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# ISCHEMIC STROKE IN YOUNG ADULTS: A MODERN OVERVIEW

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**ABSTRACT**

The incidence of ischemic stroke in young adults (typically defined as population under 50 years of age) is rising measurably in recent years, an estimated 10-14% of strokes worldwide. It brings severe implications for global public health and economic productivity. This review provides a comprehensive analysis of evidence regarding the changing epidemiology, heterogeneous etiology and long-term outcomes of stroke in the young age. In opposition to older people, where atherosclerosis is one of the most common causes, among young adults there's a wider variety of risk factors including cervical artery dissection, diverse heart defects, genetic blood clotting disorders and autoimmune diseases. However in a significant number of cases, the cause is difficult to diagnose and often requires advanced diagnosis beyond standard protocols. Modern lifestyle habits, such as obesity, hypertension and substance abuse are responsible for escalating stroke rates. The findings might prove themselves useful in establishing specialized care models for young adults' stroke.

**KEYWORDS**

Ischemic Stroke, Young Adults Stroke, Patent Foramen Ovale and Stroke, Stroke Treatment, Stroke, Risk Factors, Thrombophilia

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## Introduction

Ischemic stroke is a clinical and radiological syndrome that results from an abrupt reduction of cerebral blood flow due to arterial occlusion with consequent focal neurological injury and potential long term disability. [1] It's still one of the leading causes of death and long-term disability worldwide, and when it affects younger people, it brings unique challenges in both diagnosis and treatment. [2] Recent studies show that both the number and percentage of ischemic strokes in young adults have gone up in many parts of the world over the last decade. This is a serious public health and economic issue, because having a stroke at a young age often leads to many more years of disability. [3] Large meta-analyses confirm that the incidence and causes of these conditions vary a lot between different regions and demographic groups. This means that prevention and diagnostic strategies should be tailored to local risk patterns, rather than relying on a single approach designed for older populations. [4] Recent research reveals a clear increase in ischemic strokes among young adults and points out the serious personal and societal impact of having a stroke during the most productive years of life. This trend leads to greater lifelong disability and higher overall costs for medical care and rehabilitation. [5]

The term young adult is variably defined in clinical literature, but most contemporary studies and clinical reviews use an onset age range that begins in late adolescence and extends to approximately fifty and fifty five years of age in order to capture the population in which conventional age related cerebrovascular risk is expected to be lower and alternative mechanisms are relatively more frequent. [6] [2]

The causes of ischemic stroke in younger adults are heterogeneous and include both traditional vascular risks that are increasingly prevalent at earlier ages and nontraditional mechanisms such as cervical artery dissection, autoimmune and inflammatory vasculopathies, cardioembolic sources including paradoxical embolism, inherited and acquired thrombophilias, pregnancy related events, increasingly recognized contributions from atherosclerotic disease driven by adverse cardiometabolic exposures emerging at earlier ages and other rarer disorders. [7] A significant number of ischemic strokes in young adults are still labeled as cryptogenic after standard testing. Research has linked these strokes to factors such as migraine with aura, patent foramen ovale, recreational drug use, and pregnancy-related conditions. Together, these findings suggest that some patients may benefit from additional, but carefully chosen, diagnostic tests tailored to their specific clinical situation. [8] Cervical artery dissection constitutes a leading single cause of ischemic stroke among younger patients and warrants particular attention because its recognition affects both acute management and secondary prevention planning. [9]

Traditional vascular risk factors such as hypertension, diabetes, high cholesterol, obesity and smoking are still very important. Their prevalence has been increasing in younger adults, and they can interact with other, less typical factors to raise the risk of having a stroke at a younger age. [6]

Current evidence suggests that strokes in younger adults are becoming more common partly because this age group now has higher rates of obesity, problems with glucose metabolism, high blood pressure, and greater exposure to tobacco and other substances. At the same time, better neuroimaging and increased clinician awareness have led to improved detection of milder or unusual cases. These combined factors highlight the need for coordinated public health efforts and focused clinical research to identify the most effective prevention strategies for younger populations. [10]

