythmogenic ARVD, and other inherited cardiomyopathies, thereby enabling earlier interventions[87].

However, the use of genetic testing in these patient populations remains limited due to factors such as cost, accessibility, and challenges with insurance reimbursement. Furthermore, interpreting genomic variants is complex, as many mutations exhibit low penetrance, meaning not all carriers will develop HF. These challenges hinder effective patient education and clinical management[88].

The increasing prevalence of genetic cardiomyopathies has been accompanied by advancements in diagnostic techniques, including cardiac magnetic resonance imaging (MRI) and genetic testing. These methods have enhanced understanding of disease progression, clinical heterogeneity, and genetic-morphological correlations[73,89]. Consequently, precision medicine is becoming more prevalent in cardiology, enabling treatment and prevention plans tailored to patients' genetic profiles. This approach holds significant potential for improving care for young HF patients, mainly those with familial DCM, by allowing for earlier and more precise intervention[89].

Long-term outcomes of CHDs and HF

Recent advancements in pediatric cardiology and surgical interventions have significantly improved survival rates for children born with CHD, enabling many affected individuals to reach adulthood[90]. However, these patients remain at lifelong risk of developing HF due to residual cardiac abnormalities and the long-term consequences of their initial corrective surgeries.

Specific forms of CHD are mainly associated with a high risk of HF. For instance, patients with TOF, even after surgical repair, may experience complications such as pulmonary regurgitation and subsequent RV dilatation, which can eventually lead to HF in adulthood[91,92]. Likewise, systemic RV failure is frequently observed in patients with TGA who have undergone Mustard or Senning procedures. In such cases, the RV is subjected unphysiologically designed systemic pressures[93].

Unrepaired lesions also pose significant risks for HF. Large ventricular septal defects (VSDs) and uncorrected single ventricle physiology are particularly notable. Patients with unoperated VSDs often develop pulmonary HTN and Eisenmenger syndrome, leading to severe HF due to increased pulmonary vascular resistance and RV strain. Even in cases where CHDs have been repaired, residual defects, arrhythmias, or ventricular dysfunction may predispose patients to HF[94,95]. Besides, patients with single ventricle physiology who have undergone the Fontan procedure face unique long-term complications, such as Fontan-associated liver disease and circulatory inefficiency, both of which often result in HF as they transition into adulthood[96].

The distinction between repaired and unrepaired CHD is crucial, as both pathways can lead to HF through different pathophysiological mechanisms. Repaired congenital defects may result in HF due to long-term surgical sequelae, including scarring, valvular insufficiency, and arrhythmias, all of which impose chronic stress on the heart. Conversely, unrepaired disabilities may cause HF through chronic volume overload, increased pulmonary pressures, and progressive RV failure. These risks emphasize the importance of lifelong cardiac monitoring and individualized management strategies tailored to each adult with CHD. Such approaches aim to optimize long-term outcomes[97].

Role of chronic stress and mental disorders in the development of HF

Psychosocial stressors, particularly depression, significantly contribute to CVD and HF pathogenesis. An 18-year longitudinal study demonstrated that depression is an independent risk factor for incident coronary heart disease in women, mediated by chronic inflammation and autonomic nervous system dysregulation[98,99].

In other words, the rising prevalence of mental illnesses among high school students has become a growing public health concern, contributing to the increasing burden of HF. Currently, approximately one in five adolescents worldwide experience an anxiety disorder, depression, or substance use disorder, and most of these individuals do not receive treatment[100]. For example, the rate of major depressive episodes among children and adolescents aged 12 to 17 years in the United States increased from 13.3% in 2017 to 15.7% in 2021, mirroring global trends[101]. Research indicates that the coronavirus disease 2019 (COVID-19) pandemic exacerbated this issue, with anxiety and depression among adolescents increasing by 25% during this period[102,103].

Several contributing factors, including low socioeconomic status, financial and academic pressures, and associated lifestyle choices such as smoking, alcohol and drug use, poor nutrition, and physical inactivity, are recognized risk factors for CVD. Depression and other mental health disorders are not only independent predictors of HF but also contribute to its development through pathways such as increased pro-inflammatory cytokines, elevated cortisol levels, and autonomic dysfunction[104,105]. Furthermore, adults with a history of childhood mental health disorders face significantly higher risks of premature CVD, including HF. These risks can persist for decades, with mental health disorders serving as long-term predictors of cardiovascular outcomes[106].

Given the interplay between mental health and HF, a multisectoral approach to prevention is essential. Integrating mental health assessments and care with the management of CVD risk factors among youth could help mitigate this emerging public health issue. This combined strategy offers a promising pathway to reducing HF complications and promoting better long-term health outcomes[107,108].

The hidden long-term cardiovascular burden of COVID-19: Insights into HF

Emerging evidence thus demonstrates that COVID-19 is associated with significant long-term cardiovascular risk, particularly in young and otherwise healthy populations. Myocarditis has been reported at a rate of 11 per 100000 young adults following COVID-19, a rate notably higher than that observed after other viral infections[109]. This condition, charac-

terized by persistent inflammation, can lead to ventricular dysfunction and increase the risk of HF[110].

