Artery Research

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Advances in Knowledge Regarding Arterial Curvature and its Relationship to Ischemic Stroke: A Narrative Review

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Abstract

Curvature of large arteries in the cervical and intracranial regions represents a common morphological vascular alteration. However, the precise mechanisms underlying its formation and progression remain unclear. Increasing evidence suggests a potential correlation between arterial curvature and ischemic stroke. This manuscript involves a review of the relevant literature that delineates the measurement techniques for assessing arterial curvature, the mechanisms contributing to its formation, and recent evidence linking arterial curvature to stroke. Arterial curvature is primarily influenced by genetic factors, hypertension, sex, and age, and it may indicate weakened arterial walls. Furthermore, the haemodynamic changes associated with arterial curvature can result in reduced cerebral perfusion pressure, endothelial dysfunction, oxidative stress, and inflammatory responses. These factors contribute to the development of atherosclerosis and the formation of arterial dissections. The degree of arterial curvature may serve as a risk factor for cerebral ischemia, alongside traditional vascular risk factors. Further investigation into arterial curvature could yield valuable insights for clinicians managing patients with curved vessels.

Keywords Arterial curvature, Ischemic stroke, Atherosclerosis, Arterial dissection

1 Introduction

Arterial curvature is a prevalent morphological alteration that has garnered increasing attention in recent years, particularly in the cervical and intracranial regions. In 1913, otolaryngologists first recognized the significance of the internal carotid artery (ICA) curvature, noting fatal haemorrhagic surgical complications in patients with a curved artery close to the posterior pharyngeal wall [1]. Extreme intracranial arterial curvature and dilatation, known as intracranial arterial dolichoectasia, primarily affects the basilar artery (BA). This condition is believed to be linked to cerebral infarction, cerebral haemorrhage, and subarachnoid haemorrhage in the event of aneurysm rupture [2]. Diagnostic criteria for vertebrobasilar dolichoectasia have been established [3], but criteria for dolichoectasia of the systemic arteries in the anterior circulation are lacking. Despite the presence of arterial curvature in many cases that do not meet the diagnostic criteria for dolichoectasia, the association between abnormal arterial morphological changes and cerebrovascular disease remains unclear.

Most of the research investigating vascular curvature is qualitative. For example, carotid artery morphology is classified according to the Weibel–Fields criteria as tortuosity, kinking, and coiling [1], while BA morphology is classified as C-shape, S-shape, and J-shape [3]. In recent years, advancements in imaging technology have led to the development of quantitative methods for describing vascular curvature. The most commonly used method involves calculating the ratio of the actual length of the artery between two points to

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the straight-line distance between the same points [4]. Additionally, quantitative analysis of arterial angulation [5] has been developed. This allows for a more objective and accurate description of the degree of arterial curvature and provides a basis for creating uniform criteria for evaluating the degree of arterial curvature in the future.

Intracranial arteries are particularly vulnerable to haemodynamic changes compared to extracranial arteries due to their smaller diameters, thinner media and external membranes, and fewer elastic fibers [6, 7]. Multiple studies have confirmed the link between the carotid, vertebral (VA), and intracranial artery curvature and cerebrovascular disease [8–15]. Despite this, the debate continues as to whether arterial curvature should be considered a marker of pathological changes in the arteries and a risk factor for ischemic events. This manuscript aims to review the measurement techniques for assessing the curvature of large arteries, analyse the mechanisms contributing to its formation, and explore the roles of the carotid artery, VA, and intracranial arterial curvature in the development and progression of ischemic stroke.

2 Different Methods of Measuring Arterial Curvature

2.1 Morphological Classifications of Arterial Curvature

There are three widely used morphological classifications for the carotid arteries. Metz's criteria [16], introduced in 1961, categorises morphological changes based on the angle of curvature into three types: type 1 (60–90 degrees), type 2 (30–60 degrees), and type 3 (< 30 degrees). Weibel–Fields' criteria [1], established in 1965, classify carotid arteries as straight without curvature, tortuosity, kinking, or coiling. Barbour's criteria [17], introduced in 1994, categorises morphological changes as kinking, coiling, or looping (Fig. 1).