Younger patients present specific diagnostic and management challenges. The diversity of potential etiologies requires a broader diagnostic algorithm than is commonly applied in older patients and care pathways that account for reproductive health, long term vocational and psychosocial needs, and the potential decades long risk of recurrence. Long-term outcomes after ischemic stroke in young adults include ongoing functional impairments and higher long-term mortality compared with people of the same age. Together, these findings support the need for early secondary prevention and structured follow-up that target modifiable risk factors. [11] Recent guidelines and policy reports recommend creating dedicated centers and age-specific care models to improve evaluation, speed up appropriate secondary prevention, and coordinate multidisciplinary rehabilitation and long-term follow-up. [12]

The aim of this manuscript is to give a clear and rigorous summary of current evidence on the epidemiology, main causes, and modifiable risk factors for ischemic stroke in young adults. It also seeks to propose a practical definition of the study age range and to outline recommendations for diagnostic approaches and prevention priorities based on published population and clinical data. By clarifying which mechanisms are most prevalent and which interventions are most likely to reduce lifetime disability, a focused study on young adult ischemic stroke can inform clinicians, health systems and policy makers in order to reduce the growing burden of stroke that occurs during the most productive decades of life and improve long term outcomes for younger survivors. [2] [4]

**Epidemiology of Ischemic Stroke in Young Adults**

Over the past few decades, ischemic stroke in younger adults – commonly defined as individuals under 50 years of age- has emerged as a serious public health concern. This group of affected individuals makes up about 10–14% of all stroke cases [13,14]. Data has reached this upward trend according to its steadily increasing proportional incidence. Trend pattern in this case has been recorded in higher income countries such as the U.S. [15], the Netherlands [14], France [13], and low- and middle-income countries where the development is more clear, as seen in data from India [15]. Our widening spread is predominantly among those over 35 years of age - particularly [14], which illustrates a convergence of classic vascular risk factors - hypertension, metabolic dysfunction and obesity, as well as the vascular risk factors common in younger groups, such as migraine with aura or hormonal factors. An additional relevant way of looking at stroke epidemiology in young adults is still sex − related differences. Ischemic stroke is more common in women <35 than men <35 and was reported in 19 studies from a meta-analysis with a 44% increased prevalence among this age group [16]. This difference decreases between 35 and 45 years [16]; this rising rate of classic vascular risk factors in both genders is likely to be explained. In younger women, mostly premenopausal, non-atherosclerotic and non-traditional mechanisms might be more prevalent [17]. It is important to realize this sex-biased profile to strengthen preventive treatments and clinical strategies. Other ethnic differences are influencing young adults stroke epidemiology. Population-based research showed an elevated stroke rate in young Black and Hispanic people in comparison to white [18,19], and both ischemic and bleeding stroke subtypes displayed significant disparities [19]. Access to intravenous thrombolysis and mechanical thrombectomy are integral adjunctive therapies for acute strokes, but, similar across ethnic group, stroke is correlated with poorer functional outcomes in Black or Hispanic patients [18]. Moreover, early-onset mortality, particularly at 30-days mortality is highest among Black patients [20,21]. Patients general functional recovery is significantly improved, compared with short-term mortality in older individuals. Young adults suffer from the long term effects of a disability have serious long-term impacts, including social and professional occupational and psychological burdens as well as high lifetime risk of second stroke and the following challenges and lifetime risk for disability at work-related complications, are significant for most of young adults. These findings highlight a demand for targeted interventions aimed at recognizing and early identification of modifiable risk factors, as well as sex - and ethnicity-specific risk profiles for those with modifiable risk factors and preventive strategies at a more targeted level.

**Risk Factors and Immediate Etiologies of Ischemic Stroke in the Young Adult Population**

The etiology of ischemic stroke in young adults differs substantially from that observed in older populations [22][23][24]. Whereas in individuals over 50 years of age stroke is predominantly caused by atherosclerosis, hypertension, and atrial fibrillation, the causes in younger adults are more heterogeneous [23]. These differences in etiology necessitate a different diagnostic approach; for instance, standard classifications such as TOAST may be insufficient for stroke evaluation in young adults, as they do not account for all relevant factors [23][24]. In this review, we aim to present the most common causes of stroke in the young adult population.

