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Review Article

Etiologies and Risk Factors of Ischemic Stroke in Young Adults: A Comprehensive Approach to Diagnosis and Management

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Abstract

Ischemic stroke in young adults has become a significant clinical concern, as its incidence has been increasing in recent years. Although traditional cardiovascular risk factors (CVRF) such as smoking, hypertension, and dyslipidemia are commonly seen in this population, there are also other specific etiologies, including congenital conditions like patent foramen ovale (PFO), cervicocephalic arterial dissection, genetic diseases, inflammatory and non-inflammatory vasculopathies, and the use of illicit drugs. A thorough understanding of the diverse etiologies of ischemic stroke in young adults is essential for accurate diagnosis and effective management. This article reviews the various causes of ischemic stroke in the young adult population, with an emphasis on the underlying cardiovascular risk factors and congenital conditions. A significant proportion of strokes in this population are attributed to PFO, a congenital anomaly that can increase the risk of ischemic stroke. Cervicocephalic arterial dissection, particularly following minor trauma, is another key cause of stroke in young individuals. Additionally, genetic diseases, inflammatory vasculopathies, and conditions such as thrombophilia can contribute to the occurrence of ischemic strokes. Moreover, illicit drug use and lifestyle factors, such as excessive alcohol consumption and physical inactivity, play a critical role in the rise of ischemic stroke cases in young people. By exploring these causes, this article presents an overview of the diagnostic approach and therapeutic strategies, underscoring the need for early identification and tailored interventions. Prevention, through lifestyle modification and timely interventions for those at high risk, is crucial to reduce the burden of ischemic stroke in this age group.

Keywords: *Risk Factors of Ischemic Stroke; Young Adults; Cervicocephalic Arterial Dissection*

Introduction

The incidence of ischemic stroke in young adults has risen significantly in recent years, prompting healthcare professionals to recognize the importance of studying this demographic. The adult population considered "young" for the purposes of stroke research typically ranges from 18 to 50 years of age. While stroke incidence generally increases with age, the prevalence of ischemic stroke in young adults has seen a marked rise, attributed to the growing number of cardiovascular risk factors (CVRF) prevalent in this population. Factors such as smoking, physical inactivity, hypertension, dyslipidemia, and obesity are increasingly common among young adults, leading to a higher risk of stroke in this group.

However, there are unique etiological factors at play in young adults who experience ischemic stroke, differentiating them from older populations.

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For example, conditions such as patent foramen ovale (PFO), cervicocephalic arterial dissection, and genetic predispositions play a more significant role in this age group. These etiologies not only complicate diagnosis but also require specific management strategies, making the recognition of risk factors and timely intervention all the more important. Furthermore, lifestyle factors, including illicit drug use and sedentary behavior, have become more pronounced as contributors to the rising rates of ischemic stroke in young adults.

In addition to the established cardiovascular risk factors, ischemic stroke in young adults can also be caused by less common factors, such as genetic diseases and inflammatory vasculopathies, which present unique diagnostic challenges. These conditions underscore the importance of considering a wide range of potential causes when evaluating young patients presenting with ischemic stroke. Understanding these multifactorial causes is essential for developing effective prevention, diagnosis, and treatment strategies for this vulnerable group.

1. Cardiovascular risk factors

Numerous studies have highlighted an increase in the prevalence of cardiovascular risk factors (CVRF) in young patients with ischemic stroke. These factors are of utmost importance, not only due to their association with this pathology and their high frequency in patients who suffer from it, but also because of their potential in the primary and secondary prevention of this population.

According to the Stroke in Young Fabry Patients (SIFAP1) study, which included 4,467 European patients aged 18 to 55, the most common cardiovascular risk factors were smoking (55.5%), physical inactivity (48.2%), hypertension (46.6%), dyslipidemia (34.9%), and obesity (22.3%). On the other hand, abdominal obesity was present in up to 64% of the studied population, being more prevalent in women, especially from the age of 25 [1].

