

## โรคหัวใจและโรคหลอดเลือดสมอง: ภาระโรคในระดับโลกและบทเรียนจากประเทศไทย Heart Disease and Stroke: Global Burden and Lessons from Thailand

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### บทคัดย่อ

โรคหัวใจและหลอดเลือด (CVD) และโรคหลอดเลือดสมองยังคงเป็นสาเหตุหลักของการเสียชีวิตและทุพพลภาพทั่วโลก คิดเป็นเกือบหนึ่งในสามของการเสียชีวิตทั้งหมด ในปี 2564 มีผู้เสียชีวิตจากสาเหตุโรคหัวใจและหลอดเลือดประมาณ 20.5 ล้านคน และมีผู้เสียชีวิตจากโรคหลอดเลือดสมองมากกว่า 6.5 ล้านคน โดยอุบัติการณ์โรคนี้สูง โดยเฉพาะในประเทศที่มีรายได้ต่ำและปานกลาง ซึ่งมีการเสียชีวิตจากการป่วยด้วยโรคทั้งสองนี้มากกว่า 80% ในประเทศไทยมีผู้เสียชีวิตจากโรคหัวใจมากกว่า 65,000 รายต่อปี และมีผู้ป่วยโรคหลอดเลือดสมองรายใหม่ประมาณ 350,000 รายในปี 2566 ส่วนใหญ่มีภาวะทุพพลภาพระยะยาว บทความนี้เป็นบทความปริทัศน์ที่สรุปและวิเคราะห์ข้อมูลจากรวรรณกรรมวิชาการที่เกี่ยวข้องเกี่ยวกับโรคหัวใจและหลอดเลือดสมอง ทั้งในระดับโลกและบริบทของประเทศไทย ทั้งสองโรครามีพยาธิสรีรวิทยาาร่วมกัน เช่น ความผิดปกติของเซลล์บุผนังหลอดเลือด การอักเสบเรื้อรัง ภาวะออกซิเดชันเกิน และหลอดเลือดแข็งตัว ซึ่งส่งผลกระทบต่อการทำงานของหลอดเลือดและหัวใจ ปัจจัยเสี่ยงที่สามารถป้องกันได้ เช่น ความดันโลหิตสูง เบาหวาน โรคอ้วน การสูบบุหรี่ การบริโภคอาหารที่ไม่เหมาะสม และมลพิษทางอากาศ มีส่วนทำให้เกิดโรคเพิ่มขึ้น ขณะที่ปัจจัยเสี่ยงที่ไม่สามารถเปลี่ยนแปลงได้ เช่น อายุ เพศ และพันธุกรรมมีผลร่วมในการเพิ่มความเสี่ยง การวินิจฉัยที่มีความก้าวหน้า เช่น การใช้ MRI สำหรับโรคหลอดเลือดสมองตีบ และการตรวจ Troponin สำหรับกล้ามเนื้อหัวใจตายเฉียบพลัน ทำให้การวินิจฉัยมีความแม่นยำมากขึ้น การรักษามีการพัฒนา เช่น การดัดเลือดด้วยสายสวน การใช้ยาต้านการแข็งตัวของเลือดชนิดใหม่ และการใช้เครื่องมือในการรักษา ผู้ป่วยโรคหัวใจระยะรุนแรง ทำให้ผลลัพธ์การรักษาดีขึ้น การป้องกันโรคในระดับปฐมภูมิ ทุติยภูมิ และตติยภูมิ ได้รับการดำเนินการแล้ว แต่ยังมีช่องว่างในการวิจัย เช่น ผลกระทบระยะยาวของมลพิษทางอากาศ การวิจัยทางพันธุกรรม ในประชากรเอเชีย และการพัฒนาโปรแกรมป้องกันที่เหมาะสมในระบบสุขภาพของไทย การเสริมสร้างการป้องกันโรค

การเพิ่มการเข้าถึงการวินิจฉัยและการรักษาที่มีคุณภาพ และการเติมเต็มช่องว่างในการวิจัยเป็นแนวทางสำคัญในการลดการเสียชีวิตก่อนวัยอันควรและภาวะทุพพลภาพจากโรคหัวใจและโรคหลอดเลือดสมอง

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## Abstract

Cardiovascular diseases (CVD) and stroke remain the leading causes of death and disability worldwide, accounting for nearly one-third of all global deaths. In 2021, cardiovascular diseases caused approximately 20.5 million deaths, while stroke was responsible for more than 6.5 million deaths. The burden of these diseases is particularly high in low- and middle-income countries (LMICs), where over 80% of all deaths from these conditions occur.

In Thailand, more than 65,000 people die from heart disease each year, and there were approximately 350,000 new stroke cases reported in 2023 most of which resulted in long-term disability. This review summarizes and analyzes current literature on cardiovascular disease and stroke, integrating global evidence with data relevant to the Thai context.

Both CVD and stroke share common pathophysiological mechanisms, including endothelial dysfunction, chronic inflammation, oxidative stress, and atherosclerosis, which affect the functioning of blood vessels and the heart. Modifiable risk factors such as hypertension, diabetes, obesity, smoking, poor diet, and air pollution contribute significantly to the increased prevalence of these diseases, while non-modifiable factors such as age, sex, and genetics interact with modifiable ones to exacerbate the risk. Advances in diagnostics, such as MRI for ischemic stroke and troponin tests for acute myocardial infarction, have improved diagnostic accuracy, while treatment innovations, including mechanical thrombectomy, newer anticoagulants, and device-based therapies for advanced heart disease, have enhanced patient outcomes. Prevention strategies are implemented at the primary, secondary, and tertiary levels; however, gaps remain in areas like the long-term effects of air pollution, genetic research in Asian populations, and the development of suitable prevention programs in Thailand's healthcare system. Strengthening disease prevention, improving access to quality diagnostics and treatment, and addressing these research gaps are crucial steps to reduce premature mortality and disability due to CVD and stroke.