**Obesity and Hypertension**

The correlation between the growing obesity epidemic and the resulting hypertension is a well-documented cause of the increase in the number of ischaemic strokes [25]. Excessive body weight contributes to the development of metabolic disorders, damaging the vascular endothelium and causing inflammation, which has a detrimental effect on the vascular system.[26] In young adults, the synergistic occurrence of obesity and hypertension exacerbates the harmful effects on cerebral vessels. This accelerates atherosclerotic processes and promotes the formation of blood clots, which can lead to acute ischemic stroke. [27] A population-based case–control study published in 2015, designed with 1201 cases and 1154 controls, was conducted, providing insight into the relationship between obesity and a higher risk of ischemic stroke. The results adjusted for nicotine use, hypertension, and diabetes did not allow obesity to be isolated as an independent risk factor for stroke [28].]

**Diabetes mellitus**

Another factor known to lead to negative changes in the vascular endothelium is diabetes mellitus. Because of its direct effect on the vascular system, the risk of cerebral vascular events in the course of this disease is evident. Chronic hyperglycaemia, often accompanying patients with diabetes, contributes to the intensification of atherosclerotic processes, thereby impairing coagulation processes, which promotes the formation of blood clots and narrowing of the cerebral vessels. Patients diagnosed with diabetes mellitus have a 2-6 times higher risk of cerebrovascular complications.[22]

**Illicit substances**

The use of illicit psychoactive substances is increasing among young adults. The cause of ischaemic stroke in young people often remains unknown, and some cases are not related to the factors listed above. Substances that may predispose to this condition include cannabis, cocaine, methamphetamine, heroin, and androgenic anabolic steroids[30]. It has been proven conclusively the use of illegal intoxicating substances contributes to a 6.5 times higher risk of stroke than in non-users of such substances [30, 31].

The causes of ischaemic stroke after use of illicit substances such as cocaine are most often atherosclerosis of large intracranial vessels, cardiac arrhythmias caused by blockage of the cardiac conduction system. In contrast, the effect of androgenic anabolic steroids is closely related to anabolism, which affects cardiac tissue growth, leading to hypertrophic cardiomyopathy [30]. In summary, the pathophysiology of drugs affects the structure and function of cardiac muscle tissue and blood vessels, including cerebrovascular vessels.

**Alcohol**

An interesting relationship appears to exist between alcohol consumption and the risk of experiencing an ischaemic stroke. Some sources indicate that consuming small amounts of alcohol may reduce the risk of this event occurring. However, regular consumption of large amounts of alcohol results in an increased risk [32]. The differences in the level of risk have been proven to occur not solely at the level of alcohol consumption, but also in terms of the risk associated with the gender of the alcohol consumer. A recent study compared a group of men and women who excessively consumed alcohol. The results surprisingly demonstrated that the risk of cryptogenic ischaemic stroke was significantly higher in men, but not in women [33].

**Cardioembolism**

According to the literature, 15–30% of all ischaemic strokes may be of cardiac origin. Determining whether a stroke has a cardiogenic cause is a major diagnostic challenge requiring comprehensive assessment, including electrocardiography, echocardiography, brain imaging and heart monitoring [34]. A multicenter hospital-based observational study published in 2022, focused on identifying the vascular causes of ischaemic stroke in young adults. The study helped to establish a list of sources of embolism, which can be listed as follows: dilated cardiomyopathy, arterial myxoma, patent foramen ovale and atrial septal defect. The sources listed are factors contributing to cardioembolism more frequently in young adults than in older individuals, but they are relatively less numerous [35].

**Cryptogenic Stroke**

A study conducted in the Netherlands on 1,322 patients aged 18–49 years reported that 25.2% of patients had a cryptogenic stroke, where no cause of embolism could be identified despite comprehensive diagnostics [36]. Other sources report rates of 30–40% [37][38]. Several studies have attempted to identify the main factors contributing to cryptogenic stroke.

