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Review Article

Stroke in the Young: Shifting Epidemiology, Aetiological Breadth, and the Case for a Structured, Equity-Conscious Diagnostic Approach: A Narrative Review

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Abstract

Background: Ischaemic stroke in young adults, usually taken to mean the years between 18 and 50, has long been regarded as a marginal corner of cerebrovascular disease. That view is now hard to sustain. Age-standardised rates have indeed fallen among older people; in the young they have held flat or risen across many high-income settings, and the absolute global burden keeps climbing, most steeply in low and lower-middle income regions.

Summary of review: This review draws together current evidence on the epidemiology, causes, diagnostic evaluation, and secondary prevention of ischaemic stroke in young adults. The causes are varied and shift with age. Cervical artery dissection, patent foramen ovale with paradoxical embolism, premature atherosclerosis fed by a rising burden of early-onset vascular risk factors, monogenic arteriopathies, prothrombotic and inflammatory disorders, and the mechanisms peculiar to pregnancy all warrant consideration, and yet a sizeable share of cases still defies classification. We set out an original tiered diagnostic algorithm, weigh the randomised evidence behind patent foramen ovale closure and the place of the RoPE score, and examine what remains unsettled in cervical artery dissection and in embolic stroke of undetermined source.

Conclusions: What most often decides outcome in young stroke is not a shortage of effective treatment. More commonly it is a failure of recognition, of complete investigation, and of fair access to the treatments that already exist. A disciplined, phenotype-driven work-up, genuine attention to the psychosocial and occupational toll carried by survivors, and a deliberate effort to narrow gaps in diagnostic access together offer the most realistic route to better outcomes in this group.

Keywords: *Ischaemic Stroke; Young Adults; Cervical Artery Dissection; Patent Foramen Ovale; Cryptogenic Stroke; Monogenic Arteriopathy; Secondary Prevention*

1. Introduction

Stroke has long been cast as a disease of later life. There is reason for that: most cerebrovascular events do occur in people past their seventh decade. But the framing has carried a cost, a blind spot that is easy to overlook. Stroke in young adults, usually meaning those aged between 18 and 50, makes up something like 10 to 18 percent of all ischaemic strokes, and the share of productive life it destroys is far larger than that number suggests. [1,5] A stroke at 35 is not a younger version of a stroke at 80, in clinical terms, in social terms, or in economic terms. The survivor is looking at decades of disability, a working life and earnings cut short, family roles upended, and a long stretch of years over which recurrence and further vascular disease can pile up.[10]

Two things mark stroke in the young as a distinct problem, worth approaching on its own terms. The first is the sheer breadth of causes. Older patients are dominated by large-artery atherosclerosis, small-vessel disease, and atrial fibrillation; the young bring a far longer list: arterial dissection, paradoxical embolism through a patent foramen ovale, monogenic disorders, prothrombotic states, inflammatory arteriopathies, and the mechanisms of pregnancy. [1,12] The second is how stubbornly the diagnosis resists us. Even after a thorough work-up, a large minority of young strokes end up labelled undetermined. Sometimes the mechanism is genuinely hidden; just as often the work-up was incomplete, or came too late.[13]

This review brings together current evidence on the changing epidemiology, the range of causes, a structured approach to diagnosis, and the secondary-prevention decisions that most often land on the clinician. One argument runs through all of it: what limits outcomes in this group is rarely a shortage of effective treatment. Far more often the shortfall sits elsewhere, in recognition, in proper investigation, and in fair access to treatments that already exist.

2. A Changing Epidemiological Landscape

The most important epidemiological finding of the past twenty years is a divergence. Stroke incidence has fallen among older adults across many high-income countries. Among adults under 55 it has not, and in several well-characterised cohorts it has done the opposite and risen. [2,6] Global Burden of Disease analyses tell a similar story: age-standardised rates in those aged 15 to 39 have edged down worldwide, yet the absolute number of events has grown substantially, and the sharpest rises sit in low and lower-middle sociodemographic-index regions. [3,4] The burden, in other words, is at once growing and migrating towards the settings least able to absorb it.