BA curvature can be classified according to its morphology as C-shape, S-shape, J-shape, no deformation, or flat [13]. Elongation and deviation of the BA have been divided into three types based on the relative position between the arteries and cranial bone markers by Smoker's criteria [3], as presented in Table 1.

2.2 Quantitative Classifications of Arterial Curvature

Quantitative evaluation of arterial curvature was first proposed in 1994 and applied to the coronary arteries

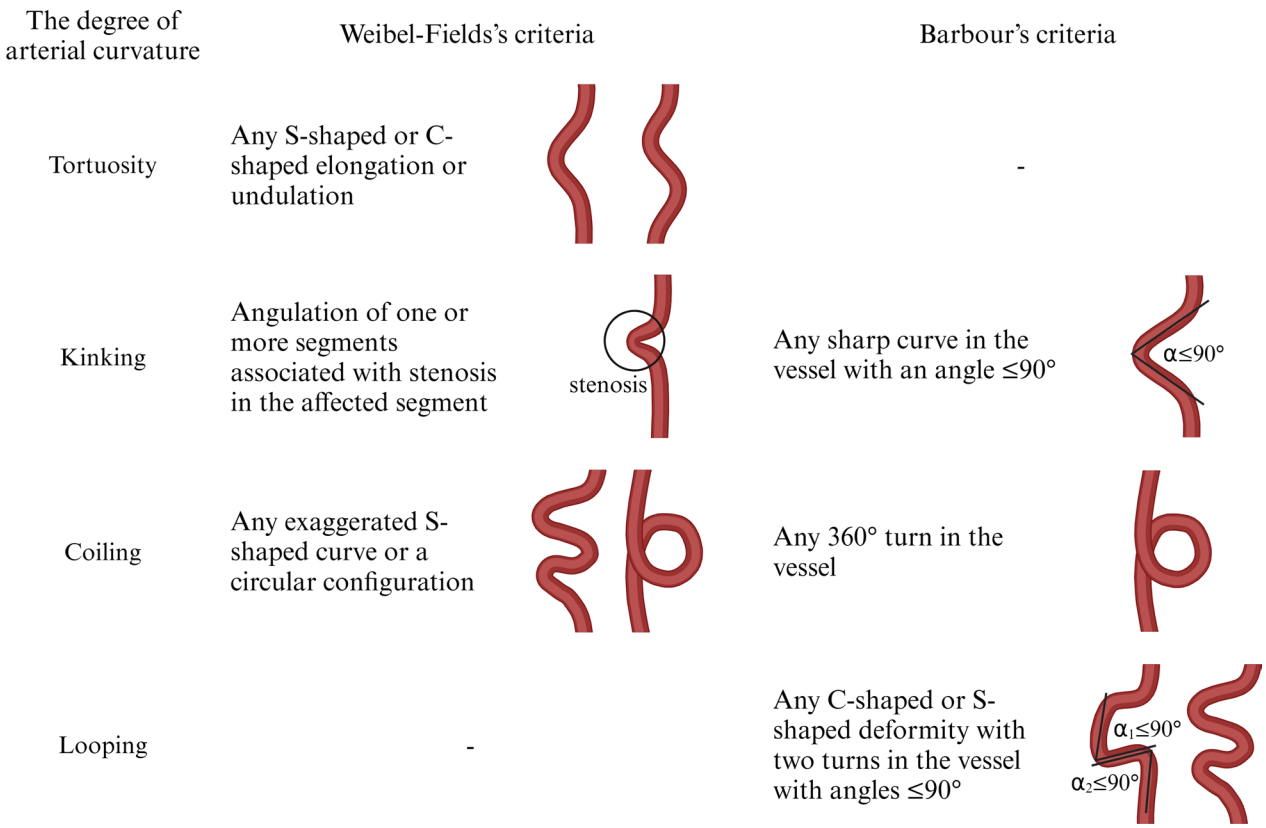

Fig. 1 Comparison of Weibel–Fields' criteria and Barbour's criteria. (Created with BioRender.com.)