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## Introduction

Cardiovascular disease (CVD) encompassing both heart disease and stroke remains the foremost cause of death and disability worldwide. Together, these conditions are responsible for nearly one-third of global mortality. Beyond their contribution to premature death, they place a substantial and growing strain on health systems through long-term disability, escalating healthcare expenditures, and reduced quality of life. The impact is particularly pronounced in low- and middle-income countries (LMICs), where the majority of cardiovascular deaths occur<sup>(1,2)</sup>.

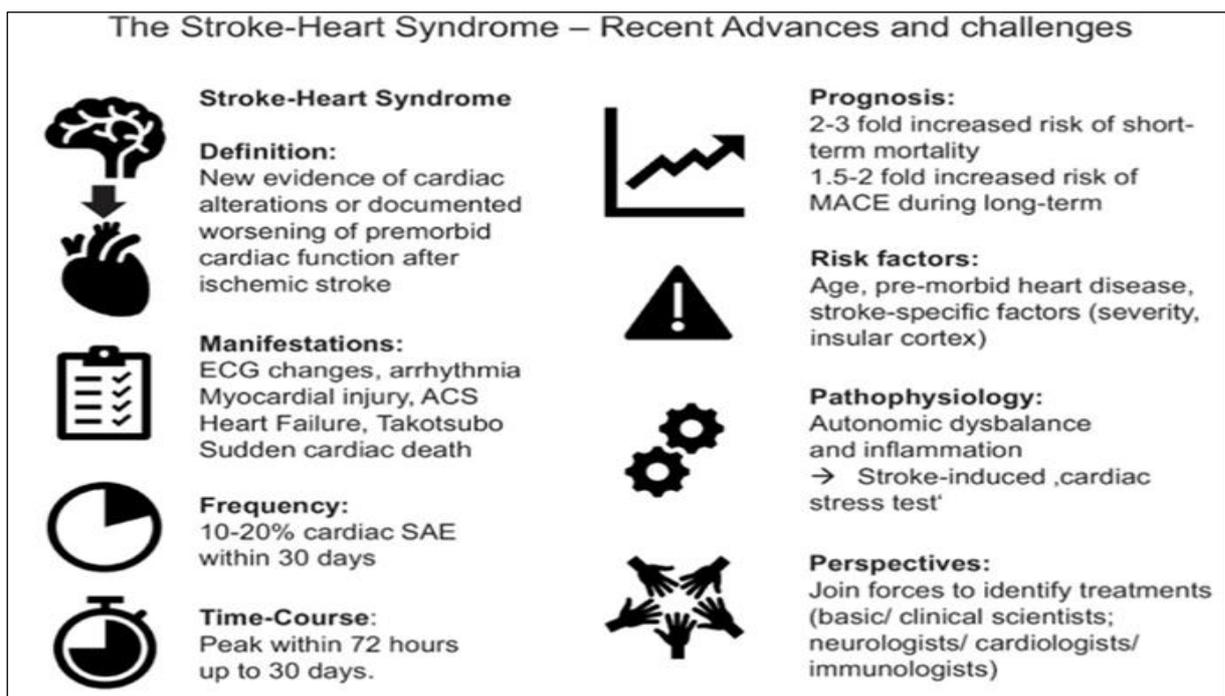
Over recent decades, the burden of CVD and stroke has increased steadily, driven by population aging, urbanization, and persistent lifestyle-related risk factors. In Thailand, this global pattern is reflected in the continued prominence of heart disease and stroke as leading causes of death and disability, highlighting the urgent need to strengthen prevention, early detection, and long-term management efforts<sup>(3,4)</sup>.

Understanding the biological and mechanistic basis of these rising trends is crucial for developing effective prevention and management strategies. Therefore, the following section reviews current scientific evidence on the underlying pathophysiology of cardiovascular disease and stroke.

A comprehensive literature search was conducted using PubMed, Scopus, and official WHO and Thai Ministry of Public Health databases to identify English and Thai-language publications from January 2013 to August 2025. Search terms included cardiovascular disease, stroke, Thailand, prevention, and thrombectomy. Studies were selected based on methodological rigor, relevance to cardiovascular and cerebrovascular disease, and applicability to the Thai context. Preference was given to clinical guidelines, meta-analyses, large cohort studies, and government reports that provided current epidemiological or policy data.