Several studies have reported that COVID-19 can cause myocarditis, even in young and otherwise healthy individuals. The chronic inflammatory response triggered by the virus may predispose these individuals to HF in the future[111,112]. For example, a study by Puntmann *et al*[113] found that 78% of recovered COVID-19 patients exhibited cardiac involvement, including myocarditis, as detected by cardiac MRI.

A large population-based study by Tuvali *et al*[109] found that the incidence of myocarditis after COVID-19 was only 11 per 100000 individuals, which is lower than previously reported. Additionally, some researchers argue that the long-term cardiovascular risks of COVID-19 may be confounded by pre-existing conditions or the severity of the acute infection. For example, a study by Alvarez-Garcia *et al*[114] found that prior HF, rather than COVID-19 itself, was the strongest predictor of adverse cardiovascular outcomes post-infection.

The virus can also induce a cytokine storm, directly causing myocardial damage, fibrosis, and vascular inflammation factors that contribute to an elevated risk of cardiomyopathy and HF, even in individuals without prior CVD[115].

According to a study, post-recovery COVID-19 has been associated with a 45% increased risk of developing new-onset HF, particularly among those who required hospitalization during the acute phase of the infection[116]. Additionally, indicates that 10%-40% of COVID-19 survivors experience long COVID syndrome, often presenting with cardiovascular symptoms such as chest pain and fatigue, further contributing to the long-term cardiovascular burden in younger populations[117].

Despite this, other studies suggest that the risk of HF post-COVID-19 may be overstated. Trimaille *et al*[118] found that most young patients with COVID-19-related cardiac symptoms recovered fully without developing HF. Some researchers argue that the observed increase in HF cases may be due to heightened surveillance and diagnostic testing rather than a direct effect of the virus[110]. While COVID-19 can lead to HF in some patients, the overall risk may be lower than initially feared, especially in young and healthy individuals. More longitudinal studies are needed to assess the actual burden of COVID-19-related HF.

Given this evidence, it is clear that managing the long-term cardiovascular effects of COVID-19, will remain a critical public health priority. Future research should prioritize the development of cardiac monitoring and management strategies for COVID-19 survivors, emphasizing on early detection and intervention to prevent the potential progression to HF. Moreover, public health measures should shift toward systematic screening for cardiovascular sequelae, including among younger COVID-19 survivors, as they may face an increased risk of HF over an extended period following infection[119].

The long-term cardiovascular effects of COVID-19 on HF in young individuals have become a significant area of concern. Studies indicate that myocarditis, a condition characterized by persistent inflammation, occurs at a rate of 11 per 100000 young adults following COVID-19 infection, which is notably higher than the incidence observed after other viral infections[109].

A study conducted by Xie *et al*[120] revealed that survivors of COVID-19 had a significantly higher risk of developing HF compared to non-infected controls, with an increased risk of up to 45% among those hospitalized during the acute phase of the infection. Additionally, 10%-40% of survivors experience prolonged symptoms like chest pain and fatigue, increasing the cardiovascular burden[117].

Despite these findings, some studies suggest that the overall risk of HF post-COVID-19 may be lower than initially feared, especially in healthy young individuals. Trimaille *et al*[118] reported that most young patients with mild or moderate COVID-19-related cardiac symptoms fully recovered without progressing to HF. However, it remains crucial to monitor this population closely due to potential delayed complications.

The data illustrated the need for ongoing research and targeted interventions to address the long-term cardiovascular consequences of COVID-19 in younger individuals. Regular cardiac monitoring and early detection strategies are essential for mitigating the progression to HF in this demographic (Figure 4).

SYNTHESIS OF FINDINGS ACROSS RISK FACTORS

The interplay between MetS and substance abuse reveals a compounded risk for HF in younger populations. Studies have demonstrated that MetS, characterized by obesity, HTN, and insulin resistance, predisposes individuals to cardiovascular dysfunction, including LVH and diastolic dysfunction. When combined with substance abuse such as chronic alcohol consumption or cocaine use, the cardiovascular strain intensifies due to overlapping mechanisms, including oxidative stress, endothelial dysfunction, and systemic inflammation[101,121]. This synergistic effect suggests that youth with coexisting MetS and substance use disorders may have an accelerated trajectory toward HF.

Many studies on the cardiovascular sequelae of COVID-19 rely on retrospective designs, which may introduce selection bias. In addition, the long-term cardiovascular outcomes in younger populations remain underexplored, and further prospective cohort studies are needed[122,123]. A recent retrospective cohort study revealed that COVID-19 survivors face elevated long-term risks of cardiovascular, cerebrovascular, and thrombotic complications; however, the retrospective design may introduce selection bias (*e.g.*, overrepresentation of hospitalized), underscoring the need for prospective studies to assess these outcomes in younger populations[124].

While the cardiotoxic effects of these substances are well-established, many of the existing studies are based on small sample sizes or animal models, which may limit the generalizability of the findings to human populations[125].

Furthermore, few studies have explicitly examined how risk factors such as MetS and substance abuse interact[13,42,43,126]. This gap highlights the need for research that integrates these overlapping pathways, particularly in young individuals at high risk for HF.