What is driving this is no mystery. The conventional vascular risk factors, hypertension, diabetes, obesity, dyslipidaemia, smoking, have all become markedly more common in people in their twenties, thirties, and forties.[7,8] A nationally representative study of United States adults aged 20 to 44 found rising rates of hypertension and diabetes, and poor treatment and control of both, in exactly the age band that was once assumed to be low-risk.[7] Layered on top of these familiar factors is a set of less conventional ones that weigh particularly heavily on the young: physical inactivity and long hours spent sedentary, chronic psychosocial stress, and substance use.[9] The conclusion is an awkward one. A meaningful share of young stroke traces back to the same modifiable exposures that drive cardiovascular disease at large, which means that, in principle, much of it could be prevented.

3. Definitions and Aetiological Classification

No single upper age cut-off for young stroke commands agreement; thresholds of 45, 50, and 55 years all circulate in the literature, which makes comparison across cohorts awkward.[1] Here we use 18 to 50 years, the band applied most often, while keeping in mind that the mix of causes changes steadily across it: dissection, cardioembolic, and cryptogenic mechanisms dominate at the younger end, and atherosclerotic disease carries progressively more weight as 50 approaches.

The Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification is still the usual reference framework, dividing ischaemic stroke into large-artery atherosclerosis, cardioembolism, small-vessel occlusion, stroke of other determined aetiology, and stroke of undetermined aetiology.[11] The weighting of these categories looks very different in the young.

The category of other determined cause, slight in older patients, becomes substantial; within cardioembolism, paradoxical embolism rather than atrial fibrillation does most of the work; and the undetermined group stays stubbornly large. [12,13] Figure 1 sets out an aetiological framework adapted to the young patient, and Figure 2 gives the broad ranges of relative contribution reported across the major cohort studies.

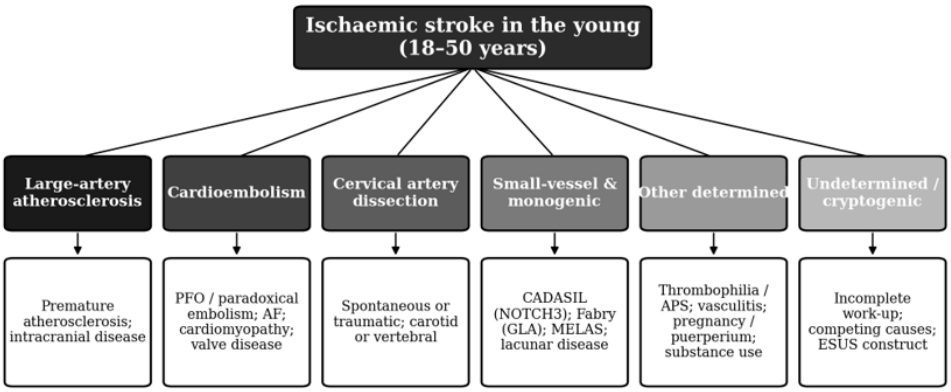

Figure 1. Aetiological framework for ischaemic stroke in the young. A working classification of mechanisms organised around the TOAST framework [11] and expanded to reflect causes over-represented in young adults. The categories are not mutually exclusive: a patent foramen ovale, for example, may be incidental rather than causal, and competing mechanisms frequently coexist. Original figure created by the authors.