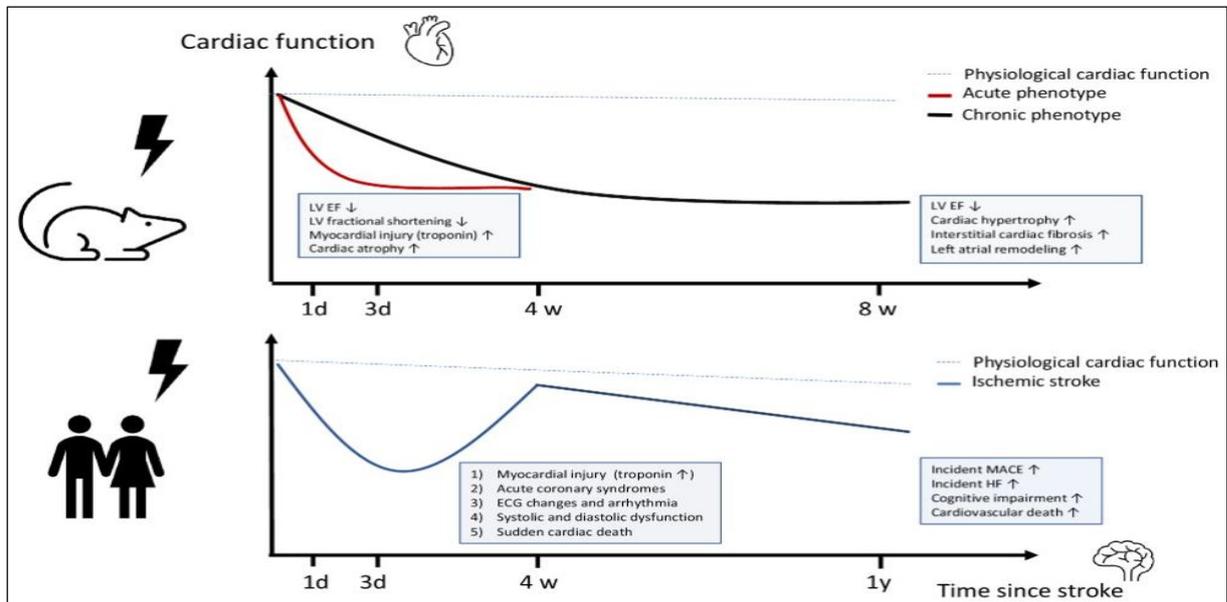
## Literature Review

Despite advances in treatment and prevention, the pathophysiology of CVD and stroke underscores their complex, multifactorial nature. Endothelial dysfunction, chronic inflammation, oxidative stress, and genetic predispositions interact with lifestyle-related risk factors to accelerate atherosclerosis, vascular remodeling, and plaque instability, ultimately leading to acute cardiovascular and cerebrovascular events<sup>(5)</sup>. Importantly, the interconnection between these conditions is increasingly recognized, as illustrated by the “stroke-heart syndrome,” where acute ischemic stroke precipitates cardiac dysfunction through systemic inflammation and autonomic dysregulation<sup>(6)</sup>.



**Figure 1** Summary of key criteria and of the Stroke-Heart Syndrome. ACS indicates acute coronary syndrome; MACE, major adverse cardiovascular events; and SAE, severe adverse events<sup>(6)</sup>.

The majority of cardiovascular and stroke events are attributable to modifiable risk factors, such as hypertension, diabetes, obesity, smoking, poor diet, and environmental exposures including air pollution<sup>(7)</sup>. However, non-modifiable risks, including age, sex, and genetic predispositions such as APOE and NOTCH3 mutations, remain important determinants of disease burden<sup>(8,9)</sup>. This duality highlights the importance of comprehensive strategies that combine risk prediction, early detection, evidence-based treatment, and population-level prevention.



**Figure 2** Phenotypes and time course of the Stroke-Heart Syndrome in rodent models and clinical practice<sup>(6)</sup>.

Given the immense burden of CVD and stroke, and the persistence of preventable risk factors despite decades of intervention, it is essential to critically review current evidence. This paper synthesizes recent literature on the epidemiology, pathophysiology, risk factors, diagnostics, management, and prevention of heart disease and stroke. Special attention is given to the Thai context within a global framework, identifying knowledge gaps and highlighting future directions for research, policy, and practice. To begin this synthesis, the following section examines the global and national epidemiology of cardiovascular disease and stroke, outlining the magnitude, trends, and distribution of their burden across populations.

### Epidemiology & Burden

Heart disease and stroke remain the two leading contributors to global mortality and disability, imposing an unparalleled burden on public health systems worldwide. Cardiovascular disease (CVD) causes an estimated 20.5 million deaths each year, representing nearly one-third of all global deaths, while stroke contributes approximately 6.5 million deaths annually. Together, these conditions are responsible for the greatest share of noncommunicable disease (NCD) deaths globally, with stroke ranking as the second leading cause of death and the third leading cause of combined death and disability worldwide<sup>(1)</sup>. Beyond mortality, millions of survivors live with long-term sequelae, including physical disability, cognitive impairment, and diminished quality of life, which place an enormous strain on healthcare systems, economies, and families.

The global prevalence of CVD has risen dramatically over the past three decades, nearly doubling from 271 million in 1990 to 523 million in 2019. This escalation is driven not only by demographic shifts such as population aging and urbanization but also by persistent lifestyle-related risk factors, including poor diet, physical inactivity, smoking, and obesity<sup>(2)</sup>. Similarly, the global burden of stroke has increased substantially, with projections estimating an 81% rise in new cases by 2050, underscoring the urgency of more effective prevention, screening, and management strategies<sup>(3)</sup>.

In Thailand, these global patterns are mirrored with alarming implications. Heart disease remains a leading cause of premature mortality, responsible for over 65,000 deaths annually equivalent to five deaths every hour<sup>(4)</sup>. Stroke is the second leading cause of death, with 349,126 reported cases in 2023, a case fatality rate of about 10%, and long-term disability in more than 60% of survivors<sup>(4)</sup>. Hospital data further illustrate the challenge: heart failure admissions increased from 1.2% in 2018 to 1.5% in 2019, and the combined rate of rehospitalization and mortality among these patients remains persistently high at around 34%, reflecting ongoing gaps in chronic disease management.

At the regional level, Thailand's cardiovascular mortality rates fall within the mid-range of the ASEAN average (123.2-406.2 deaths per 100,000 population in 2022). While ASEAN as a whole has experienced a 16.9% decline in CVD mortality since 1990, the absolute burden in Thailand continues to rise due to population growth, increased life expectancy, and escalating NCD risk factors, particularly diabetes, hypertension, and obesity<sup>(10)</sup>. Unlike many high-income countries that have successfully reduced CVD mortality through early detection, advanced acute care, and preventive strategies, Thailand faces challenges related to uneven healthcare access, delayed diagnosis, and limited integration of preventive programs at the community level.

These trends highlight the urgent need for targeted prevention and control strategies tailored to Thailand's demographic and socioeconomic context. Priority areas include scaling up risk factor screening, strengthening health literacy, improving access to diagnostic and rehabilitation services, and implementing population-wide interventions such as tobacco control, dietary salt reduction, and policies addressing environmental exposures. Without decisive action, the burden of CVD and stroke in Thailand is projected to rise further, exacerbating health inequities and straining national healthcare resources. The subsequent section discusses the key pathophysiological mechanisms linking cardiovascular and cerebrovascular disease.