The best known predisposing factor is a patent foramen ovale (PFO). PFO presents as a small opening between the right and left atria present physiologically during fetal life. In approximately 25–30% of the general population, the PFO fails to close completely and usually remains asymptomatic [39][40]. Amid patients with cryptogenic stroke, approximately 35–40% of them preset with PFO [41]. With particular stroke risk are associated large PFOs with significant right-to-left shunting [41]. Three main mechanisms are behind the PFO-related stroke: 1) paradoxical embolism, in which a thrombus from the venous circulation passes into the arterial circulation, 2) in situ thrombosis within the PFO, and 3) PFO-related arrhythmia [42].

It is estimated that 45% of cryptogenic strokes do not fit either the vascular model (stroke caused by vessel disease such as atherosclerosis or vasculopathies) or the cardiac model (atrial fibrillation, PFO) [43], suggesting the presence of other risk factors. Evidence suggests that deficiencies in proteins S and C may contribute to cryptogenic stroke, especially in young adults and middle-agaleed women [44][45]; however, there's no confirmation of those findings in controlled clinical trials [46][47].

**Vasculopathies**

Vasculopathies fundamentally lead to stenosis, occlusion, or embolism in the vessels supplying blood to the brain.

**Moyamoya Disease** is a disease leading to progressive stenosis of intracranial arteries, mainly affecting the terminal segments of the internal carotid arteries. The result is characteristic collateral networks resembling a "puff of smoke" [48]. Moyamoya occurs more frequently in women and is most commonly diagnosed around the age of 39 [49]. Clinical data indicate that approximately 20.4% of patients with moyamoya disease experienced ischemic stroke [50].

**Cervical and Vertebral Artery Dissection**, or separation of the layers of the arterial wall, is a common cause of stroke in young adults, accounting for approximately 8% of cases [20][21]. The mechanism involves arterio-arterial embolism, where clot formed within the vessel wall dislodge and fly into cerebral arteries, causing ischemia [51]. Risk factors for dissection include migraine with aura, infections, neck trauma, smoking [52], and connective tissue disorders (e.g., Marfan syndrome, Ehlers-Danlos syndrome) [53]. The analysis also showed that patients with dissection were less likely to have hypertension, and their hospitalisation lasted significantly longer than in the group without dissection. The authors emphasise that vascular dissection is a significant cause of stroke in young adults, especially when typical risk factors are not present [52].

**Inflammatory Vasculopathies** may result from infectious agents (HIV, VZV, syphilis) or autoimmune diseases such as systemic lupus erythematosus (SLE). Infectious factors can trigger coagulation and intensify endothelial dysfunction, promoting a hypercoagulable state. They may also directly affect the vessel wall by inducing smooth muscle proliferation and cytokine release [54]. In SLE, type I interferons reduce nitric oxide synthase activity, weakening vasodilation and promoting endothelial activation. Interferons also stimulate IL-18 production via the NLRP3 inflammasome, leading to inflammation and endothelial dysfunction [55]. Vascular walls may also be damaged by immune complex deposition, which activates complement and recruits inflammatory cells [56].

Other, less common vasculopathies include genetic small-vessel diseases (e.g., CADASIL, CARASIL) and vascular malformations.

**Thrombophilias**

The most common thrombophilia connected with ischemic stroke is antiphospholipid syndrome (APS). Diagnosis is confirmed by the occurrence of an arterial or venous thrombotic event or pregnancy loss in the presence of antiphospholipid antibodies [57]. The main antibodies include lupus anticoagulant, anticardiolipin antibodies, and anti-β2 glycoprotein I antibodies [58]. Antibody positivity must be confirmed at least 12 weeks apart [57]. Among thrombotic events in APS, it's ischemic stroke that is the most frequent and serious arterial complication [59]. Studies suggest that in population under 45 years of age, over 20% of strokes are associated with APS [60]. Patients with triple-positive antibodies or a positive lupus anticoagulant test are at highest risk of thrombotic events [61][62].

The precise mechanisms underlying thrombosis in APS are not fully elucidated but may involve disruption of the annexin shield, allowing antiphospholipid antibodies to damage the endothelium, platelet activation, release of adhesion molecules and tissue factor, and complement activation [63][64].