Table 1 Smoker's criteria of BA curvature

The degree of BA curvature	Plane of Basilar Bifurcation (Height)	Most lateral position identified throughout the Course of BA (Position)
0	At or below dorsum sellae	Midline throughout
1	Within suprasellar cistern	Medial to the lateral margin of clivus or dorsum sellae
2	At the level of the third ventricle floor	Lateral to the lateral margin of clivus or dorsum sellae
3	Indenting and elevating the floor of the third ventricle	In cerebellopontine angle cistern

[18]. The tortuosity index [19] was introduced in 2011 and has been widely applied to the carotid arteries, VA, and BA. The tortuosity index is based on the ratio of the actual length of the vessel to the straight-line length between the two endpoints, representing the distance factor metric (DFM) [20] (Fig. 2). The tortuosity index is calculated using the following formula:

$$\text{Tortuosity index} = (\text{DFM} - 1) \times 100$$

Additionally, more complex measurements of the arterial curvature have been developed based on the angle and the number of inflection points, including the sum of angular measures (SOAM), a product of angle and distance (PAD), inflection point counting metric (ICM), and triangular index (TI) [5, 21]. Relative length (RL) is used to describe the general degree of arterial curvature (Table 2) (Fig. 2).

Other studies have evaluated arterial curvature using band length (BL) to measure the BA curvature that refers to the longest distance from the greatest curvature of the artery to the straight line between the start and end points of the curve [14, 22]. When applied to the ICA, this method is called 'ICA distance' [23].