### **Pathophysiology**

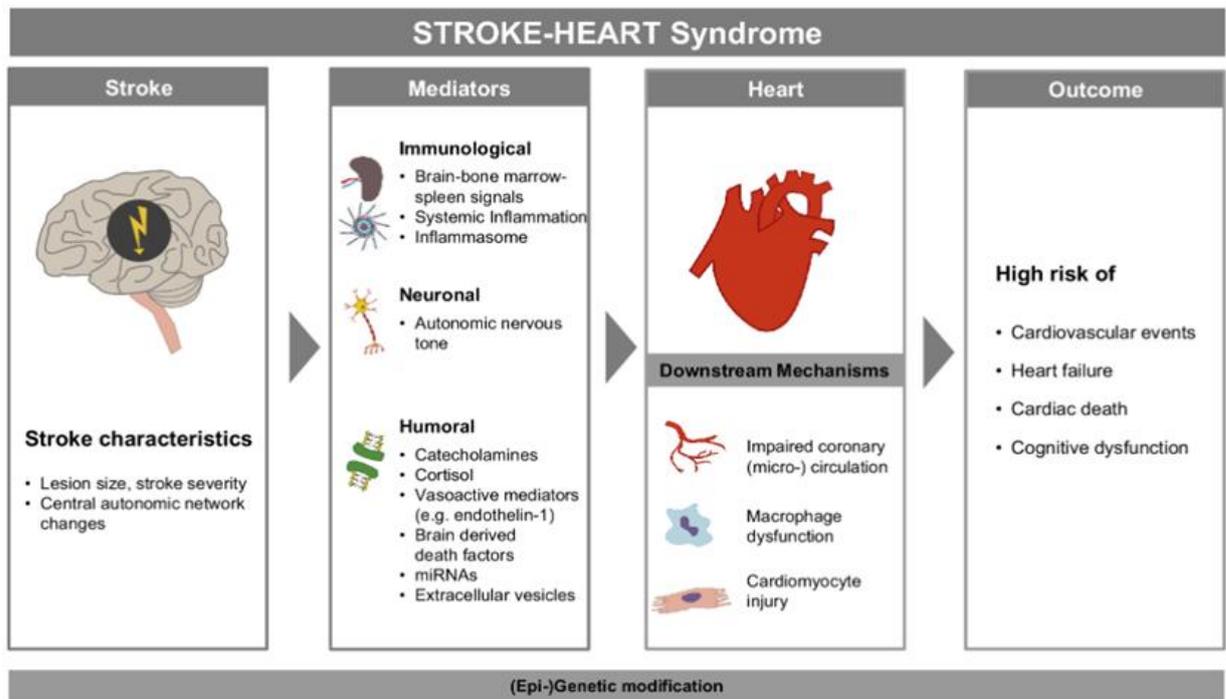
Cardiovascular disease (CVD) develops through a complex interplay of vascular, metabolic, inflammatory, and genetic mechanisms. A central initiating event is endothelial dysfunction, which disrupts the normal barrier and regulatory functions of the vascular endothelium. This dysfunction increases vascular permeability, promotes lipid infiltration into the arterial wall, and reduces nitric oxide bioavailability, thereby impairing vasodilation<sup>(5)</sup>. Over time, low-density lipoprotein (LDL) cholesterol particles accumulate beneath the endothelium and undergo oxidative modification. These oxidized lipids stimulate

the recruitment of monocytes and the formation of foam cells, which contribute to the development of fatty streaks, the earliest visible lesions of atherosclerosis.

As the disease progresses, chronic inflammation and oxidative stress drive further vascular injury. Activated macrophages and T cells release cytokines, proteases, and reactive oxygen species (ROS), which weaken the fibrous cap covering atherosclerotic plaques and increase the risk of rupture<sup>(5)</sup>. This process, combined with vascular remodeling, leads to narrowing of the arterial lumen and reduced blood flow. When plaques rupture or erode, they expose thrombogenic material to circulating blood, activating platelets and the coagulation cascade. The result is thrombosis, which can abruptly occlude blood flow, producing acute clinical events such as myocardial infarction or ischemic stroke<sup>(11)</sup>.

The pathophysiology of stroke depends on its subtype. Ischemic stroke, which accounts for about 87% of cases, occurs when an arterial occlusion restricts blood supply to brain tissue. This triggers a cascade of energy failure due to reduced oxygen and glucose delivery, leading to ATP depletion, ionic pump dysfunction, and accumulation of intracellular sodium and calcium. These changes induce cytotoxic edema and excitotoxicity, whereby excess glutamate overstimulates NMDA and AMPA receptors, further amplifying neuronal injury. Secondary neuroinflammation, involving microglial activation and cytokine release, exacerbates tissue damage and contributes to blood-brain barrier disruption<sup>(12)</sup>.

In contrast, hemorrhagic stroke results from the rupture of a weakened cerebral vessel, causing bleeding into brain tissue or the subarachnoid space. The initial primary injury involves mechanical compression of adjacent neurons and glia by the expanding hematoma. Subsequently, secondary injury mechanisms including oxidative stress from hemoglobin breakdown, iron-induced toxicity, and inflammatory cytokine release contribute to edema, disruption of neural networks, and progressive neurological deterioration<sup>(12)</sup>. An emerging concept linking cardiovascular and cerebrovascular pathology is the “stroke-heart syndrome.” Following an acute ischemic stroke, systemic autonomic imbalance and heightened sympathetic nervous system activation can trigger myocardial injury, arrhythmias, and left ventricular dysfunction<sup>(6)</sup>. This illustrates the bidirectional relationship between the brain and the heart, where vascular events in one organ precipitate dysfunction in the other, reinforcing the need for integrated prevention and management strategies, as presented in Figure 3 illustrates the mechanistic framework of the Stroke-Heart Syndrome, highlighting how stroke characteristics trigger immunological, neuronal, and humoral responses that mediate downstream cardiac injury and contribute to adverse outcomes.



**Figure 3** Conceptual framework of the Stroke Heart Syndrome.

Stroke severity and lesion characteristics initiate immunological, neuronal, and humoral pathways that impair coronary microcirculation, disrupt macrophage function, and injure cardiomyocytes, ultimately increasing the risk of cardiovascular events, heart failure, cardiac death, and cognitive dysfunction<sup>(6)</sup>. A clear understanding of the biological mechanisms underlying cardiovascular and cerebrovascular disease provides the foundation for identifying the major risk factors that influence their development and progression.