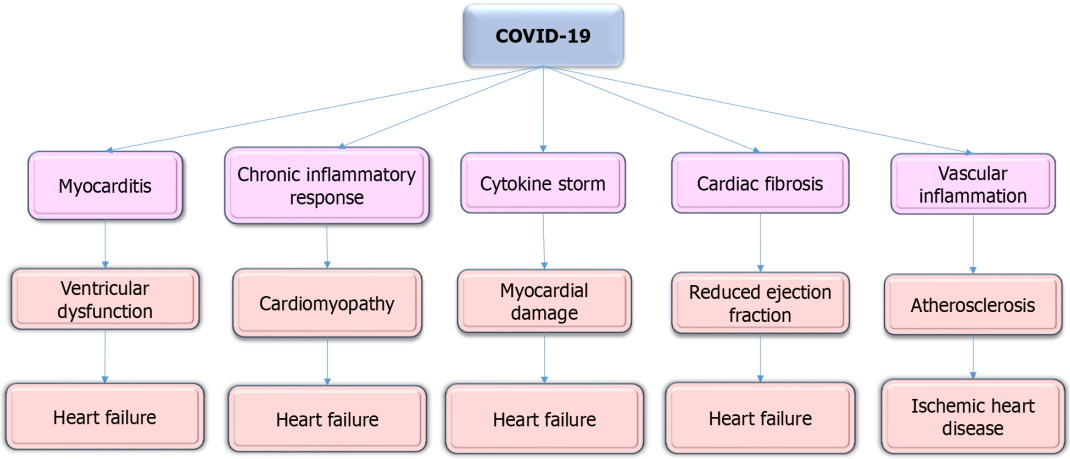

Figure 4 Long-term cardiovascular effects of coronavirus disease 2019 on heart failure in young individuals. COVID-19: Coronavirus disease 2019.

In summary, HF in younger populations is influenced by a complex interplay of various risk factors, including MetS, substance abuse, autoimmune diseases, genetic predispositions, environmental exposures, and the long-term cardiovascular effects of COVID-19. These factors often interact synergistically, exacerbating the risk of HF in this demographic. Figure 5 provides a comprehensive overview of these diverse risk factors, highlighting their cumulative impact on the rising incidence of HF among young individuals (Figure 5).

Gender differences in HF risk factors

Gender differences in HF risk factors have gained increasing attention in recent years, as emerging evidence suggests that men and women may experience distinct pathways and risk profiles leading to HF. While the section on autoimmune diseases highlights the disproportionate impact of conditions such as SLE and RA on young women, other risk factors also exhibit significant gender disparities that warrant further exploration.

MetS and obesity: MetS and obesity are major contributors to HF, but their impact varies by gender. Women with MetS are more likely to develop HF with HFpEF, whereas men are more prone to HF with HFrEF[126,127]. This difference is partly attributed to hormonal influences, as estrogen has protective effects on vascular health. However, these benefits diminish after menopause, increasing women's susceptibility to HFpEF[128]. Additionally, women tend to accumulate more visceral fat, which is strongly associated with insulin resistance and diastolic dysfunction, further predisposing them to HFpEF[129].

Substance abuse: Substance abuse, specifically alcohol and illicit drugs, affects men and women differently. Men are more likely to engage in heavy alcohol consumption and illicit drug use, which are strongly linked to DCM and HFrEF [130]. However, the gender gap in substance abuse is closing, and healthcare practitioners may not always investigate substance abuse history in detail, which can limit the recognition of its role in the pathogenesis of cardiomyopathy and HF. In contrast, women are more susceptible to the cardiotoxic effects of substances Such as cocaine, even at lower doses, due to differences in body composition and metabolism[131]. Furthermore, women with substance use disorders often face additional psychosocial stressors, such as domestic violence and caregiving responsibilities, which can exacerbate HF risk[132].

Psychosocial stressors and mental health: Psychosocial stressors, including chronic stress, depression, and anxiety, disproportionately affect women and are independent risk factors for HF. Women are twice as likely as men to experience depression, which is associated with worse HF outcomes due to poor adherence to treatment and unhealthy lifestyle behaviors[133]. Chronic stress, often linked to caregiving roles, can lead to endothelial dysfunction and inflammation, further increasing HF risk in women[8].

Environmental factors: Exposure to environmental pollutants, such as PM2.5 and NO_2, affects men and women differently. Women may be more vulnerable to the cardiovascular effects of air pollution due to hormonal fluctuations and differences in lung physiology[37]. For example, long-term exposure to PM2.5 has been related to a higher incidence of HFpEF in women than men[134].

Genetic predisposition: Genetic cardiomyopathies, such as HCM and arrhythmogenic ARVD, also exhibit gender differences. Men with HCM are more likely to experience SCD and severe hypertrophy, while women often present with more subtle symptoms but a higher risk of HF progression[135,136]. Furthermore, mutations in genes such as *TTN* and *LMNA* may have varying penetrance and clinical manifestations based on gender, necessitating gender-specific management strategies[137].

COVID-19 and cardiovascular risks: The long-term cardiovascular effects of COVID-19 also show gender disparities.