The relationship between these factors and the risk of stroke in young people is particularly notable. Smoking, for example, is associated with a significant increase in risk, with reported odds ratios (OR) ranging from 1.6 to 7.7 in different cohorts. Hypertension also stands out as an important risk factor, with OR ranging from 1.6 to 8.9 in case-control studies conducted on young adults [2]. Despite the known associations with other mentioned CVRFs, there is limited data on their strength of association with stroke in young adults.

These factors showed a progressive increase with age, being significantly more frequent in men, except for abdominal obesity and physical inactivity, which were more common in women [2]. Furthermore, obesity, particularly abdominal obesity, is a significant factor in the young population, with an impact largely mediated by hypertension and diabetes [3]. On the other hand, diabetes mellitus was present in 10.3% of patients, also with higher prevalence in men and older age groups [1].

Some less-established risk factors also play an important role in stroke in young people. High-risk alcohol consumption, defined as regular intake of large quantities, was present in 33% of the patients studied in SIFAP1, with higher prevalence in men. Inadequate sleep patterns, such as short nighttime duration (≤6 hours), were also identified, with a prevalence of 17.9% in this population. These factors further contribute to the risk of cerebrovascular events [1].

The high prevalence of modifiable cardiovascular risk factors in young patients with stroke emphasizes the need for preventive measures to be implemented from an early age. Interventions targeting smoking cessation, promoting physical activity, treating hypertension and dyslipidemia, as well as strategies to address obesity, could have a significant impact on reducing the stroke burden in this population.

2. Patent foramen ovale

The patent foramen ovale (PFO) is a congenital cardiac anomaly caused by the failure of the fetal interatrial communication to close and represents the most common cause of right-to-left circulatory shunt in adults. Its prevalence in the general population is estimated to be around 25% [4]. This defect has been postulated as one of the main causes of cryptogenic ischemic stroke in young patients, as its prevalence in this group increases to 40-50% [5].

Although the mechanisms and characteristics associated with PFO that increase the risk of ischemic stroke are still being studied today, paradoxical venous embolism has been proposed as the main pathophysiological mechanism, as an increased risk is observed in patients who present both factors.

It is postulated that thrombi formed in the venous system could pass through the foramen ovale, bypassing the pulmonary circulation, and reaching the cerebral vascular bed, causing ischemic stroke [6]. On the other hand, there are certain characteristics associated with PFO that may increase this risk, such as a larger foramen size, which can be confirmed by a higher bubble passage during the bubble test, the presence of interatrial septal aneurysms, the Eustachian valve, or the Chiari network [6-7].

To individually study the risk of recurrent ischemic stroke associated with PFO in each patient, several scales have been created to identify those patients who would benefit from percutaneous closure of the PFO. The Risk of Paradoxical Embolism (RoPE) scale, which includes criteria such as age, history of hypertension, diabetes, smoking, previous stroke or TIA, and cortical infarction on brain imaging [8]. A score between 0-6 (those patients with older age and cardiovascular risk factors) is associated with a lower risk of recurrence and an etiological link to PFO, while a score between 7-10 (corresponding to younger patients) is associated with a higher recurrence rate and a causal relationship between stroke and PFO [9]. It is in this latter population group where current studies show the most indications for performing percutaneous PFO closure.

In conclusion, PFO is a common congenital anomaly that may contribute to ischemic stroke in young patients through paradoxical embolism, particularly when combined with other risk factors. The presence of larger PFOs and additional structural features like septal aneurysms or the Chiari network further elevate stroke risk. Screening for PFO, especially in cryptogenic stroke cases, is essential to guide diagnosis and inform treatment decisions.

3. Cervicocephalic arterial dissection

Cervicocephalic arterial dissections are a significant cause of stroke, particularly in young individuals. These injuries occur when the layers of the arterial wall separate, creating a false lumen where blood accumulates. This process can lead to either occlusive stenosis or dissecting aneurysms, depending on whether the bleeding occurs beneath the intimal or adventitial layer of the vessel.