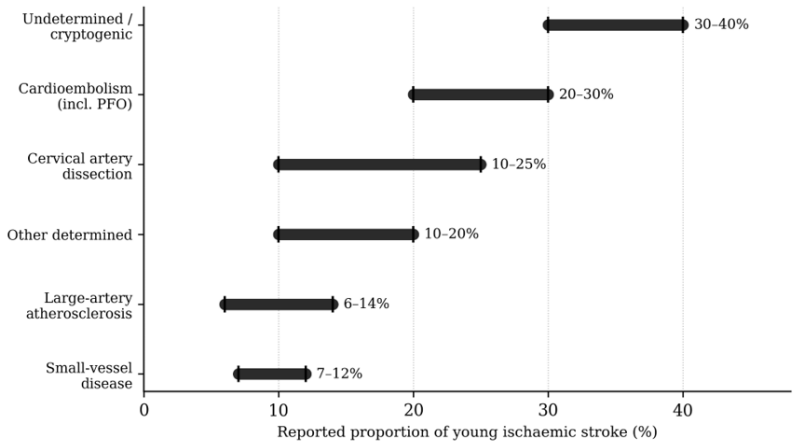

Figure 2. Representative reported ranges of aetiological categories in young ischaemic stroke. Indicative proportional ranges synthesised from cohort and registry studies of ischaemic stroke in young adults. [1,12,13] Categories overlap across classification systems and are not mutually exclusive; reported proportions vary substantially with population, age band, and intensity of investigation. Original figure created by the authors; values derived from cited published cohorts.

4. The Aetiological Spectrum

4.1 Premature atherosclerosis and early-onset vascular risk

Atherosclerotic stroke was once thought rare before middle age. Not any longer: it contributes increasingly to events in young adults, tracking the climbing rates of early-onset hypertension, diabetes, and dyslipidaemia. [7,8] Intracranial atherosclerotic disease, in particular, varies by geography and ethnicity, running higher among South and East Asian and Black populations, a pattern that bears directly on the global epidemiology set out above.[2] The lesson for the clinic is simple enough: conventional risk factors in a young patient should not be waved away as incidental, because they often point to a real, and treatable, atherosclerotic mechanism.

4.2 Cervical artery dissection

Cervical artery dissection, of either the carotid or the vertebral arteries, ranks among the leading causes of stroke at this age, behind a substantial share of events in adults under 50, with reported figures often falling between 10 and 25 percent.[15] It begins with a tear in the arterial wall and an intramural haematoma, which narrows or occludes the lumen or throws emboli downstream. It may happen spontaneously or after trivial neck trauma, strenuous exertion, or chiropractic manipulation. The textbook triad, unilateral head or neck pain, a partial Horner syndrome, and delayed cerebral ischaemia, is often incomplete, and the headache is repeatedly mistaken for migraine, costing time at exactly the moment when treatment counts for most. [15]

Diagnosis turns on vascular imaging. Fat-saturated T1-weighted cervical magnetic resonance imaging showing intramural haematoma, together with computed tomographic or magnetic resonance angiography, has largely replaced catheter angiography. On the choice of antithrombotic therapy, two randomised trials, CADISS and TREAT-CAD, set antiplatelet against anticoagulant treatment and found no convincing advantage for anticoagulation; antiplatelet therapy is a sensible default for most patients, with anticoagulation held back for selected high-risk presentations. [16,17]

4.3 Cardioembolism and patent foramen ovale

A patent foramen ovale is found in about a quarter of the general adult population, and it turns up more often than chance would predict in young patients whose stroke is otherwise cryptogenic, where it can let a venous thrombus cross paradoxically into the arterial circulation.[18] The hard part is attribution. Because the background prevalence is so high, a foramen found after a stroke may be the culprit or merely a bystander. The Risk of Paradoxical Embolism (RoPE) score was built to put a number on that likelihood, scoring a causal relationship as more probable in younger patients who have a cortical infarct and no conventional vascular risk factors.[18] Figure 3 shows the pattern: the higher the score, the larger the fraction of strokes attributable to the foramen. The more recent PASCAL classification goes further, combining the RoPE score with anatomical high-risk features, an atrial septal aneurysm or a large interatrial shunt, to sharpen selection. [19]

Paradoxical embolism aside, the cardioembolic causes seen in the young include cardiomyopathy, valvular disease, infective endocarditis, and, far less often than in older adults, atrial fibrillation. A structured cardiac work-up, electrocardiography, prolonged rhythm monitoring, and echocardiography with an agitated-saline study, is therefore part of the core evaluation.