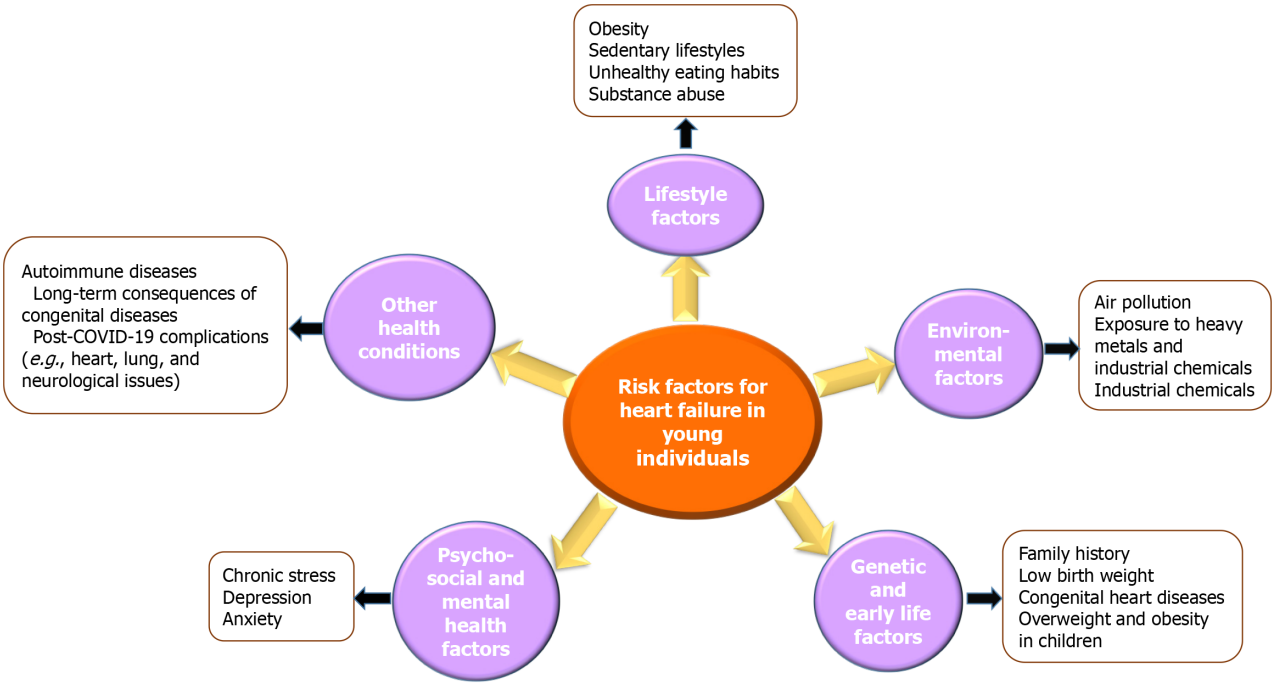

Figure 5 Various risk factors for heart failure in young individuals. COVID-19: Coronavirus disease 2019.

Women are more likely to experience post-COVID-19 myocarditis and persistent cardiovascular symptoms, such as chest pain and fatigue, which can contribute to HF[138,139]. These differences may be due to variations in immune responses and hormonal influences, which modulate the inflammatory cascade triggered by the virus[140].

Gender differences in HF risk factors are significant and multifaceted, encompassing metabolic, behavioral, psychosocial, environmental, and genetic dimensions[141]. Addressing these disparities requires a gender-specific approach to prevention, diagnosis, and treatment. Future research should focus on elucidating the underlying mechanisms of these differences and developing targeted interventions to improve outcomes for both men and women at risk of HF.

Gender differences play a crucial role in the prevalence and progression of HF, with distinct risk factors influencing disease outcomes in men and women. Understanding these gender-specific variations is essential for developing targeted prevention and treatment strategies to improve cardiovascular health outcomes.

PREVENTING HF AND BREAKING THE RISK FACTOR CHAIN IN THE YOUNG INDIVIDUALS

The rising prevalence of HF in younger populations is a significant public health concern driven by a complex interplay of emerging risk factors. These include MetS, environmental exposures, genetic predispositions, lifestyle behaviors, substance abuse, autoimmune diseases, and the long-term cardiovascular effects of COVID-19. Addressing these challenges requires a multidisciplinary approach incorporating lifestyle interventions, public health policies, and early screening programs.

Studies have emphasized the role of lifestyle modifications, including physical activity and dietary changes, in reducing the incidence of MetS and obesity, which are significant contributors to HF[142]. Moreover, environmental factors such as air pollution and exposure to heavy metals have been linked to adverse cardiovascular outcomes, emphasizing the need for stricter environmental regulations and public health initiatives[143] Furthermore, the COVID-19 pandemic has introduced new challenges, with emerging evidence suggesting that severe acute respiratory syndrome coronavirus 2 infection may exacerbate cardiovascular risks, notably in young individuals with pre-existing conditions [144]. Effectively mitigating these multifaceted risk factors through targeted interventions, public health initiatives, and interdisciplinary collaboration is essential preventing of HF and improving cardiovascular outcomes in young populations.

ACTIONABLE RECOMMENDATIONS

MetS and obesity

Social mobilization plays an essential role in encouraging heart care from childhood onward by reducing exposure to conditions like MetS, which can lead to LVH and CAD.