Regarding their location, extracranial carotid dissections are typically found two centimeters or more beyond the carotid bifurcation, usually near the skull base. In contrast, intracranial carotid dissections most commonly occur in the supraclinoid segment. In the vertebral artery, dissections frequently involve the V2 segment, which passes through the transverse processes of the cervical vertebrae from C6 to C2, or the V3 segment, which extends from the transverse process of C2 to the foramen magnum at the skull base [10].

The underlying pathophysiology begins with an intimal tear or damage to the vasa vasorum, allowing blood to infiltrate the layers of the arterial wall. Subintimal dissections tend to narrow or occlude the vessel lumen, while subadventitial dissections are more likely to lead to dissecting aneurysms. These structural changes predispose the vessel to ischemic or hemorrhagic events [11].

The etiology of these dissections involves a combination of intrinsic factors, such as structural weakness of the arterial walls, and extrinsic factors, such as minor trauma. Over time, numerous risk factors and triggering activities have been identified, although the presentation of dissections can vary widely [11,12].

From an epidemiological perspective, dissections of cervical and cerebral arteries occur in approximately three out of every 100,000 individuals across all ages. However, they account for up to a quarter of all strokes in young people, highlighting their clinical significance in this population [12].

Clinically, dissections often present with ischemic symptoms, such as strokes or transient ischemic attacks (TIA), frequently preceded or accompanied by local signs such as neck pain, headache, Horner syndrome, or cranial neuropathies. In rarer cases, intracranial dissections can lead to subarachnoid hemorrhage. However, asymptomatic cases or those presenting only with local symptoms may be underreported in epidemiological studies [13].

The diagnosis of arterial dissections relies on the urgent acquisition of multimodal imaging. Magnetic resonance imaging (MRI) combined with magnetic resonance angiography (MRA) of the head and neck, or computed tomography (CT) with computed tomography angiography (CTA), are commonly used.

These tools help identify characteristic findings, such as long and tapered arterial stenosis, occlusion with a tapered appearance, dissecting aneurysms (pseudoaneurysms), intimal flaps, double lumens, or intramural hematomas [14]. These findings not only confirm the diagnosis but also guide subsequent therapeutic decisions.

In conclusion, cervicocephalic arterial dissection is a critical cause of stroke in young individuals, and its diagnosis requires a thorough clinical and imaging-based approach. Early identification of these events can significantly impact patient outcomes.

4. Genetic Diseases.

Like other diseases, the increased risk of ischemic stroke can be inherited in the context of systemic genetic diseases or those primarily affecting the nervous system [15]. It is estimated that monogenic etiology is behind 1-5% of cases, although it is suspected that this value is underestimated, likely due to genetic and phenotypic variability and challenges in obtaining a definitive diagnosis. In these cases, it is important to consider family history. In this regard, the following table (Table 1) lists the main syndromes or genetic diseases associated with ischemic stroke, whether as a primary manifestation or associated with and recognized as the result of the genetic alteration [16].

Table 1. Monogenic syndromes with ischemic stroke as the main manifestation

Disorder and inheritance pattern	Gene	Chromosome	Stroke features	Other clinical features
Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (C ADASIL). AD	NOTCH3	19p13.12	Mostly recurrent lacunar ischemic. Rarely hemorrhagic.	Migraine with aura; cognitive decline; mood disorders; seizures
Cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL). AR	HTRA1	10q26.13	Mostly lacunar ischemic. Rarely hemorrhagic	Similar to CADASIL; in addition, premature alopecia, back pain, spondylosis
COL4A1 disorders. AD.	COL4A1	13q34	Recurrent lacunar ischemic or hemorrhagic	Cerebral aneurysms; porencephaly; retinal vascular tortuosity; kidney disease; muscle cramps
Moya-Moya disease. AD or AR	ACTA2, MTCP1, RNF213	Several, including X	Ischemic or hemorrhagic	Headaches; neurologic events triggered by hypoperfusion;
Monogenic syndromes with ischemic stroke as the main manifestation				
				telangiectasia; intellectual disability; seizures; bilateral stenosis or occlusion of terminal portion of internal carotid arteries and/or proximal portions of anterior and middle cerebral arteries
Retinal vasculopathy with cerebral leukodystrophy (RCVL). AD.	TREX1	3p21.31	Ischemic	Visual loss; cognitive problems; mild kidney or liver dysfunction; retinal microangiopathy