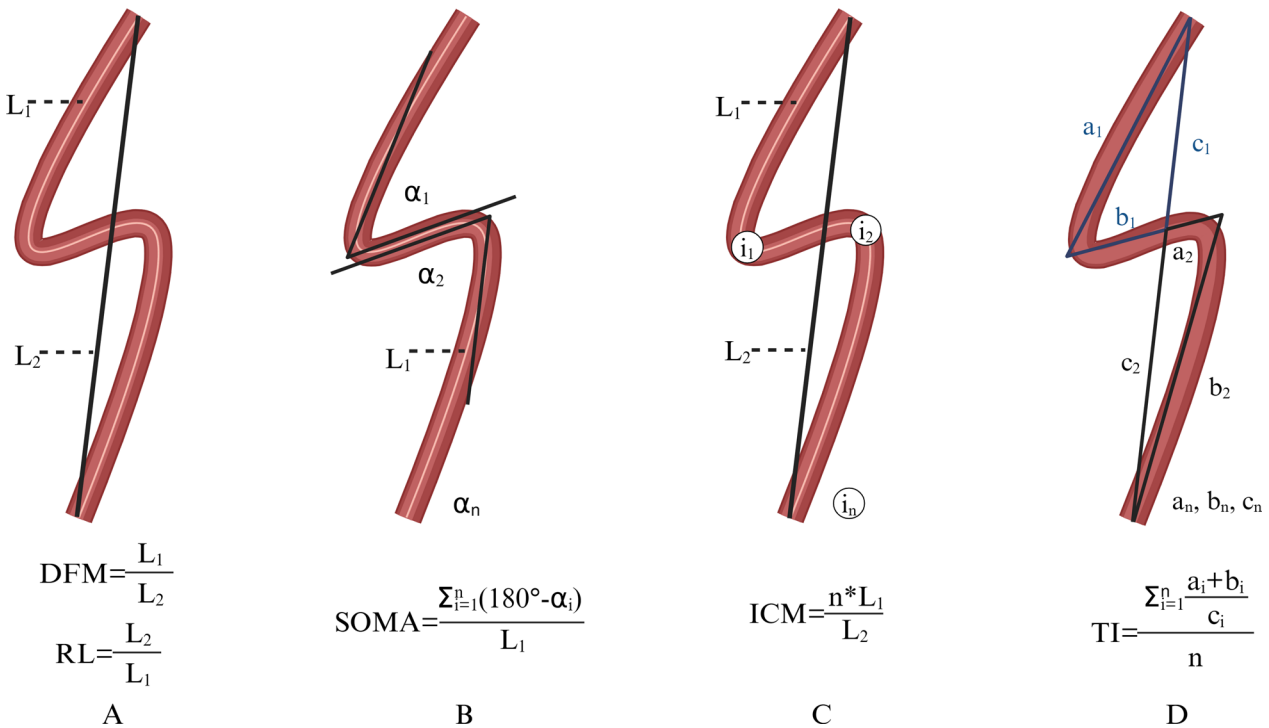

Fig. 2 **A** Definition of distance factor metric (DFM) and relative length (RL). L1 is the actual length of the curve; L2 is the length of the straight-line between the start and end points of the curve. **B** Definition of sum of angular measures (SOAM). α indicates measured angles, and n is its count. L1 is as mentioned above. **C** Definition of inflection point counting metric (ICM). n is the number of the curve's inflection points. L1 and L2 are as mentioned above. **D** Definition of triangular index (TI). *n* is the number of the triangle obtained by partitioning, a and b are triangle sides, and c is its base. (Created with BioRender.com.)

Table 2 Description of quantitative measurements

Formulas	Value change in case of greater arterial curvature	Description of aspects of arterial curvature
$DFM = \frac{L_1}{L_2}$	Larger	Describes the general degree of arterial curvature
$RL = \frac{L_2}{L_1}$	Smaller	Describes the general degree of arterial curvature
$SOMA = \frac{\sum_{i=1}^{n}(180° - \alpha_i)}{L_1}$	Smaller	Describes in terms of angles that it makes
$PAD = \frac{SOMA}{RL}$	-	Describes the global degree of arterial curvature that is a combination of RL and SOAM
$ICM = \frac{n * L_1}{L_2}$	Larger	Describes the degree of arterial curvature in terms of the number of inflection points
$TI = \frac{\sum_{i=1}^{n} \frac{a_i + b_i}{c_i}}{n}$	Larger	Describes the degree of arterial curvature in terms of its arc lengths

3 Clinical Factors in the Occurrence of Arterial Curvature

3.1 Congenital Factors in arterial Curvature

Arterial curvature is sometimes associated with congenital factors. Arterial tortuosity syndrome (ATS) is a rare genetic connective tissue disorder characterised by significant arterial abnormalities, including elongation, tortuosity, and aneurysms in medium and large arteries [24]. Furthermore, other congenital hereditary connective tissue disorders such as Loeys–Dietz syndrome (LDS) [25], Late-Onset Pompe disease (LOPD) [26], and Marfan syndrome [25, 27] are also believed to be linked to multiple arterial curvatures.

Research indicates that a higher degree of arterial curvature is associated with earlier and more severe cardiovascular complications in individuals with congenital disorders such as LDS and Marfan syndrome [28]. In children with dissection, cryptogenic ischemic stroke, transient ischemia, and aneurysms, cerebral arteries often exhibit increased curvature [4, 20]. These findings suggest that the presence of vascular curvature in children may indicate an underlying congenital condition or poor prognosis, highlighting the need for vigilance among clinicians.