Implementing school-based and community programs to encourage regular physical activity is essential. Programs such as the Coordinated Approach to Child Health (CATCH) have proven effective in increasing physical activity levels and improving dietary habits among children and adolescents[145]. Encouraging diets rich in fruits, vegetables, and whole grains while reducing the consumption of processed foods, salt, and unhealthy fats is crucial. For instance, the Dietary Approaches to Stop HTN (DASH) diet, has been demonstrated to lower blood pressure and enhance cardiovascular health[140]. Also, introducing taxes on sugary drinks and providing subsidies for healthier food options can make nutritious choices more accessible. For example, Mexico's tax on sugar-sweetened beverages significantly declined their consumption[146]. These measures can play a vital role in improving public health outcomes.

Environmental factors

Environmental modifications are equally important, as poor air quality and exposure to heavy metal toxicity are known to increase the risk of CVD in young populations[147]. Strengthening air quality standards and enforcing regulations to reduce emissions of PM2.5 and NO_2. are essential steps toward improving public health. For example, the Clean Air Act in the United States, has illustrated significant success in reducing air pollution and its associated health risks[148,149].

Promoting the development of green spaces in urban areas can help minimize exposure to harmful air pollutants. Research has consistently shown that urban green spaces are associated with better cardiovascular health outcomes[150, 151]. Likewise, public awareness campaigns play a vital role in educating communities about the dangers of air pollution and encouraging the adoption of protective measures, such as using air purifiers in homes and schools, particularly in regions with high pollution levels. Effective communication strategies, including air quality advisories and targeted awareness campaigns, can help improve environmental health literacy and promote public understanding of the health impacts of air pollution. Participatory sensing activities and citizen science approaches can also enhance public engagement and interest in addressing air pollution issues. While public awareness campaigns can help protect against the decline of public concern and motivate individuals to take protective actions, they may not directly influence public protective behaviors in the absence of other supporting measures[151,152].

Substance abuse

School-based substance abuse prevention programs, such as Project Adolescent Lifestyle Education and Risk Training and LifeSkills Training, have indicated effectiveness in reducing substance abuse among adolescents by providing valuable tools and education to help young people make healthier choices[153].

Increasing access to evidence-based treatment programs for substance abuse, including medication-assisted treatment (MAT) for opioid addiction, is essential. MAT integrates medications with counseling and behavioral therapies, offering a comprehensive approach to effectively manage and treat opioid use disorders[154].

Public health campaigns play a significant role in raising awareness about the cardiovascular risks associated with substance abuse. For instance, programs aimed at reducing smoking have successfully decreased smoking rates among young adults through targeted media campaigns that emphasize the dangers of tobacco use[155,156].

Autoimmune diseases

Early screening programs for autoimmune diseases, such as SLE and RA, are essential to prevent cardiovascular complications. The Lupus Foundation of America highlights the importance of routine cardiovascular risk assessments in patients with SLE to identify and manage potential issues at an early stage[157,158].

Cardiovascular risk assessment tools, such as the QRISK3 calculator, can help identify and manage cardiovascular risks more effectively in patients with autoimmune diseases. These tools provide valuable insights into an individual's risk profile, enabling healthcare providers to tailor interventions accordingly[159].

Raising patient awareness regarding the cardiovascular risks tied to autoimmune diseases is crucial. Educating them on how adopting healthier habits, like stopping smoking and maintaining a healthy weight, is essential to lower their risk of cardiovascular issues. Such education empowers patients to take proactive steps toward improving their long-term health outcomes[160].

Genetic factors

Early diagnosis and treatment are indispensable for preventing HF. Regular check-ups, including annual physical exams and cardiovascular screenings, should be emphasized, even for young individuals without evident CVD risk factors[147, 161,162]. Identifying risk factors early allows for timely intervention, potentially preventing the progression to HF. Individualized care plans that consider genetic predispositions and social determinants of health can significantly enhance patient outcomes[163]. Promoting genetic screening for familial cardiomyopathies, such as HCM and ARVD, is critical for individuals with a family history of HF. For ARVD, Muller *et al*[164] emphasize that cascade genetic testing identifies 70% of at-risk relatives before symptom onset, enabling interventions like implantable cardioverter-defibrillator (ICD) placement to prevent sudden cardiac death. Their study highlights that early genetic screening reduces diagnostic delays by an average of 4 years compared to symptom-based diagnosis.

For HCM, Dellefave-Castillo *et al*[165] demonstrate that multi-gene panel testing identifies pathogenic variants in 15%-30% of cases, with mutations in MYBPC3 and MYH7 accounting for the majority of familial cases. This aligns with American Heart Association (AHA) guidelines, which recommend genetic testing for first-degree relatives to enable early risk stratification and management. Importantly, Dellefave-Castillo *et al*[165] also show that combined testing for cardiomyopathy and arrhythmia genes increases diagnostic yield by 40% compared to single-gene approaches, ensuring comprehensive identification of at-risk individuals across both HCM and ARVD. Integrating these strategies reduces morbidity and mortality in high-risk populations.

The application of precision medicine can significantly improve treatment strategies by customizing therapies based on individual genetic variations. Programs such as the National Institutes of Health's (NIH) All of Us Research Programactively gathers comprehensive genetic data to advance personalized medicine and enhance patient outcomes [166,167].

Increasing public awareness about the importance of genetic testing and its role in preventing HF is equally vital. Educating communities about the benefits of genetic screening can encourage more individuals to seek testing, leading to earlier detection and better management of cardiovascular conditions[168-170].