AD—autosomal dominant; AR– autosomal recessive.

Table modified and extracted from: Putaala J. Putaala, Jukka. «Ischemic Stroke in Young Adults». CONTINUUM: Lifelong Learning in Neurology 26, n.o 2 (abril de 2020): 386.

Table 2. Monogenic syndromes with ischemic stroke as a recognized manifestation

Disorder and inheritance pattern	Gene	Chromosome	Stroke features	Other clinical features
Fabry disease. X-linked recessive.	GLA	X	Ischemic or hemorrhagic	Acroparesthesia, angiokeratomas, corneal opacity, hypohidrosis, renal and/or cardiac disease.
Familial hemiplegic migraine. AD.	CACNA1A , ATP1A2, SCAN1	Several	Ischemic	Migraine with aura with motor impairment.
Homocystinuria. AR.	MTHFR, MTR, MTRR, MMADHC	Several	Ischemic	Poor growth in childhood, neuropsychiatric deficits, myopia, lens dislocation, osteoporosis, cardiovascular disease, thromboembolic events.
Marfan syndrome. AD.	FBN1	15q21.1	Ischemic or hemorrhagic	Lens dislocation, cataract, myopia, aortic aneurysm/ dissection, carotid artery dissection, cerebral aneurysms, arthritis, tall habitus, pectus excavatum, dural ectasia.
Mitochondrial myopathy, encephalopathy, lactic	Transfer RNA-Leu most	Mitochondrial DNA	Stroke like episodes	Muscle weakness, migraine like headache, seizures, short stature,
Neurofibromatosis type 1. AD.	NF1	17q11.2	Ischemic or hemorrhagic	Neurofibromas, café au lait spots, optic glioma, freckling, Lisch nodules, learning disabilities, seizures, cerebral aneurysms, autism, hypertension, short stature, macrocephaly, skeletal abnormalities, moyamoya syndrome.
Pseudoxanthoma elasticum. AR	ABCC6	16p13.1	Ischemic or hemorrhagic	Reduced vision, retinal angioid streaks, yellowish papules in flexor areas, elastic skin, claudication, gastrointestinal, bleeding, arterial dissection.
Sickle cell disease. AR.	HBB	11p15.5	Ischemic or hemorrhagic	Anemia, pain episodes, infections, lung/kidney/ spleen manifestations, moyamoya syndrome.
Vascular Ehlers-Danlos syndrome. AD.	COL3A1	2q31	Ischemic or hemorrhagic	Joint hypermobility, subluxation, and pain, cerebral aneurysm, dissection, short stature, bruises, intestinal and uterine fragility.

Table modified and extracted from: Putaala J. Putaala, Jukka. «Ischemic Stroke in Young Adults». CONTINUUM: Lifelong Learning in Neurology 26, n.o 2 (abril de 2020): 386.

5. Inflammatory and Non-inflammatory Vasculopathy

This text provides an overview of infectious and inflammatory vasculopathies associated with stroke, particularly in younger patients, emphasizing their diverse etiologies, clinical features, and diagnostic approaches. Here's a summary with key points:

1. Infectious Vasculitis and Vasculopathies [17]:
   - ♦ Associated with infections like HIV, varicella-zoster virus (VZV), and syphilis.
   - ♦ Stroke can arise due to:
     - Local inflammation (e.g., cerebral parenchyma or meninges).
     - Systemic inflammation (leading to coagulation and endothelial dysfunction).
     - Direct invasion of the arterial wall by pathogens, resulting in smooth muscle proliferation and cytokine release.