Other thrombophilias, such as protein C deficiency or hyperhomocysteinemia, have not shown significant differences in young adults with ischemic stroke compared to controls [65][66][67]. Some studies report a higher prevalence of protein S deficiency [68][69]; however, due to limited evidence, APS remains the most important and prevalent thrombophilia in this population.

**Diagnosis, Management, and Prevention of Ischemic Stroke**

**Imaging Diagnostics**

All young patients with stroke require non-contrast computed tomography (CT) to exclude primary intracranial hemorrhage, which is one of the main contraindications to thrombolytic treatment [70]. Magnetic resonance imaging (MRI) is not required for the clinical diagnosis of ischemic stroke; however, it may be helpful in determining the etiology of the stroke and differentiating it from other conditions. MRI is the imaging modality of choice for the posterior fossa and skull base. Due to its high resolution, MRI also better visualizes small lacunar infarcts.[71,72], In young adults, CT angiography (CTA) is also recommended because of the relatively high prevalence of cerebral and cervical artery dissection. [70]

**Cardiological Diagnostics**

Unlike in older populations—where atrial fibrillation is one of the leading causes of ischemic stroke—cardiac arrhythmias are less common in younger individuals [70]. Nevertheless, a standard 12-lead electrocardiogram is mandatory [70,71]. Transthoracic echocardiography (TTE) with bubble contrast is also recommended to exclude potential causes such as thrombi in the left atrium and left ventricle, infective endocarditis, non-infective endocarditis, valvular heart disease, or persistent foramen ovale (PFO) [71]. Persistent foramen ovale is associated with ischemic stroke, but management in young adults with cryptogenic stroke and PFO remains controversial.[72] In such cases, transesophageal echocardiography (TEE) is additionally recommended. [70,71].

**Acute Treatment**

Acute management is carried out during the first hours after the onset of ischemic stroke and aims to minimize brain injury. It consists of two primary strategies: Intravenous thrombolysis with alteplase (rtPA) administered within a 4.5-hour therapeutic window is recommended for young adults with ischemic stroke in the absence of contraindications. The high effectiveness of this method supports its use, provided that the drug is administered as early as possible. Mechanical thrombectomy, performed within 6 hours, is indicated in patients with large-vessel occlusion. It is associated with lower mortality compared with older adults due to the lower prevalence of comorbidities in this age group [70, 73]. Supportive treatment includes careful control of blood pressure, glycemia, and body temperature. [70]

**Prevention**

Ischemic stroke is associated with up to a fourfold increase in mortality in adults compared with healthy individuals [74]. Stroke in young patients has serious long-term socioeconomic consequences.[74, 75] A comprehensive and interdisciplinary approach is necessary to reduce the risk of primary and secondary events. The main strategies to reduce stroke-related morbidity and mortality include modification of risk factors and lifestyle changes. There are no specific guidelines dedicated to primary and secondary stroke prevention in young adults [75].Intensive blood pressure control, optimal glycemic regulation, and dietary modifications are beneficial for this population. Smoking cessation should be strongly encouraged.[74,75,76]. The Mediterranean and DASH diets are recommended to promote weight reduction and improve glycemic control[72,74,75,77]. Reducing sodium intake to less than 1500 mg per day further lowers stroke risk[74,75,77]. Lifestyle changes—including smoking cessation and reduction of alcohol consumption—significantly decrease the risk of ischemic stroke [76]. High levels of physical activity markedly reduce stroke risk compared with light or moderate activity [75,76]. The mechanisms through which exercise decreases stroke risk are multifactorial and include lowering blood pressure, reducing body mass index (BMI), improving lipid profiles, and enhancing glycemic control [77]. Hypertension is one of the leading and most modifiable risk factors for stroke. Studies have demonstrated that individuals with hypertension benefit the most from blood pressure reduction. Treatment should be individualized, with a target blood pressure ≤140/90 mmHg in patients without diabetes and ≤130/80 mmHg in patients with diabetes [72,74,75,77]. The risk of stroke is two- to six fold higher in patients with diabetes [72]. However, there is no clear consensus on whether tighter glycemic control effectively prevents macrovascular complications such as stroke [72,73]. Therefore, diabetes management should be individualized [73]. In stroke patients with atherosclerotic disease and high 10-year cardiovascular risk, statin therapy is recommended [74].