Chronic stress and mental health

Integrating mental health services into primary care settings is essential for addressing chronic stress, depression, and anxiety, significant contributors to cardiovascular risks. The Collaborative Care Model has proven effective in improving mental health outcomes and, in turn, reducing the likelihood of cardiovascular complications[171].

Incorporating mental health initiatives within school settings, such as *Mind Matters and FRIENDS*, is essential for mitigating anxiety and depression among adolescents. By equipping young individuals with the necessary coping strategies and support systems, these programs contribute to their long-term psychological well-being[172].

Encouraging the adoption of workplace wellness programs that integrate stress management and mental health support is a crucial initiative. The World Health Organization's *Mental Health Gap Action Programme* is vital in addressing mental health challenges globally[173].

COVID-19 and cardiovascular risks

Young individuals recovering from COVID-19, including those with ongoing symptoms, should undergo regular cardiac monitoring to enable early detection and effective management of potential cardiovascular complications. According to the American College of Cardiology, cardiac MRI is suggested for patients suspected of myocarditis following a COVID-19 infection, as this method supports accurate diagnosis and timely intervention[113].

Launching public health campaigns to raise awareness about the long-term cardiovascular risks involved in COVID-19 is essential. These campaigns should emphasize the importance of early intervention and regular follow-ups to mitigate potential health issues and improve outcomes for affected individuals.

Investing in research to better understand the long-term cardiovascular effects of COVID-19 is equally important. Initiatives like the NIH *RECOVER Initiative* are actively examining the prolonged impacts of COVID-19, emphasizing on cardiovascular complications, to develop targeted interventions and enhance patient care[174].

Effective prevention also depends on interprofessional collaboration among cardiologists, primary care physicians, and other specialists focused on young adults[175]. Additionally, ongoing research the socioeconomic and environmental predictors of HF and implementing new policies addressing these factors, is essential for long-term prevention.

Raising awareness and promoting education are critical components in mitigating the risk of HF among younger individuals. Public health campaigns targeting schools, workplaces, and communities can play a pivotal role in educating young people about the importance of maintaining a healthy lifestyle, recognizing early warning signs of HF, and understanding the impact of modifiable risk factors such as obesity, substance abuse, and poor dietary habits[108]. For instance, school-based programs like the CATCH have adolescents' physical activity levels and nutritional choices[145]. Furthermore, integrating mental health education into existing frameworks can help address psychosocial stressors, which are increasingly recognized as significant contributors to CVD[133]. Additionally, educational initiatives to inform the public about the long-term cardiovascular effects of environmental pollutants and substance use disorders can empower individuals to make healthier decisions[143]. By fostering a culture of preventive care through targeted education and awareness campaigns, it is possible to reduce the burden of HF in younger populations.

To effectively mitigate the rising burden of HF in younger populations, it is essential to adopt a comprehensive and multidisciplinary approach targeting the complex network of risk factors. By advancing early intervention strategies and encouraging collaboration across healthcare disciplines, significant progress can be made in reducing HF prevalence and enhancing cardiovascular health outcomes[108]. Addressing these challenges requires integrating lifestyle modifications, public health policies, and early screening programs into routine care. Healthcare providers must remain vigilant in identifying cardiovascular risks, particularly among young patients with family histories of heart disease or additional risk factors such as obesity, substance abuse, or autoimmune disorders (Figure 6)[109].

Public health initiatives promoting physical activity, healthy diets, and substance abuse prevention are crucial for reducing the long-term cardiovascular burden, including complications associated with conditions like COVID-19[110]. Programs to educate young populations about the dangers of unhealthy behaviors and environmental exposures can play a pivotal role in fostering healthier lifestyles[119]. Furthermore, implementing school-based interventions, community awareness campaigns, and evidence-based treatments for substance use disorders will strengthen preventive efforts[153]. These coordinated actionscan break the chain of risk factors contributing to HF and improve long-term health outcomes in younger generations.

TREATMENT STRATEGIES FOR HF IN YOUNGER POPULATIONS

While prevention is imperative, effective treatment strategies are equally crucial for managing HF in younger populations. Given the unique risk factors and etiologies of HF in this demographic, treatment approaches must be tailored to meet the specific needs of younger patients. Recent evidence and guidelines suggested key treatment strategies.

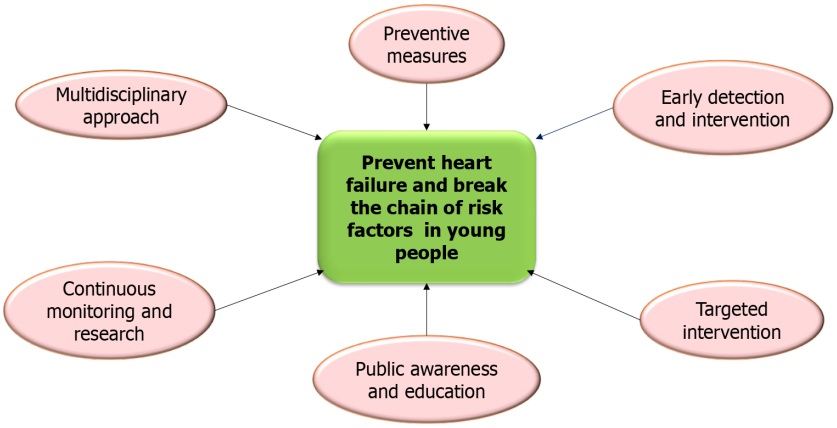

Figure 6 Prevent heart failure and break the risk factor chain in young people.