2. Non-Infectious and Inflammatory Vasculopathy [18]:
   - ♦ Includes conditions like Cogan syndrome, Susac syndrome, Sneddon syndrome, and Eales disease.
   - ♦ Often presents with unusual stroke patterns, locations, or accompanying symptoms.

3. Isolated CNS Vasculitis:
   - ♦ A progressive disease involving stroke, encephalopathy, and seizures.
   - ♦ Requires a high index of suspicion for accurate diagnosis and management.
4. Other Vasculopathies:
   - ♦ Moyamoya syndrome/disease: Chronic progressive stenosis of intracranial vessels.
   - ♦ Reversible cerebral vasoconstriction syndrome (RCVS): Transient vasoconstriction of cerebral arteries.
5. Key Syndromes:
   - ♦ Susac syndrome:
     - Affects small arterioles in the cochlea, retina, and cerebrum.
     - Symptoms: Encephalopathy, hearing loss, vision loss.
     - Corpus callosum infarcts are a characteristic finding.
   - ♦ Sneddon syndrome:
     - Features include livedo reticularis (lacy rash), seizures, renal involvement, cognitive impairment, and stroke.
6. Diagnostic Approach:
   - ♦ Essential for accurate diagnosis:
     - Cerebrospinal fluid (CSF) analysis.
     - Angiographic imaging.
     - Additional tests: Autoimmune work-up, possibly brain biopsy.
   - ♦ These methods guide appropriate immunosuppressive therapy when necessary.

7. Clinical Importance:
    - Early identification of vasculopathies is crucial, especially in younger stroke patients with atypical presentations.
    - Comprehensive evaluation can prevent misdiagnosis and enable timely treatment.

6. Inherited and Acquired Thrombophilia

Thrombophilia, characterized by an increased tendency to form blood clots, is a significant factor in certain strokes, particularly in young individuals under 40 years old. It may be inherited or acquired, with both types playing distinct roles in arterial and venous thrombosis.

Inherited thrombophilias, such as Factor V Leiden and prothrombin 20210 mutation, are the most common genetic risk factors. These mutations are more prevalent in younger stroke populations and are associated with venous thromboembolism. Their role in arterial strokes, however, remains controversial, as studies often show weak or no associations in older populations, where traditional risk factors like hypertension and hyperlipidemia dominate [19-21].

Acquired thrombophilias include conditions such as antiphospholipid antibody syndrome and hyperhomocysteinemia, which have stronger links to arterial thrombosis and stroke. Antiphospholipid syndrome, associated with recurrent thrombosis and pregnancy loss, contributes to cerebral infarctions and unique conditions like Sneddon's syndrome (stroke with livedo reticularis). Hyperhomocysteinemia, linked to deficiencies in vitamins B6, B12, or folic acid, is a major risk factor for vascular occlusion and may be mitigated with appropriate supplementation [19].

The use of oral contraceptives amplifies thrombotic risk, particularly in individuals with genetic predispositions like Factor V Leiden or prothrombin mutations. This effect is particularly pronounced in young women and emphasizes the importance of targeted screening in high-risk groups [20].

Diagnostic strategies focus on identifying thrombophilic conditions in younger stroke patients or those with recurrent thrombosis, unusual thrombotic locations, or family histories of thrombosis. Screening includes tests for genetic mutations, antiphospholipid antibodies, and homocysteine levels. However, routine testing in older patients is generally not recommended due to the low diagnostic yield [19].