3.2 Age is a Factor in Arterial Curvature

Multiple studies have indicated that the prevalence of ICA curvature is higher among older individuals [29–32]. Additionally, the prevalence was significantly higher at the extremes of the age spectrum [29, 30]. In one study, the first peak was observed at nine years of age, with a gradual increase in prevalence in individuals $\geq$ 40 years of age and ultimately peaking at 80 years of age [29]. Another study demonstrated a significantly higher prevalence of extracranial ICA kinking and coiling in those $\leq$ 20 years old and those > 60 years old [30]. This phenomenon can be explained by the asymmetries in arterial and skeletal development proposed by Harrison and Dávalos [29, 33]. According to this hypothesis, the skeleton continues to grow after birth, leading to a reduction in the degree of ICA curvature. In older individuals, osteoporosis results in the thinning of intervertebral discs and a shortening of the cervical spine, consequently increasing the degree of ICA curvature. This hypothesis is supported by studies examining arterial curvature in children that have demonstrated a decrease in both carotid [34] and vertebral artery [30] curvature after a minimum follow-up period of 1 year. However, conflicting evidence exists. One study observed a comparable prevalence of carotid artery curvature in two distinct age groups: infants aged 1 day–15 years and individuals aged 16–96 years [35]. Notably, this study lacked a detailed breakdown of the participants' age groups, and this may have affected the interpretability of the results.

In addition to the carotid system, studies have demonstrated a higher degree of BA [36] and VA [32] curvature in older patients. Most current studies are cross-sectional, and few have investigated the changes in arterial curvature with age in the population [30, 34]. Taken together, the available evidence suggests that advanced age and arterial curvature are related. However, further prospective, large-sample-size studies in community-based populations are needed.

3.3 Female Gender is a Factor in Arterial Curvature

Most studies suggest that the prevalence of arterial curvature is higher in females compared to males in the same age group [37–39]. Females were significantly more likely to experience bilateral ICA kinking than were males. Specifically, 57.8%of patients with unilateral ICA kinking were female, whereas 77.9% of patients with bilateral ICA kinking were also female [40].

3.4 The Relationship Between Arterial Curvature and Traditional Risk Factors for Cerebrovascular Disease is Controversial

Research has indicated that ICA curvature may be linked to hypertension [29, 37, 41, 42]. One study reported that a higher degree of ICA curvature is also linked to hyperlipidaemia, diabetes mellitus, and heart disease [37]. However, not all studies suggest a clear connection between vascular curvature and common risk factors for vascular disease. Some researchers suggest that arterial curvature may not be linked to traditional risk factors such as hypertension, diabetes mellitus, and dyslipidaemia [31, 35, 40, 43]. Further research is necessary to confirm the relationship between vessel curvature and traditional risk factors for vascular events such as hypertension, hyperlipidaemia, and diabetes mellitus.

3.5 Analysis of Possible Mechanisms for the Occurrence of Arterial Curvature

Arterial curvature is influenced by various mechanical factors, including blood pressure, blood flow, axial tension, and structural changes in the vessel wall [44]. Vascular curvature can be induced by high pressure in the lumen and asymmetric blood flow that affects the blood vessel wall. The high prevalence of ICA kinking in patients with hypertension [29, 37, 41, 42] along with the observation that carotid kinking is more common in the left carotid artery [31, 37, 45, 46] supports the theory that high intraluminal pressure plays a role in its pathogenesis. The left ICA is expected to exhibit a higher intraluminal pressure compared to that of the right ICA due to its direct connection to the aortic arch. Changes in haemodynamic could play an important role in the formation of arterial curvature. The difference in diameter between the right and left vertebral arteries may cause asymmetry in the VA blood flow, resulting in severe BA curvature [15, 47, 48]. Recent research has uncovered an inverse correlation between the dominant side of the VA and BA curvature in majority of patients, ranging from 70 to 89.1% [15, 36, 47].