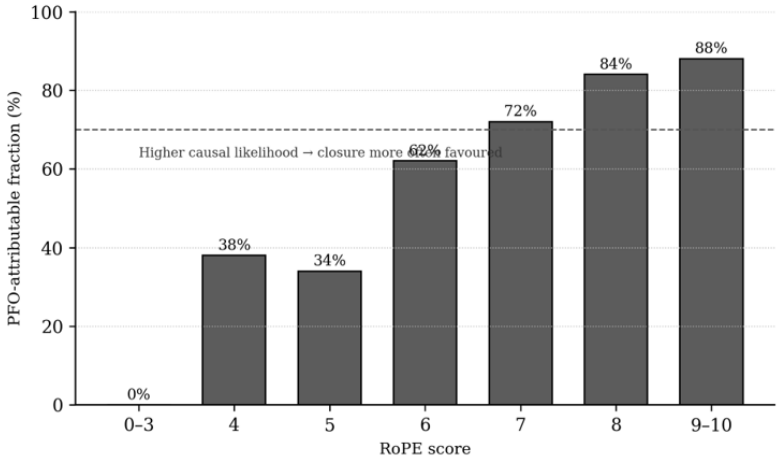

Figure 3. RoPE score and the estimated PFO-attributable fraction. Estimated proportion of cryptogenic strokes attributable to a patent foramen ovale, stratified by RoPE score.[18] Higher scores, driven largely by younger age and the absence of conventional vascular risk factors, indicate a greater probability that an identified foramen is causal rather than incidental, and accordingly that closure is more likely to benefit. Original figure created by the authors; values derived from the cited RoPE analysis.

4.4 Small-vessel and monogenic arteriopathies

Single-gene disorders cause only a small fraction of strokes overall. But that fraction is concentrated in the young, and picking one up matters far more than its rarity suggests, for the patient's prognosis, for the chance of a disease-specific treatment, and for what the family is told.[5] Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), caused by pathogenic NOTCH3 variants, is the commonest inherited small-vessel disease. Its signature, white-matter hyperintensities in the anterior temporal pole and external capsule on magnetic resonance imaging, should trigger testing. Recent genomic data suggest pathogenic NOTCH3 variants are a good deal more common than was once assumed.[28,29] Fabry disease, an X-linked lysosomal disorder caused by GLA variants, warrants special attention on two counts: it responds to enzyme replacement, and the stroke often comes before the diagnosis is ever made; spotting it should prompt a search for the renal and cardiac involvement that drives long-term prognosis.[30] Mitochondrial encephalopathy with lactic acidosis and stroke-like episodes (MELAS), along with an assortment of rarer connective-tissue and metabolic disorders, rounds out the group.

The practical point is that conventional vascular risk factors do not rule out a monogenic cause. A phenotype-driven approach, alert to family history, telltale imaging patterns, and multisystem involvement, should steer who gets selective genetic testing. Systematic genetic assessment of young patients with otherwise cryptogenic stroke is an active field, and it has been shown to uncover diagnoses that would otherwise slip through.[31]

4.5 Prothrombotic, inflammatory, and other determined causes

A mixed bag of disorders fills out the determined causes. Inherited and acquired thrombophilias play a part, though their causal role in arterial stroke is often overstated; the antiphospholipid syndrome is the one that matters most clinically, because it mandates anticoagulation and has consequences for pregnancy.[33] Inflammatory and infectious arteriopathies, primary angiitis of the central nervous system, systemic vasculitides, and infection-associated vasculopathy, require directed serological, cerebrospinal fluid, and vessel-wall imaging evaluation. Migraine, particularly migraine with aura, is associated with a modestly elevated risk of ischaemic stroke in young women, an association amplified by combined oral contraceptive use and smoking.[32] Substance use, notably cocaine and amphetamines, is an under-recognised and growing contributor and should be actively sought through history and toxicology.[35]