The article highlights the need for personalized approaches in managing thrombophilia-related strokes. While anticoagulation therapies like warfarin are effective in many cases, the choice of treatment depends on the underlying condition, thrombotic history, and risk factors. The management of arterial thrombosis remains less clear, necessitating further research into the long-term outcomes and optimal treatment strategies [20,21].

In conclusion, thrombophilia is a crucial consideration in unexplained strokes, particularly in young patients. Early identification and tailored interventions can significantly improve outcomes, underscoring the need for further studies to refine screening and treatment protocols.

7. Neoplasms / Tumors (Hypercoagulability, Direct Local Effects of the Tumor, Treatment, and Marantic Endocarditis).

The relationship between cancer and ischemic stroke has been confirmed by numerous studies. A postmortem study showed that 14.6% of cancer patients had cerebral infarcts in their autopsies [22]. Another study conducted in Helsinki found that of all patients with ischemic stroke at a young age (<50 years), about 8% had been diagnosed with cancer, of which 3.6% were diagnosed either prior to or during the initial study, and 3.8% were diagnosed after the etiological study and initial hospitalization [23,24].

The mechanisms by which cancer predisposes to ischemic stroke are numerous, varied, and sometimes not well understood. Some mechanisms are directly associated with the tumor, such as the hypercoagulable state that develops in this type of pathology, compression or direct invasion of the tumor on brain tissue, blood vessels in the brain or neck, or the immunosuppressive state associated with the tumor, which can facilitate the development of cardiac or CNS conditions, thereby increasing the risk of ischemic stroke [24]. On the other hand, mechanisms related to therapies for treating the underlying tumor pathology have been identified.

These treatments increase the risk of stroke of cardiovascular origin by damaging the vascular endothelium or promoting a prothrombotic state, as seen with hormone therapy. Chemotherapy treatment can also lead to an immunosuppressive state, increasing the risk of infections, which in turn may associate with an increased risk of stroke. Cases are beginning to be reported, and the first studies have been published on thrombotic events and stroke associated with new immunotherapies (anti-PD1 and anti-PDL1), as they increase the immune response against the tumor, which could raise cytokine levels, inflammatory substances, and inflammation at the vascular level, thus increasing vascular risk. Finally, other treatments like radiotherapy in the cranio-cervical regions have shown an association with stroke by causing vascular damage, fibrosis, and possible stenosis of blood vessels in irradiated areas [25].

When ruling out a neoplasm as the cause of ischemic stroke in a young patient, it is important to exclude more common etiologies and pay attention to specific analytical parameters such as tumor markers or D-dimer, which can be elevated in tumor pathologies, among other causes. A systematic review found that patients with stroke associated with active cancer had significantly higher levels of D-dimer, with a mean difference of 4.84 μg/ml compared to patients without cancer. This finding underscores the relationship between hypercoagulability in the context of cancer and the increased risk of thromboembolic events, such as ischemic stroke [26].

In conclusion, cancer is an important risk factor for ischemic stroke, with multiple mechanisms contributing to this association, including hypercoagulability, tumor invasion, and treatments that increase vascular risk. Screening for cancer in young stroke patients is crucial, with particular attention to D-dimer levels, which can aid in identifying hypercoagulability and guide diagnosis and management.

8. Illicit Drug Use

Numerous studies have shown an association between the use of drugs, such as cocaine, cannabis, amphetamines, opioids, steroids, alcohol, and an increased risk of ischemic stroke. The pathophysiological mechanisms favoring this predisposition are not clear in all cases, but it is known that they include a tissue hypoxia state associated with vasoconstriction, vasospasm, or altered endothelial regulation. Another prominent mechanism is direct vascular damage due to the toxicity of the compounds, which can cause necrotizing vasculitis, or indirectly, by accelerating arteriosclerosis through refractory hypertension or altered lipid metabolism. Additionally, they favor the development of arrhythmias and embolic heart diseases. Finally, they can alter platelet aggregation, promoting it and producing a hypercoagulable state by altering hepatic production of factors involved in coagulation (Factor VII, antithrombin complex activated by FVII, and tissue factor) [27,28].