Additionally, increased blood flow can lead to increased degree of arterial curvature. Recent studies have successfully induced basilar arterial vascular tortuosity using a bilateral common carotid artery ligation model in rats or rabbits [49, 50]. This suggests that a reduction in anterior cerebral blood flow may indirectly result in an increase in posterior blood flow, potentially influencing the curvature of the posterior circulation vessels. An increase in the curvature of the posterior cerebral artery has been observed in patients following middle cerebral artery (MCA) occlusion [51]. Elevated blood flow and increased wall shear stress (WSS) stimulate the activation of matrix metalloproteinase (MMP) and tissue inhibitor of matrix metalloproteinases (TIMP), promote fibrin degradation, and enhance endothelial cell proliferation in both animal models [52, 53] and humans [54, 55]. These may explain the observed arterial curvature in patients with hypertension and high blood flow.

Degradation of elastin weakens the arterial wall, reduces the critical pressure, and thus contributes to vessel curvature [56]. Higher concentrations of MMP-2 and MMP-9 and lower concentrations of TIMP-1 were observed in pathological tortuous ICA, resulting in the degradation of elastin [57]. Congenital factors such as connective tissue diseases can also increase vessel curvature by affecting the wall structure. Additionally, aging leads to elastin breakdown and restructuring of the cerebral arteries, promoting atherosclerosis and reducing vascular compliance [58]. These mechanisms may explain the occurrence of arterial curvature in older patients.

Although arterial curvature is linked to atherosclerosis, its association with traditional cardiovascular disease factors is debated, and the underlying mechanisms remain unknown, necessitating further investigation.

4 Correlation Between Arterial Curvature and Ischemic Stroke and Transient Ischemic Attack (TIA)

Stroke is the second leading cause of disability and death worldwide. Approximately 87% of strokes are ischemic, with an estimated 10–20% of the large artery occlusion type [59]. The relationship between ischemic stroke and arterial curvature has garnered significant research attention in recent years. Studies have indicated that half of stroke patients exhibit supra-aortic arterial curvature [60], and 39.2% of patients undergoing thrombectomy for ischemic stroke due to large artery occlusion possess ICA curvature [61].

Recent studies have revealed a strong association between anterior circulation stroke and carotid artery curvature [8–11]. Moreover, the prevalence of subcortical ischemic changes was significantly higher in patients with bilateral ICA kinking than it was in those with unilateral ICA kinking [40]. In a separate case–control study, quantitative values were used to predict acute ischemic stroke. A tortuosity index $\geq$ 16.91 for the extracranial segment of the ICA was associated with an increased likelihood of anterior circulation infarction (sensitivity of 0.71 and specificity of 0.60) [12]. Another retrospective study revealed that patients with ICA kinking exhibited an increase of 0.55-points in the Essen Stroke Risk Score and a 10.34-fold higher risk of small artery obstructive stroke than did those without ICA kinking [10]. However, a prospective study yielded negative results when comparing a group of 34 patients with hypertension with ICA kinking and coiling with a control group of 36

well-matched individuals over a 7-year follow-up period. There was no statistically significant difference in the incidence of vascular events between the two populations [62]. In contrast, a recent prospective study with a larger sample size of 615 patients over a 1-year follow-up period indicated that 4% of patients with carotid artery tortuosity and 2.88% of those without experienced stroke or transient ischemic attack events [63]. Overall, available studies support the association between carotid artery tortuosity and ischemic cerebrovascular disease.

The posterior circulation demonstrates higher geometric variability compared to that of the anterior circulation, increasing the likelihood of ischemic stroke due to curvature in posterior arteries [11]. Studies have indicated a connection between the VA curvature and posterior circulation ischemic stroke [11, 12]. A retrospective study determined that severe kinking of the VA was independently linked to a 39% higher risk of acute ischemic stroke or TIA [11]. Research has indicated that a tortuosity index ≥ 22.96 in the extracranial segment of the VA indicates a heightened risk of posterior circulation infarction (sensitivity of 0.85 and specificity of 0.70) [12]. Recent studies have also suggested that patients with BA curvature experience a greater risk of posterior circulation ischemic stroke [13–15]. Specific locations of pontine infarction are closely linked to BA curvature, with greater BA curvature associated with lateral and central pontine infarction, and a greater degree of BA curvature linked to paracentral or caudal pontine infarction [64]. The incidence of infarcts in the posterior inferior cerebellar artery (PICA) and BA region was significantly higher in the BA curvature group compared to that of the BA flattening group [47]. This discrepancy may be attributed to the observation that BA curvature compresses and stretches the cerebral bridge-penetrating arteries [65].