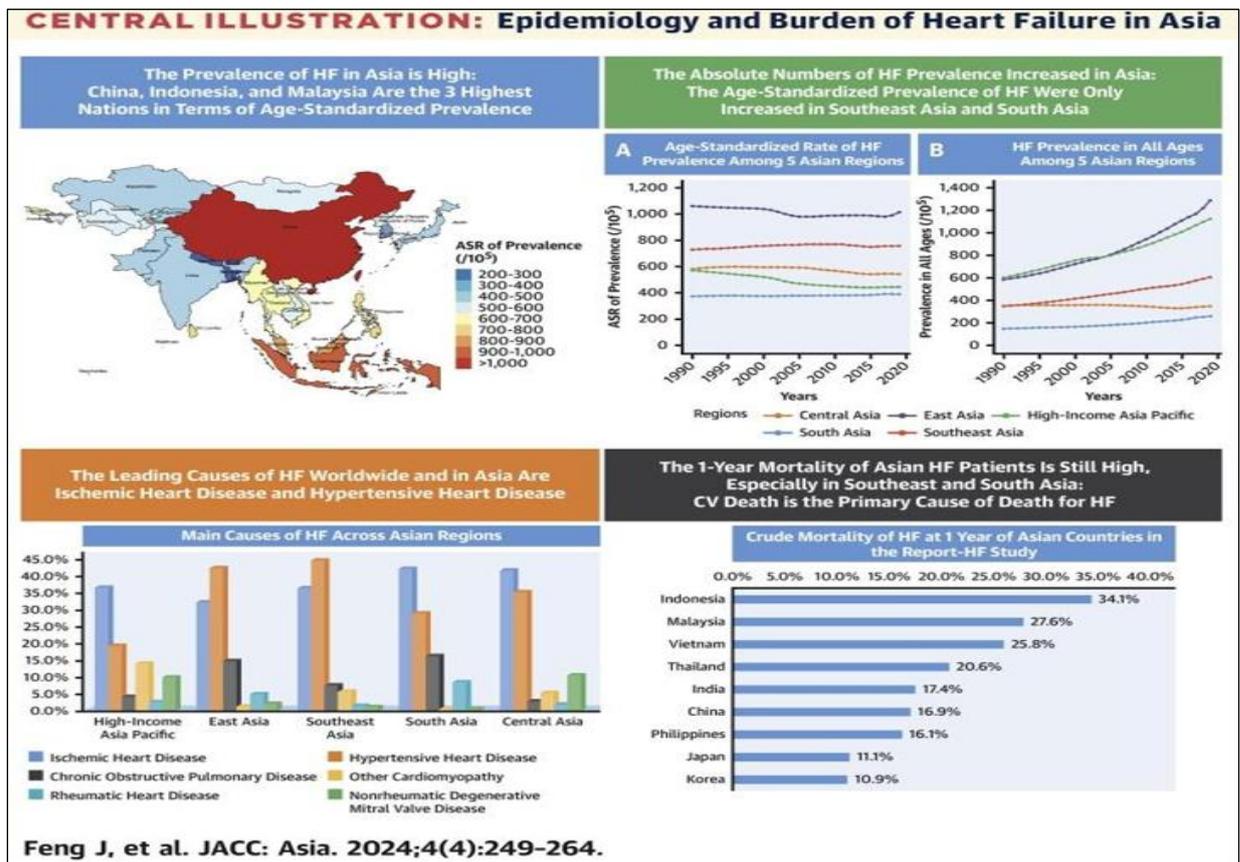
### Risk Factors

The development of cardiovascular disease (CVD) and stroke is influenced by a combination of non-modifiable and modifiable risk factors, which interact across the lifespan to determine individual and population-level disease burden.

### Non-Modifiable Risk Factors

Age remains the strongest predictor of CVD and stroke. The risk of coronary artery disease, myocardial infarction, and ischemic stroke rises steeply after age 65, reflecting cumulative vascular damage, arterial stiffening, and the long-term effects of risk factor exposure<sup>(7)</sup>. Sex differences are also well documented: men have higher rates of ischemic heart disease during middle age, but women often experience worse long-term outcomes after stroke, including greater disability and reduced quality of life. Hormonal influences, particularly the loss of estrogen after menopause, contribute to accelerated atherosclerosis in women<sup>(11)</sup>.

This trend in Thailand reflects broader regional patterns. Figure 4 illustrates the epidemiology and burden of heart failure across Asia, showing high prevalence in Southeast Asia and persistently high one-year mortality rates, including Thailand Figure 4.



**Figure 4** Epidemiology and burden of heart failure in Asia.

The figure shows age-standardized prevalence trends, regional variation in underlying causes, and one-year mortality rates across Asian countries, including Thailand<sup>(13)</sup>.

Genetics also play a critical role in predisposing individuals to cardiovascular events. Inherited conditions such as familial hypercholesterolemia (FH) result in lifelong elevations of low-density lipoprotein (LDL) cholesterol, dramatically increasing the risk of premature atherosclerosis and coronary events<sup>(8)</sup>. Variants of the apolipoprotein E (APOE) gene affect lipid metabolism and are associated with elevated cholesterol levels and an increased risk of ischemic stroke. Likewise, mutations in the NOTCH3 gene are responsible for cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), a hereditary small vessel disease that substantially raises the risk of ischemic stroke<sup>(9)</sup>. These findings highlight the importance of early detection and family-based screening in high-risk populations.

### Modifiable Risk Factors

In contrast, modifiable risk factors account for the majority of global cardiovascular events, underscoring the potential for prevention. Hypertension is the leading contributor to both ischemic and hemorrhagic stroke, as well as ischemic heart disease, by promoting vascular remodeling, endothelial dysfunction, and left ventricular hypertrophy<sup>(1)</sup>. Diabetes mellitus accelerates atherosclerosis through mechanisms such as hyperglycemia-induced endothelial injury, oxidative stress, and increased coagulability. Obesity, particularly central obesity, contributes to insulin resistance, dyslipidemia, and systemic inflammation, magnifying cardiovascular risk.