Pharmacological management

Pharmacological treatment remains the cornerstone of HF management, even for younger patients. However, the selection of medications may vary depending on the underlying cause of HF, such as genetic cardiomyopathies, substance abuse, or autoimmune conditions, for patients with HF and HFrEF, guideline-directed medical therapy is recommended. This includes angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, beta-blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter-2 (SGLT2) inhibitors. These medications have demonstrated significant benefits in improving survival rates and reducing hospitalizations in younger patients, similar to their effects in older populations[176,177].

Recent research has emphasized the advantages SGLT2 inhibitors, such as dapagliflozin and empagliflozin, in lowering the risk of HF-related hospitalizations and improving outcomes in younger individuals, including those without diabetes[178,179]. These drugs are particularly noteworthy due to their cardioprotective properties and ability to address metabolic risk factors. In younger patients with genetic cardiomyopathies, such as HCM or ARVD, antiarrhythmic medications like amiodarone or beta-blockers are frequently prescribed to reduce the risk of SCD[180].

Device therapy

Device-based interventions are essential in the management of HF in younger patients, especially those with genetic or congenital heart conditions. ICDs are often recommended for younger individuals with HF and HFrEF who are at high risk of SCD. This is especially relevant for patients with genetic cardiomyopathies or a history of VT. The use of device-based therapies, including ICDs, is crucial for the management of cardiac rhythm abnormalities and congestive heart failure in patients with congenital heart disease and inherited cardiac conditions[181]. Early implantation of ICDs in high-risk patients has been shown to significantly lower mortality rates[182]. Moreover, cardiac resynchronization therapy (CRT) is a valuable treatment option for younger patients with HFrEF who exhibit electrical dyssynchrony, such as a wide QRS complex. CRT effectively improved symptoms, reduced hospitalizations, and enhanced overall quality of life in younger populations[183].

Surgical and interventional approaches

For younger patients with structural heart disease or CHD, surgical or interventional procedures may be necessary. In cases of valvular heart diseases, surgical repair or replacement is often required. For example, patients with RHD may benefit from mitral valve repair or replacement to prevent the progression to HF[184]. In addition, patients with unrepaired or residual CHD may require surgical or catheter-based interventions to correct structural abnormalities. For instance, patients with TOF may need pulmonary valve replacement to address pulmonary regurgitation and prevent RV failure[97].

Lifestyle and behavioral interventions

Lifestyle and behavioral interventions play a crucial role in managing HF in young adults, especially with MetS or a history of substance abuse. Adopting a heart-healthy diet, such as the DASH diet, and engaging in regular physical activity, can significantly improve cardiovascular health and alleviate HF symptoms[185,186]. Structured exercise training programs, including aerobic and resistance training, have been demonstrated to enhance functional capacity and quality of life in younger patients with HF[187,188]. Furthermore, for individuals with HF related to substance abuse, such as cocaine or alcohol, cessation programs are essential. Behavioral interventions and medications like naltrexone for alcohol use disorder can significantly reduce substance consumption and lead to improved HF outcomes[189].

Diet and exercise: A heart-healthy diet, such as the DASH diet, combined with regular physical activity, can significantly improve cardiovascular health and reduce HF symptoms. Exercise training programs, including aerobic and resistance training, have enhanced functional capacity and quality of life in younger HF patients. Goyal *et al*[190] validates the DASH diet's role in reducing heart failure incidence, aligning with the manuscript's recommendation for dietary

interventions.

While this study focuses on adults, its findings reinforce the broader applicability of heart-healthy diets (*e.g.*, DASH) to younger populations with metabolic syndrome. The exercise training benefits cited in the text are supported by Volterrani *et al*[187] and Myers *et al*[188], ensuring a comprehensive evidence base for lifestyle strategies in HF management.

Substance abuse cessation: In patients with HF linked to substance abuse-particularly alcohol-cessation programs are a cornerstone of therapy. Chronic alcohol use not only exacerbates HF through direct myocardial toxicity and oxidative stress but also contributes to comorbidities like alcohol-associated cirrhosis, which further impairs cardiac function *via* systemic inflammation and hemodynamic instability[189]. Behavioral interventions, combined with pharmacotherapy such as naltrexone, have emerged as critical strategies to reduce alcohol dependence. A landmark Indian cohort study demonstrated that naltrexone significantly diminishes alcohol cravings and improves hepatic outcomes in cirrhotic patients, indirectly alleviating cardiac workload and reducing HF decompensation risks[189]. This dual benefit-targeting both addiction and its hepatic complications-highlights naltrexone's role in mitigating the bidirectional relationship between liver dysfunction and cardiovascular decline. Furthermore, by antagonizing opioid receptors, naltrexone may attenuate neurohormonal activation and inflammation, key drivers of adverse cardiac remodeling in this population[189].