There is one case report of ATS causing recurrent TIA in a young patient [66]. This suggests there may be an independent correlation between arterial curvature and TIA. However, there is a lack of large-scale analytical studies on the relationship between TIA and arterial curvature.

5 Correlation Between Arterial Curvature and Arterial Dissection

Cervical and cerebral arterial dissection is a significant contributor to ischemic stroke in young and middle-aged individuals [67]. Arterial dissection is a potential cause of stroke in children [4]. Research indicates that 64% of patients experiencing acute dissection-related cerebrovascular issues experienced carotid artery dissection, while 36% experienced VA dissection [68]. Recognizing the association between arterial curvature and the likelihood of spontaneous arterial dissection can aid in identifying patients with a higher risk of this condition.

Current research indicates that individuals with ICA dissection tend to exhibit a higher degree of ICA and VA curvature compared to that of those without dissection across different age groups. A study involving individuals aged 1 month–18 years demonstrated increased DFM of the ICA and VA in those with arterial dissection, as opposed to controls without dissection and no history of certain medical conditions [20]. Studies in adults have also demonstrated a higher degree of ICA curvature and VA curvature in those with spontaneous carotid artery dissection [68–71]. However, the degree of VA curvature was not significantly different in patients with VA dissection [68].

Although the occurrence of arterial dissection is associated with arterial curvature, the association between arterial curvature and the recurrence of arterial dissection remains a topic of debate. One study observed that patients with carotid artery dissection who experienced recurrence in 3–6 months possessed a higher degree of VA curvature compared to those who did not. The median VA tortuosity index was 20.2 for patients with recurrence and 7.2 for those without [70]. In contrast, a separate cohort study with a follow-up period of 1–10 years did not find an association between the recurrence of ICA dissection and VA tortuosity [72]. This discrepancy in findings may be attributed to differences in the follow-up duration.

6 Correlation Between Arterial Curvature and the Prognosis of Ischemic Stroke

Arterial affects the incidence of ischemic stroke and the treatment outcomes for affected patients. Reperfusion therapy is a critical component of the management of ischemic stroke, encompassing both endovascular treatment and intravenous thrombolysis for acute cerebral infarction.

The duration of endovascular treatment serves as a key predictor of neurological outcomes [73]. Notably, carotid artery curvature has been associated with prolonged mechanical thrombectomy duration and reduced rates of successful revascularization [41, 61, 74–77]. Patients with an ICA tortuosity index of < 10 also exhibited a threefold higher functional independence rate after early reperfusion than those with a higher ICA tortuosity index [77]. Moreover, patients with curved MCA are associated with revascularization failure following endovascular treatment [78].

In patients who do not receive endovascular treatment upon admission, an increased degree of arterial curvature serves as an independent predictor of unfavourable outcomes. Specifically, an extracranial carotid artery

tortuosity index ≥ 12.5 may indicate a higher likelihood of adverse outcomes [79]. Furthermore, elongation of the BA could potentially serve as a predictor of poor prognosis at 90 days in patients with acute isolated cerebral pontine infarction [80].

A retrospective cohort study with a follow-up period of 22 ± 6 months indicated that stroke patients diagnosed with vertebrobasilar dolichoectasia may exhibit an increased risk of stroke recurrence [81]. However, there is a notable lack of studies examining the association between the degree of arterial curvature and recurrence of ischemic stroke.

7 Pathophysiological Mechanisms Linking Arterial Curvature and Ischemic Stroke

Vascular curvature may contribute to acute ischemic cerebrovascular disease in two manners. First, it induces endothelial damage as a result of altered blood flow at the tortuous site, leading to atherosclerosis, dissection, and thrombosis [8, 82]. Second, it slows down blood flow and elevates the risk of thrombus formation [83].