Tobacco use is one of the most preventable causes of CVD. Smoking increases LDL cholesterol, reduces protective high-density lipoprotein (HDL) cholesterol, and promotes

endothelial dysfunction, platelet aggregation, and chronic inflammation. These effects significantly accelerate atherosclerosis and double the risk of ischemic heart disease and stroke<sup>(14)</sup>.

Environmental and lifestyle exposures also play an increasingly recognized role. Air pollution, particularly fine particulate matter (PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>), and black carbon, is strongly associated with elevated rates of ischemic stroke, myocardial infarction, and cardiovascular mortality. Importantly, these associations persist even at concentrations below current legal thresholds, suggesting a nonlinear, supra-linear exposure-response relationship. Additional modifiable factors include excessive alcohol consumption, physical inactivity, and diets high in saturated fat, processed meat, and refined sugars all of which contribute to obesity, hypertension, and metabolic dysfunction. Given the wide range of biological and behavioral risk factors, accurate and timely diagnosis is essential to identify affected individuals and guide appropriate management.

## Findings

### Diagnostics

Accurate and timely diagnosis is critical in both stroke and cardiovascular disease (CVD), as early recognition directly influences treatment decisions, clinical outcomes, and long-term prognosis. Advances in diagnostic technologies have enabled earlier detection of structural and functional abnormalities, yet challenges remain in accessibility and implementation across different healthcare settings.

### Stroke Diagnostics

For suspected stroke, neuroimaging is the cornerstone of diagnosis and subtype classification. Computed tomography (CT) is the most widely used first-line tool because it is fast, widely available, and highly effective in ruling out intracranial hemorrhage information that is essential before administering thrombolytic therapy<sup>(12)</sup>. However, CT has limited sensitivity in detecting early ischemic changes, particularly within the first hours of symptom onset or in small or posterior fossa infarcts.

Magnetic resonance imaging (MRI), particularly diffusion-weighted imaging (DWI), provides superior sensitivity and specificity for detecting acute ischemic lesions within minutes of onset, often before changes appear on CT. MRI can also better characterize brainstem and lacunar infarcts, and additional sequences such as susceptibility-weighted imaging (SWI) and perfusion-weighted imaging (PWI) provide valuable insights into hemorrhage and tissue at risk. Despite these advantages, MRI is slower, more costly, and less available in smaller hospitals, limiting its use as a universal first-line test<sup>(12)</sup>.

Beyond imaging, clinical assessments such as the National Institutes of Health Stroke Scale (NIHSS) provide standardized evaluation of stroke severity, while vascular studies including carotid duplex ultrasound, CT angiography (CTA), and MR angiography (MRA) are critical for identifying large vessel occlusion and guiding mechanical thrombectomy decisions. Cardiac evaluations such as electrocardiography (ECG) and echocardiography may also be performed to detect potential cardioembolic sources, particularly atrial fibrillation and structural heart abnormalities.

## Cardiovascular Disease Diagnostics

For CVD, diagnosis typically begins with electrocardiography (ECG), which detects arrhythmias, conduction abnormalities, and ischemic changes such as ST-segment elevation. Cardiac biomarkers, especially troponins I and T, are essential for confirming myocardial injury and remain the gold standard for diagnosing acute myocardial infarction<sup>(11)</sup>.

Echocardiography provides real-time assessment of cardiac structure and function, including wall motion abnormalities, ejection fraction, and valvular disease. Advanced modalities such as stress testing (exercise or pharmacologic), coronary computed tomography angiography (CCTA), and cardiac magnetic resonance imaging (CMR) offer noninvasive evaluation of myocardial perfusion, viability, and fibrosis. Invasive techniques such as coronary angiography remain the gold standard for defining coronary artery anatomy and guiding revascularization strategies, including angioplasty and stenting.

In addition, risk stratification tools such as the Framingham Risk Score or SCORE2 are widely used to estimate future cardiovascular risk and guide preventive interventions. Emerging technologies, including high-sensitivity cardiac troponin assays, coronary artery calcium scoring, and artificial intelligence-enhanced imaging, further enhance early detection and risk prediction. Effective treatment depends on precise diagnosis; therefore, the next section discusses current evidence-based management strategies for stroke and cardiovascular disease.

## Management & Treatment

### Stroke Management

The management of stroke depends on accurate classification into ischemic or hemorrhagic subtypes, as therapeutic strategies differ substantially.

Ischemic stroke management emphasizes timely reperfusion. Intravenous alteplase (tPA) remains the cornerstone of therapy and is most effective when administered within 4.5 hours of symptom onset, as early treatment improves functional outcomes and reduces long-term disability<sup>(15)</sup>. For patients with large vessel occlusions (LVOs), mechanical thrombectomy has transformed care, providing substantial benefit when performed within 6 hours, and in carefully selected patients with favorable imaging profiles (e.g., DAWN or DEFUSE-3 criteria), up to 24 hours after last known well<sup>(16)</sup>. Secondary prevention is initiated immediately after the acute phase, including dual antiplatelet therapy (aspirin and clopidogrel for short-term use), statins, antihypertensives, and anticoagulation in cases of atrial fibrillation<sup>(17)</sup>.

Hemorrhagic stroke management focuses on stabilizing the patient and minimizing further bleeding. Immediate goals include blood pressure control, often targeting systolic pressures around 140 mmHg using intravenous antihypertensives such as nicardipine or labetalol<sup>(18)</sup>. For patients on anticoagulants, reversal strategies are crucial vitamin K and four-factor prothrombin complex concentrate (PCC) for warfarin, and specific reversal agents (idarucizumab for dabigatran; andexanet alfa for apixaban or rivaroxaban) for direct oral anticoagulants. Surgical options, such as hematoma evacuation, external ventricular drainage, or decompressive craniectomy, may be indicated in cases of large bleeds, brainstem

compression, or hydrocephalus<sup>(19)</sup>. Rehabilitation, including physical, occupational, and speech therapy, begins early to optimize functional recovery.