Psychosocial support and mental health care

Given the high prevalence of mental health disorders among younger patients with HF and other chronic cardiac conditions, integrating psychosocial support into treatment plans is critical. A randomized controlled trial by Holdgaard *et al*[191] demonstrated that cognitive behavioral therapy (CBT) significantly reduces psychological distress in younger patients with cardiac disease, including those with chronic conditions such as HF. By addressing maladaptive thought patterns and promoting coping strategies, CBT not only alleviates anxiety and depression but also improves adherence to medical therapies-a key factor in optimizing HF outcomes. This aligns with growing evidence that psychological interventions should be a standard component of multidisciplinary care for younger HF patients, particularly given the bidirectional relationship between mental health and cardiovascular prognosis. Focusing on mental health concerns improves emotional well-being and enhances adherence to treatment and overall clinical outcomes. Moreover, a multidisciplinary care approach is essential for managing the complex needs of younger HF patients. This team-based model, which includes cardiologists, primary care physicians, mental health professionals, and social workers, ensures comprehensive care that addresses this population's physical and mental health challenges both the physical and mental health challenges faced by this population[38]. Such an integrated strategy is vital for optimizing long-term health and quality of life in younger individuals with HF.

Advanced therapies

For younger patients with end-stage HF, advanced therapies such as heart transplantation or mechanical circulatory support (MCS) may be considered.

Heart transplantation: Heart transplantation remains the gold standard for managing end-stage HF in younger individuals. Advances in immunosuppressive therapies have significantly improved long-term survival rates, with younger patients often achieving better outcomes than older recipients[192]. For patient's ineligible for heart transplantation, MCS provides a life-saving alternative. LVADs, such as the Heart Mate 3, have shown promising results in real-world settings. A multicenter European study by Numan *et al*[193] reported 80% survival at 1 year and 70% at 2 years post-Heart Mate 3 implantation, with significant improvements in functional capacity and quality of life. Notably, younger patients (< 60 years) experienced fewer device-related complications (*e.g.*, pump thrombosis, stroke) compared to older cohorts, reinforcing the role of continuous-flow LVADs as a durable therapy for select populations.

These advanced therapies represent critical options for extending and improving the lives of younger patients with severe HF (Figure 7).

The treatment of HF in younger populations requires a multifaceted approach tailored to address the unique risk factors and underlying causes specific to this demographic. An integrated approach, including pharmacological treatments, device-based therapies, surgical interventions, lifestyle changes, and psychosocial support is fundamental for effectively managing of HF and enhancing patient outcomes.

Early diagnosis is crucial for initiating timely interventions, while personalized treatment plans ensure that care aligns with individual patient needs. Additionally, a multidisciplinary care approach involving collaboration among cardiologists, primary care providers, mental health professionals, and other specialists. By integrating these strategies, healthcare providers can optimize long-term health and quality of life for this vulnerable population.

CONCLUSION

The increasing incidence of HF in younger populations represents a significant public health challenge that demands immediate attention. Emerging risk factors, such as MetS, substance use, autoimmune diseases, genetic predisposition, and the long-term cardiovascular effects of COVID-19, underscore the urgent need for a comprehensive approach to both the prevention and management of HF in this vulnerable group.

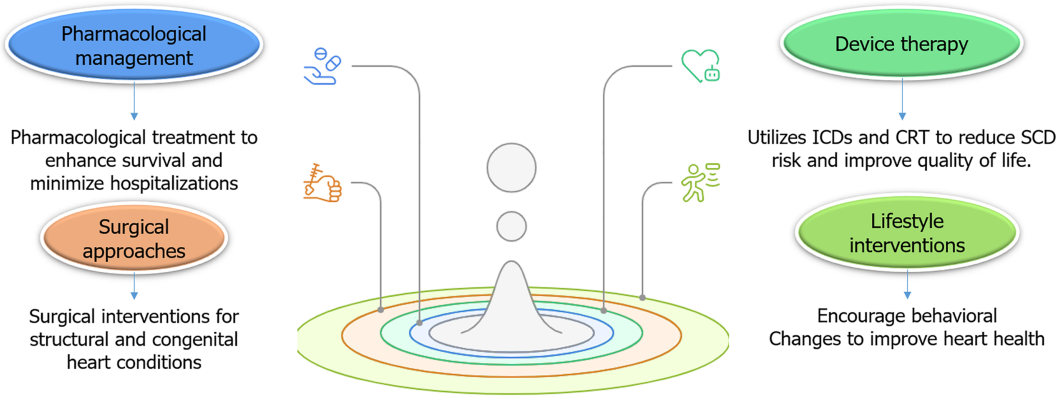

Figure 7 Treatment strategies for heart failure in younger populations. CRT: Cardiac resynchronization therapy; SCD: Sudden cardiac death; ICDs: Implantable cardioverter defibrillator.

Future research should focus on understanding the mechanisms driving these risk factors and developing targeted interventions to curb the rising rates of HF in younger individuals. By proactively addressing these factors, significant strides can be made in reducing the burden of HF and improving long-term health outcomes for younger populations. Early intervention, personalized care, and a comprehensive strategy combining medical, surgical, lifestyle, and psychosocial approaches are crucial in mitigating the impact of HF.

These efforts not only have the potential to improve survival rates but also to enhance the quality of life for younger patients, allowing them to lead healthier and more fulfilling lives. This proactive approach emphasizes the importance of prevention, timely treatment, and holistic care in managing HF in this demographic.

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FOOTNOTES

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