4.6 Pregnancy and the puerperium

Pregnancy and the weeks just after delivery raise the risk of both ischaemic and haemorrhagic stroke for a time, through physiological hypercoagulability, haemodynamic shifts, and the hypertensive disorders of pregnancy.[34] The greatest risk falls around delivery and in the first weeks afterwards. Pre-eclampsia and eclampsia, posterior reversible encephalopathy syndrome, reversible cerebral vasoconstriction syndrome, and cerebral venous sinus thrombosis all loom large here, and each calls for a diagnostic approach that differs from the one used outside pregnancy. These same mechanisms also bear on secondary prevention and on contraceptive counselling for women of childbearing age who have had a stroke.

4.7 Cryptogenic stroke and embolic stroke of undetermined source

Even after a comprehensive work-up, a large share of young strokes, often a third or more, stay cryptogenic. [12,13] The embolic stroke of undetermined source (ESUS) construct was proposed to carve out, within this group, patients with non-lacunar infarcts and neither a high-risk cardioembolic source nor relevant arterial stenosis, on the assumption that a hidden embolic mechanism was at work and might yield to anticoagulation. [25] The idea was tested and did not hold up. The NAVIGATE ESUS and RE-SPECT ESUS trials showed that rivaroxaban and dabigatran, respectively, were no better than aspirin for secondary prevention, and rivaroxaban caused more bleeding.[26,27] ESUS, then, is better read as a cue to investigate harder than as a reason to treat empirically, because the label often hides a specific and actionable mechanism, paradoxical embolism, occult atrial fibrillation, or an arteriopathy, that a more thorough work-up can bring to light.

5. A Structured Diagnostic Approach

With a differential this broad, an unstructured, scattergun evaluation is both wasteful and apt to miss treatable causes. We favour a tiered approach, set out in Figure 4, working from investigations every patient should have towards selective, phenotype-driven testing.

Every patient needs the ischaemic stroke confirmed, and here magnetic resonance imaging with diffusion-weighted sequences outperforms computed tomography for small infarcts and those in the posterior fossa. First-line work then covers vascular imaging of the head and neck, electrocardiography with at least 24 hours of cardiac monitoring, transthoracic echocardiography with an agitated-saline study, and a baseline blood panel: full blood count, glucose and glycated haemoglobin, lipid profile, and inflammatory markers.[14] If this first tier turns up a mechanism, attention can move to secondary prevention aimed at that mechanism. If it does not, a second tier follows: prolonged cardiac monitoring, transoesophageal echocardiography, vessel-wall magnetic resonance imaging, and screening for thrombophilia, antiphospholipid antibodies, autoimmune disease, and toxins. Only when these too come back empty, and then guided by the clinical phenotype, is a third tier warranted, selective genetic testing, cerebrospinal fluid analysis, and catheter angiography. [12]

Two things make this discipline worthwhile. It spares the patient the scattershot ordering of low-yield tests that marks so much young-stroke practice, and it cuts down how often a stroke is labelled cryptogenic too early and wrongly, a label that too readily becomes the end of the diagnostic process rather than a stage in it.