### **Heart Disease Management**

The management of CVD requires a combination of pharmacologic, interventional, and lifestyle-based strategies tailored to disease severity and patient risk profile.

Pharmacologic therapy forms the foundation of management. Antiplatelet agents (aspirin, clopidogrel, ticagrelor) reduce thrombotic risk in patients with coronary artery disease. Statins lower LDL cholesterol and stabilize atherosclerotic plaques, providing both primary and secondary prevention benefits. Antihypertensives such as ACE inhibitors, ARBs, beta-blockers, and calcium channel blockers reduce blood pressure and cardiovascular risk. For patients with atrial fibrillation, oral anticoagulation with warfarin or direct oral anticoagulants (DOACs) prevents cardioembolic stroke<sup>(7)</sup>.

Interventional and surgical strategies are indicated for patients with advanced disease or high-risk anatomy. Percutaneous coronary intervention (PCI) with angioplasty and stenting is standard for acute myocardial infarction and high-grade coronary stenosis. Coronary artery bypass grafting (CABG) provides durable benefit in multi-vessel or left main disease, especially among diabetic patients<sup>(20)</sup>. For heart failure, device-based therapies such as implantable cardioverter-defibrillators (ICDs), cardiac resynchronization therapy (CRT), and left ventricular assist devices (LVADs) improve survival and quality of life in appropriately selected patients.

Lifestyle modification remains critical across all stages of heart disease, including smoking cessation, weight management, dietary changes (e.g., Mediterranean diet), regular physical activity, and optimal diabetes control. Cardiac rehabilitation programs integrating exercise, education, and psychosocial support reduce recurrence and improve long-term outcomes<sup>(21)</sup>. While advances in acute management have improved survival, reducing the overall burden of disease ultimately relies on effective prevention at all stages of care.

### **Prevention**

Effective prevention of cardiovascular disease (CVD) and stroke requires a multilevel approach encompassing primary, secondary, and tertiary strategies. Each level targets different stages in the disease continuum, from risk identification to advanced care, with the overarching goal of reducing morbidity, mortality, and healthcare burden.

#### **Primary Prevention**

Primary prevention focuses on reducing the incidence of first-time cardiovascular and cerebrovascular events by addressing modifiable risk factors before disease onset. Key strategies include lifestyle modification, such as smoking cessation, maintaining a healthy diet, engaging in regular physical activity, achieving optimal body weight, and moderating alcohol intake<sup>(1)</sup>. National and international guidelines emphasize structured risk assessment tools, including the Framingham Risk Score, SCORE2, and region-specific calculators, which estimate 10-year cardiovascular risk and guide tailored interventions<sup>(22)</sup>.

The American Heart Association's Life's Essential 8 framework provides a practical roadmap, promoting four health behaviors (healthy diet, regular physical activity, adequate

sleep, and tobacco avoidance) and four health factors (optimal weight, blood pressure, cholesterol, and glucose control) to reduce CVD burden<sup>(22)</sup>. Preventive pharmacotherapy, such as antihypertensives or statins, may be considered in high-risk individuals even before clinical events occur, further strengthening primary prevention.

#### Secondary Prevention

Secondary prevention targets patients with established CVD or prior stroke, aiming to prevent recurrence, progression, or related complications. Evidence-based interventions include antiplatelet therapy (e.g., aspirin, clopidogrel), lipid-lowering agents (statins, PCSK9 inhibitors), antihypertensives, and anticoagulation in atrial fibrillation to reduce cardioembolic stroke risk<sup>(23)</sup>. In addition to pharmacologic therapy, structured rehabilitation programs including cardiac rehabilitation and post-stroke rehabilitation playing a crucial role in restoring functional capacity, improving quality of life, and reducing long-term disability.

Behavioral counseling and patient education are also integral to secondary prevention, encouraging adherence to lifestyle modification and medication regimens. Regular follow-up and risk monitoring help detect recurrent events early and optimize therapy.

#### Tertiary Prevention

Tertiary prevention is directed toward individuals with advanced disease or long-term sequelae, focusing on reducing complications, disability, and mortality while improving functional independence and quality of life. In CVD, this often involves surgical or device-based interventions such as percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), implantable cardioverter-defibrillators (ICDs), cardiac resynchronization therapy (CRT), and left ventricular assist devices (LVADs)<sup>(1)</sup>. For stroke, tertiary prevention includes surgical interventions such as carotid endarterectomy or stenting for patients with severe carotid stenosis, as well as long-term rehabilitation tailored to neurological deficits.

Equally important are psychosocial support and multidisciplinary care, which address depression, cognitive impairment, and caregiver burden frequently associated with chronic cardiovascular and cerebrovascular disease. By integrating medical, surgical, and supportive care, tertiary prevention helps patients maintain independence and enhances overall well-being despite advanced disease. To better understand how preventive and management strategies translate into real-world outcomes, the following section compares global trends with the Thai experience.

#### Thailand vs Global Context

Thailand reflects global patterns in the rising burden of cardiovascular disease (CVD) and stroke, with both conditions among the leading causes of mortality and disability. Each year, more than 65,000 deaths from CVD are reported equivalent to five deaths per hour while stroke accounts for nearly 350,000 new cases, with approximately 10% mortality and long-term disability in over 60% of survivors<sup>(4)</sup>. The burden of heart failure is also escalating, with hospitalization rates rising from 1.2% in 2018 to 1.5% in 2019 and combined rehospitalization and mortality rates remaining at a concerning 34%<sup>(4)</sup>. These figures illustrate not only the acute clinical impact but also the long-term strain on Thailand's healthcare system, social services, and families, particularly given the country's aging population and limited rehabilitation infrastructure.