**Long-Term Implications and Quality of Life Assessment**

The long-term follow-up studies show that even if the initial neurological deficit after an ischaemic stroke seems to be minor, it is in fact a serious event. In large cohort and registry studies, researchers have shown that young stroke survivors continue to face important risks later in life: increased long-term mortality, an ongoing risk of recurrent vascular events, and lasting functional limitations that can affect independence and daily activities [78,79,80,81,82]. Depending on the cohort and length of follow-up, the annual rates of recurrent stroke or combined major vascular events typically range from about 1% to 3% [79,80,81,82]. Only around a half of young survivors remain alive, free from new vascular events or significant disability after several years of follow-up [79,82]. In the Iowa Registry, for instance, only less than a half of patients were alive and were not disabled after an average follow-up of six years. Numerous young survivors report that long after the stroke they still struggle with physical, emotional or social difficulties that limit their ability to work and take part in everyday life [82]. Similar patterns observed in European and international cohorts show that, even though young adults generally have better survival and functional scores than older patients, they often live for many years with a substantial burden of stroke-related health problems [78,79,80,81]. In patients with additional vascular events especially, cognitive impairment, fatigue and mood disturbances are frequently described and may persist or even progress long after the acute phase [81,83,84,85,86]. In some cohorts, fear of stroke recurrence also is also valid as an important long-term concern [87,88].

Hence, the Health-related quality of life (HRQoL) has become an important additional outcome to traditional measures such as the modified Rankin Scale (mRS) or Barthel Index. Studies using generic instruments (SF-36, EQ-5D) and stroke-specific tools (SS-QoL, SIS, SIP) indicate that young stroke survivors report noticeably reduced HRQoL. In those cohorts that include population controls their scores are clearly lower than in age-matched individuals from the general population [84,85,86,88,89]. The most affected domains are physical functioning and role limitations caused by physical problems. Moreover, reduced vitality and energy, emotional functioning, social participation and work/productivity are also commonly reported

[84,85,86,89,90]. In a Norwegian population-based cohort, young adults reported significantly lower SF-36 scores in physical and social functioning several years after stroke, even though their neurological deficits were relatively mild [84]. Similarly, in an Estonian study, young stroke survivors had clearly lower EQ-5D utility scores than their counterparts in the general population (0.71 vs. 0.87). The greatest differences were observed in mobility, self-care and usual activities [86]. Data from low- and middle-income settings, such as Kenya, point to even more pronounced problems in physical and social functioning, underlining how medical, social and economic constraints together shape everyday life after stroke [85,89]. Importantly, several studies show that a subgroup of patients with excellent functional outcome (mRS 0–1) can achieve HRQoL that is almost as high, or as high as patients who did not experience stroke at all [85,86]. It has been suggested that, in some individuals, good adaptation, effective coping strategies and personal resilience can partly outweigh the impact of residual symptoms [85,87,88]. A broad set of factors influences HRQoL in young stroke survivors, including clinical, psychological and social aspects. Across observational studies and multivariable models, poorer functional status (higher mRS scores, dependence in daily activities, motor or hand problems, communication difficulties like aphasia or dysarthria), consistently emerges as one of the strongest clinical contributors to reduced HRQoL [84–88,90].

In addition, coronary heart disease at the time of the event, longer time since stroke, recurrent stroke, post-stroke epilepsy and a higher burden of pain or discomfort have been associated with lower HRQoL in some cohorts [85,96,89,90]. Psychological factors are equally influential. For instance, post-stroke depression, anxiety, fatigue, fear of stroke recurrence, low self-perceived health and weaker sense of coherence have all been identified as key determinants of low HRQoL [84,86,87,88,90]. Socioeconomic and vocational circumstances further modify outcomes. Inability to return to work, unemployment, lower educational attainment and worse economic status repeatedly predict lower HRQoL scores. On the other hand, successful work reintegration and stronger social support correlate with better subjective outcomes [85-89]. Age-stratified analyses suggest that, at a similar level of physical disability, younger adults may report better HRQoL than older stroke survivors [85,90]. Nevertheless, in the young age group physical limitations and communication problems appear to have a particularly strong negative impact on mood, social roles, and family and financial responsibilities [87,90]. Two recent reviews underline that existing evidence is still heterogeneous with respect to age cut-offs, stroke subtypes and outcome measures, and that longitudinal data on trajectories of HRQoL and resilience over time remain limited [85,87].