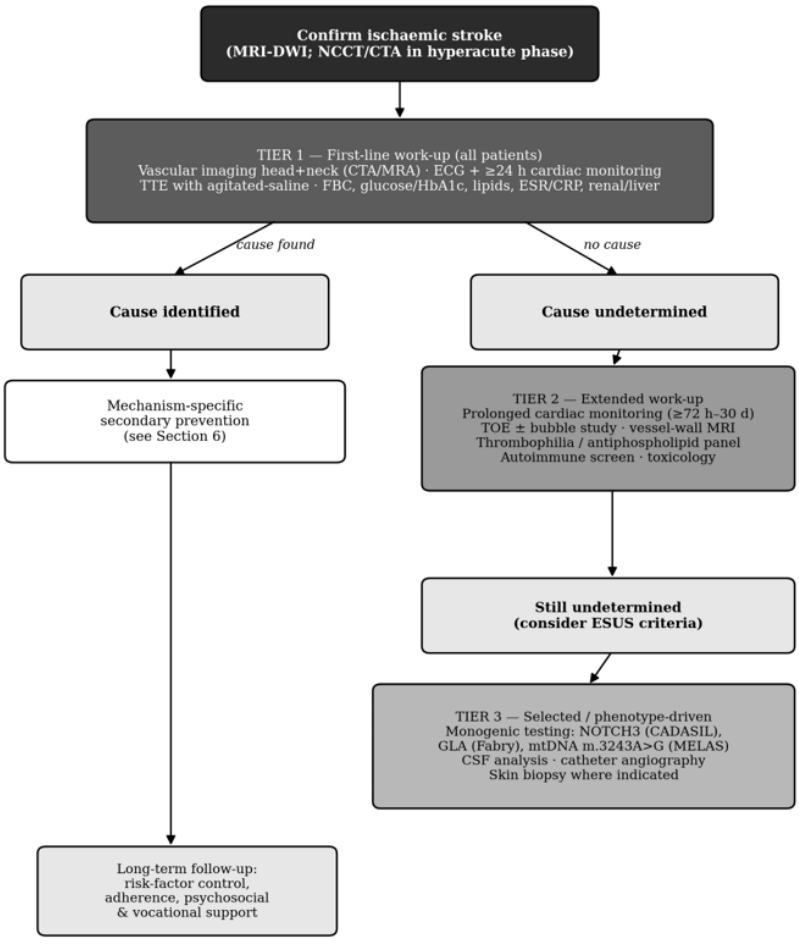

Figure 4. A tiered diagnostic algorithm for ischaemic stroke in the young.
Proposed staged evaluation proceeding from universal first-line investigations through extended and selective phenotype-driven testing. The algorithm is intended as a pragmatic framework rather than a rigid protocol, and should be adapted to clinical context and local resources. Original figure created by the authors.

6. Secondary Prevention: Where the Evidence is Firm, and Where it Runs Out

6.1 Patent foramen ovale closure

Of all the secondary-prevention questions in young stroke, this is the one where the evidence has firmed up most. The early trials were neutral, but a second wave of randomised studies, CLOSE, the long-term RESPECT analysis, REDUCE, and DEFENSE-PFO, showed that percutaneous device closure plus antiplatelet therapy cut recurrent stroke compared with medical therapy alone, in carefully chosen patients aged 60 or younger whose foramen was likely to be causal.[20,21,22,23] Pooled analyses bear out an absolute drop in recurrence, paid for by a small excess of atrial fibrillation that is usually transient.[42] European Stroke Organisation guidance backs closure in suitably selected patients after multidisciplinary assessment, and it is that selection, built on the RoPE score and the PASCAL classification, that decides whether the benefit materialises.[19,24] The question for the clinician is no longer whether closure works, that much is settled, but whether the particular foramen in front of them is the culprit, which is exactly what the scoring systems are for.

6.2 Cervical artery dissection and other determined causes

For dissection, as already noted, antiplatelet and anticoagulant therapy look broadly equivalent, and antiplatelet treatment is the sensible default for most patients.[16,17] The antiphospholipid syndrome calls for anticoagulation. Monogenic disorders are managed by pairing disease-specific therapy where it exists, enzyme replacement in Fabry disease being the obvious example, with tight control of conventional vascular risk factors, which helps whatever the underlying mechanism.[30] Across every subtype, vigorous treatment of modifiable risk factors, following current primary and secondary prevention guidance, is the shared foundation.[36,37]

6.3 Cryptogenic stroke and ESUS

When a stroke is still undetermined after a thorough work-up, the ESUS trials make clear that empirical anticoagulation is not justified; antiplatelet therapy together with risk-factor modification remains standard.[26,27] The more useful response to a cryptogenic label is to investigate again rather than to escalate empirical treatment: prolonged rhythm monitoring for occult atrial fibrillation, a second look at a foramen written off earlier, and a careful eye for phenotypic hints of arteriopathy.