Not only does the type of substance influence the risk, but the route of administration can also increase it. Parenteral drug users are at a higher risk of septic endocarditis, with the consequent risk of ischemic stroke due to septic embolism. In this regard, cocaine increases the risk of ischemic stroke by 5 times, according to available studies. This risk is multiplied by 6.4-7.9 times in patients with recent use (within the last 24 hours) who use inhaled cocaine in the form of crack [29].

It is important to note that both mortality and comorbidity in stroke of this etiology are higher than in the general population. Even substances with lower perceived risk by the general public, such as alcohol, increase the incidence of both ischemic and hemorrhagic strokes.

Ultimately, substance use is a significant contributor to ischemic stroke risk through mechanisms like vascular damage and altered coagulation. Given these risks, drug screening is essential in young patients with suspected ischemic stroke to guide diagnosis and management effectively.

9. Migraine

In recent years, studies have shown an increase in the prevalence of migraine in young patients who experience ischemic stroke. Specifically, migraine with aura has been identified as an independent risk factor for ischemic stroke in this population. In a cohort study conducted in Korea, it was concluded that patients with migraine with aura had a higher risk, with a hazard ratio of 1.44 (95% CI = 1.09 to 1.89) compared to the general population [30]. No greater association or increased risk has been seen in patients with migraine without aura [30,31].

However, the exact mechanism explaining this association remains unknown. It is hypothesized that it may be due to a direct migraine-stroke association through pathophysiological mechanisms affecting blood vessels and cerebral perfusion, as well as the physiological mechanisms responsible for their regulation. Additionally, a genetic association between the two entities has been observed in syndromes such as CADASIL or MELAS. There are also cardiovascular risk factors shared by both conditions, which could suggest an indirect association [32].

It has been shown that migraine can act synergistically with other risk factors, such as smoking, increasing the risk of ischemic stroke by up to 9 times, and in women on combined hormonal contraceptives, which increase the risk by 7 times. In this context, young women who smoke and use hormonal contraceptives have a 30 times higher risk than the general population [16].

In conclusion, migraine with aura is a notable risk factor for ischemic stroke in young patients, likely influenced by both direct pathophysiological mechanisms and shared risk factors. Its interaction with modifiable risks, such as smoking and hormonal contraceptives, underscores the importance of tailored preventive strategies for at-risk individuals.

Conclusion

In conclusion, ischemic stroke in young adults is a complex and multifactorial condition, with a wide range of etiologies contributing to its occurrence. While traditional cardiovascular risk factors such as smoking, hypertension, and obesity are prevalent in this population, other factors such as congenital anomalies (e.g., patent foramen ovale), cervicocephalic arterial dissection, genetic diseases, and inflammatory vasculopathies also play a crucial role in the development of ischemic stroke in young individuals.

Furthermore, lifestyle factors, including the use of illicit drugs and poor physical activity, are increasingly implicated in the rising incidence of stroke in this age group.

A comprehensive diagnostic approach is necessary to identify these diverse causes, and early intervention is key to reducing the impact of stroke on young adults. Tailored strategies that address modifiable risk factors, such as smoking cessation, physical activity promotion, and effective management of cardiovascular conditions, can significantly reduce the likelihood of stroke occurrence. Additionally, for patients with genetic or congenital predispositions, personalized interventions such as percutaneous closure of patent foramen ovale (PFO) should be considered to prevent recurrent strokes.

Incorporating these findings into clinical practice will ensure that young patients with ischemic stroke receive the most appropriate care. Furthermore, raising awareness among both healthcare professionals and the general public about the unique risk factors for ischemic stroke in this population is crucial for preventing long-term disability and improving overall outcomes. The need for continued research into the causes and management of ischemic stroke in young adults remains essential to better understand this condition and optimize prevention and treatment strategies.

Conflict of Interest

None declared.

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