Within the ASEAN region, Thailand's cardiovascular mortality rate falls in the mid-range of 123.2 to 406.2 deaths per 100,000 population. While the region overall has achieved a 16.9% reduction in CVD-related mortality since 1990, absolute case numbers continue to rise due to population growth, aging, and increasing exposure to modifiable risk factors such as hypertension, diabetes, obesity, and air pollution<sup>(10)</sup>. Thailand's epidemiological profile highlights a double burden of disease: infectious diseases have declined, but noncommunicable diseases (NCDs) now account for over 70% of deaths nationwide, reflecting an incomplete epidemiological transition.

Globally, the picture is similar, but disparities are more pronounced. Low- and middle-income countries (LMICs) account for nearly 80% of cardiovascular deaths, underscoring inequities in healthcare access, early detection, and preventive strategies<sup>(2)</sup>. High-income countries (HICs) have recorded sharp declines in CVD mortality over recent decades through widespread use of risk screening, preventive pharmacotherapy (e.g., statins, antihypertensives), and advanced acute interventions such as percutaneous coronary intervention (PCI) and mechanical thrombectomy. In contrast, Thailand and other LMICs face persistent challenges: limited diagnostic capacity (MRI, catheterization facilities concentrated in urban centers), delays in patient presentation, and underutilization of preventive care due to geographic, financial, and cultural barriers. For example, while thrombolysis and thrombectomy are available in tertiary hospitals, access in rural areas is limited, contributing to poorer functional outcomes compared with HICs.

These comparisons suggest that Thailand has made progress particularly through universal health coverage (UHC), which has improved access to essential services but significant implementation gaps remain. Primary prevention programs often lack sustained funding and integration into community health systems. Secondary prevention strategies, including rehabilitation, are underdeveloped and unevenly distributed. Social determinants of health, such as low health literacy, socioeconomic inequality, and environmental exposures, exacerbate disparities, particularly in rural and peri-urban populations.

For Thailand, the path forward requires both adopting global best practices and tailoring strategies to local contexts. This includes scaling up risk factor screening, investing in diagnostic and acute care infrastructure outside metropolitan centers, and strengthening rehabilitation and community-based services. Without such measures, Thailand risks falling further behind high-income counterparts, with long-term consequences for mortality, disability, and healthcare sustainability. Despite progress in prevention and management, substantial scientific and implementation gaps remain. The final section highlights key areas for future research, policy development, and health-system improvement.

## Discussion

### Knowledge Gaps & Future Directions

Despite progress in the prevention and management of cardiovascular disease (CVD) and stroke, significant gaps remain in both scientific knowledge and health system implementation. One major uncertainty concerns the long-term effects of low-level environmental exposures, particularly air pollution. While fine particulate matter (PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>), and other

pollutants are consistently linked with increased risks of ischemic stroke and cardiovascular mortality, most studies come from high-income countries and focus on short-term exposures. More longitudinal research in low- and middle-income countries (LMICs), including Thailand, is needed to clarify cumulative lifetime risks, dose–response relationships, and safe thresholds for exposure. Future work should focus on developing and applying effective implementation strategies to close evidence-to-practice gaps across urban and rural care pathways.

Another underexplored area is the stroke–heart syndrome, where the mechanistic pathways linking acute cerebrovascular events to cardiac complications remain poorly defined. Although autonomic dysfunction, systemic inflammation, and neurogenic cardiac injury are implicated, the interplay between these processes and pre-existing cardiovascular disease is not well understood. Clarifying these mechanisms would support the development of integrated neuro-cardiac care models that address both acute and chronic sequelae<sup>(6)</sup>. In addition to environmental and implementation gaps, important knowledge deficits remain in the biological and genetic determinants of cardiovascular and cerebrovascular disease.

Genetic and epigenetic factors also represent an important research gap, as Asian populations are underrepresented in cardiovascular genomics. Most genome-wide association studies (GWAS) and mechanistic research are based on Western cohorts, limiting applicability to Southeast Asia, where unique genetic variants, environmental exposures, and cultural factors may alter disease susceptibility and outcomes. Expanding precision medicine research in Asian cohorts would improve the accuracy of risk prediction and enable more personalized therapeutic strategies<sup>(8,9)</sup>. Beyond research, however, a critical challenge lies in the implementation of existing evidence into practice. In Thailand and other LMICs, uptake of clinical guidelines remains inconsistent, continuity of care between hospitals and community health systems is weak, and rehabilitation services are limited. Preventive pharmacotherapy and advanced acute care interventions, such as statins, antihypertensives, percutaneous coronary intervention, and mechanical thrombectomy, are available but remain concentrated in urban centers, leaving rural and disadvantaged populations with limited access. These gaps contribute to persistent inequities and reduce the overall impact of national prevention strategies.

Emerging opportunities such as digital health and artificial intelligence (AI) offer potential solutions but remain underdeveloped in Thailand. Telemedicine, AI-enhanced risk prediction, and mobile health applications could improve early detection, strengthen treatment adherence, and facilitate long-term follow-up in resource-limited settings. However, evidence regarding cost-effectiveness, scalability, and patient acceptance is scarce, and systematic evaluation is required before these tools can be integrated into routine practice. Moving forward, priority areas should include longitudinal cohort studies on environmental and genetic risks, deeper mechanistic research into stroke–heart interactions, and implementation science to enhance guideline adherence, continuity of care, and rehabilitation outcomes. In parallel, the development and evaluation of digital health solutions tailored to the Thai and broader LMIC context will be crucial. Addressing these gaps will not only advance scientific knowledge but also support evidence-based policymaking and improve equity in healthcare access, ultimately reducing the growing burden of CVD and stroke.

## Conclusion

Thailand continues to face a growing burden from cardiovascular disease and stroke, making it important to strengthen efforts in prevention, early diagnosis, treatment, and rehabilitation. Health policies should focus on improving access to quality care for all communities and supporting programs that reduce major risk factors. Further research is needed to better understand local genetic and environmental risks, including air pollution, and to use digital health tools to improve long-term care and disease monitoring.

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