7. Beyond Recurrence: The Hidden Burden on Survivors

Outcome in young stroke is too often judged by recurrence and gross functional disability alone, measures that capture only part of the burden. Many young survivors recover good physical function and are still profoundly affected by problems that standard scales never register. Depression and anxiety are common; a meta-analysis has documented a high prevalence of post-stroke mood symptoms in this group.[38] Returning to work, which matters enormously to people with decades of working life still ahead, is managed by only some survivors, and whether it happens depends on cognitive sequelae, fatigue, and psychosocial factors as much as on motor recovery.[39] The list runs on: cognitive impairment, fatigue, changes in relationships and in sexual function, and the loss of independence and of identity that comes with a stroke this early. Qualitative work has only begun to map this burden, and services are badly set up to meet it.[40]

There is a service-design lesson in all this. Pathways designed around older patients, and geared towards survival and basic independence, fit poorly with a 35-year-old whose main concerns are getting back to work, holding relationships together, and starting or raising a family. Age-appropriate rehabilitation, vocational support, and psychological care are not optional extras; they are core to managing young stroke well.

8. Gaps, Inequities, and Future Directions

Three priorities fall out of this review. The first is prevention. The climbing burden of early-onset vascular risk factors is the most tractable driver of young stroke, and tackling it lies largely outside the stroke unit, in public health, primary care, and policy.[7,37]

The second is diagnosis. The stubbornly large cryptogenic fraction reflects, in part, incomplete and unequal access to advanced investigation, and the yield that systematic evaluation can achieve is not realised evenly across health systems.[31] The third is equity. Because the rising burden is concentrated in low and lower-middle income regions, where diagnostic and interventional resources are thinnest, the gap between what could be done and what actually is done is widening exactly where need is greatest. [2,3]

That last point recasts the whole challenge. For most of the mechanisms covered here, effective evaluation and treatment already exist. What decides outcomes at the population level, then, is not the cutting edge of therapeutic evidence but the plumbing of recognition, investigation, and fair delivery. Research that measures and closes these access gaps may do more for young stroke survivors than another incremental tweak to interventions we already know work.

9. Conclusion

Stroke in the young is no longer a clinical curiosity. The incidence is climbing, the causes are wide-ranging and often treatable, and the fallout, spread across decades of disrupted life, is severe. A disciplined, phenotype-driven diagnostic approach shrinks the share of cases left in the cryptogenic bin and brings to light the specific, actionable mechanisms that secondary prevention depends on. And yet the evidence gathered here keeps returning to one awkward point. What stands between these patients and better outcomes is, more often than not, not a missing treatment but a missed diagnosis, an investigation never completed, and care that never reached them. Confronting that, through prevention, structured evaluation, attention to the psychosocial toll on survivors, and a real effort to close gaps in access, is the most realistic way to improve the lives of young people who have a stroke.

Declarations

No use of artificial intelligence. During the preparation of this manuscript the authors AI tools were NOT used.

Author contributions. The following statement reflects the CRediT taxonomy and must be confirmed by all named authors to ensure it accurately represents actual contributions. Conceptualisation: A.J.D. Literature review and writing of the original draft: S.K. Critical revision for important intellectual content: G.G., V.K. Supervision and final approval: A.J.D. All authors reviewed and approved the final manuscript and agree to be accountable for all aspects of the work.

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Data availability. No primary datasets were generated or analysed. All data discussed are available in the cited published sources.

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