All things considered, the available studies indicate that long-term follow-up of young ischemic stroke survivors should routinely include standardized assessment of HRQoL and psychosocial status, not only neurological and vascular outcomes [84-89]. The consistent association of functional status, mood disorders, fatigue and employment with HRQoL supports comprehensive, multidisciplinary care models. They combine intensive motor rehabilitation with systematic screening and treatment of depression and anxiety, management of fatigue and pain, and structured vocational counselling [84,86-90]. In addition, coronary heart disease, recurrent stroke and longer time since the index event have been identified as independent predictors of lower HRQoL in long-term cohorts [86,87]. These factors are also well-established determinants of mortality and recurrent vascular events in young stroke populations [78-81]. Together, they emphasize the need for strict secondary prevention and long-term vascular risk control in this group. Future research should focus on prospective, age-specific cohorts to define HRQoL patterns and clarify the role of resilience and coping strategies. Apart from that, they should also test targeted interventions that may improve long-term subjective and objective outcomes in young adults living with the consequences of ischemic stroke [85-87].

**Discussion**

Young-onset ischemic stroke is emerging as a clearly distinct clinical and public health challenge. The evidence summarized in this review shows that its epidemiology, causes, diagnostic needs, and long-term consequences differ in important ways from those seen in older adults. Global data confirm a steady rise in stroke incidence among people from late adolescence to around fifty years of age, a pattern that contrasts with the stabilization or decline observed in older groups. Because these strokes occur during the most socially and professionally active years of life, their impact is magnified at both the individual and societal levels [91].

The findings presented here highlight that ischemic stroke in young adults arises from a particularly diverse set of mechanisms. The growing prevalence of modifiable vascular risks combines with age-specific causes—such as cervical artery dissection, cardioembolic sources, and inherited or acquired prothrombotic conditions—to create diagnostic challenges that differ markedly from those in older adults. The need for

individualized evaluation is underscored by the fact that a substantial proportion of cases in young adults remain cryptogenic, even after comprehensive imaging and testing [92].

Effective management depends on precise and timely diagnosis supported by modern neuroimaging, detailed vascular and cardiac evaluation, and selective laboratory testing. The evidence reviewed shows that improving early recognition of stroke symptoms in younger people remains essential, as diagnostic delays contribute significantly to long-term disability in this age group. Strengthening acute treatment pathways and secondary prevention therefore requires clinical expertise, system-level readiness, and ongoing public education efforts [93].

The long-term consequences of young-onset ischemic stroke extend well beyond the immediate neurological deficits. Many younger survivors continue to face lasting functional limitations, increased long-term mortality compared with peers, and a substantial lifetime risk of recurrence. These realities point to the importance of coordinated, multidisciplinary care models that address both medical and psychosocial needs. Such approaches should include strict management of modifiable vascular risks, as well as comprehensive cognitive, psychological, and vocational rehabilitation tailored to individuals who aim to maintain or return to education, work, and family life [94].

In this context, the original goal of this manuscript—to synthesize contemporary evidence on epidemiology, causes, diagnostic strategies, and prevention—remains central for guiding future research and informing clinical practice. The increasing burden of early-onset stroke highlights the need for sustained investment in age-appropriate prevention programs, improved risk prediction tools that reflect the specific pathophysiology of younger patients, and long-term studies that can clarify mechanisms of recurrence and chronic decline [95].

Overall, young-onset ischemic stroke should be recognized as an expanding and urgent health concern with wide-reaching medical, social, and economic implications. Achieving better outcomes for younger patients will require coordinated international efforts to enhance surveillance, modernize diagnostic approaches, develop individualized prevention strategies, and strengthen rehabilitation systems throughout the entire continuum of care. Addressing this challenge is essential for reducing lifetime disability and safeguarding the health and productivity of a generation increasingly affected by a condition once considered rare in early adulthood [96].

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