The impact of vessel curvature on cerebral perfusion is a topic of debate. According to the haemodynamic theory, blood flow velocity decreases in curved vessels, and curvature increases intravascular resistance, leading to reduced blood perfusion [83, 84]. Severe curvature can block or occlude blood flow, as observed in coronary arteries [85]. This contributes to a decrease in cerebral perfusion pressure and subsequently reduces blood flow.

Clinical and imaging studies have demonstrated that carotid artery tortuosity and kinking can contribute to a decrease in cerebral blood perfusion pressure [86]. Those with BA curvature exhibited reduced local perfusion rates in the brainstem, particularly on the side opposite to the curvature [83]. However, another study determined that while blood flow was notably reduced at the site of the ICA curvature, the maximal systolic and end-diastolic flow rates of the ipsilateral ophthalmic artery remained largely unaffected by kinking [45]. Therefore, further research is essential to investigate the impact of arterial curvature on brain blood perfusion.

The relationship between the arterial curvature and atherosclerosis has been demonstrated in human studies. A high degree of curvature is associated with atherosclerotic plaques in the carotid artery [87, 88], MCA [89], BA [90], and superficial femoral artery [27]. Additionally, plaque formation has been observed to be associated with local blood flow patterns [87, 88]. Arteries with atherosclerotic plaques are vulnerable to mechanical bending, resulting in increased stress within the plaque and potentially increasing the risk of rupture [91].

The 'haemodynamic risk hypothesis' suggests that factors, such as low WSS, disrupted haemodynamic characteristics, and oscillatory flow patterns play a significant role in the development of atherosclerotic plaques [92]. Changes in WSS have been associated with vascular endothelial dysfunction, oxidative stress, site-specific wall remodelling, inflammation, and atherosclerosis [50, 93]. Furthermore, alterations in WSS can impact various pathways involved in embryonic development, thereby influencing the progression of atherosclerosis, as elaborated in other scholarly reviews [94]. A high WSS has been shown to promote high-risk plaque formation [95] and rupture [96], resulting in thrombosis, sudden arterial occlusion, and rapid infarction.

Studies on the pathophysiology of cervical and intracranial arterial dissection are limited. Neuropathological specimens typically reveal disruption of the internal elastic and medial membranes [97]. The breakdown of elastin and weakening of the vessel wall are contributing factors in the formation of arterial curvature and dissection.

Figure 3 summarizes the relationship between arterial curvature and stroke. Arterial curvature may indicate factors that weaken the arterial walls, and this may also lead to arterial dissection. Arterial curvature can change haemodynamic, leading to decreased cerebral perfusion pressure, endothelial dysfunction, oxidative stress, and inflammatory responses, all of which contribute to the development of atherosclerosis and dissection. High WSS may destabilize atherosclerotic plaques, potentially precipitating acute vascular events.

8 Conclusions

This review suggests a new research direction to identify individuals at high risk of stroke by assessing cervical and intracranial arterial curvature alongside traditional risk factors. In individuals with connective tissue disease, hypertension, atherosclerotic disease, and aging, arterial curvature serves as a significant marker of vascular pathological changes. Furthermore, the presence of arterial curvature is associated with an accelerated progression of atherosclerosis and an increased risk of ischemic events. Evidence suggests that patients with a high degree of arterial curvature are at a higher risk of cerebral ischaemic events and may exhibit a worse prognosis compared to patients with a low degree of curvature. However, the use of arterial curvature as an independent predictive risk factor for these conditions and the need for early intervention remain uncertain.

Future studies should focus on establishing a connection between the degree of arterial curvature and the traditional cerebrovascular disease risk factors. Additionally, large-sample prospective clinical research is necessary to determine whether individuals with severely curved arteries, particularly those with only

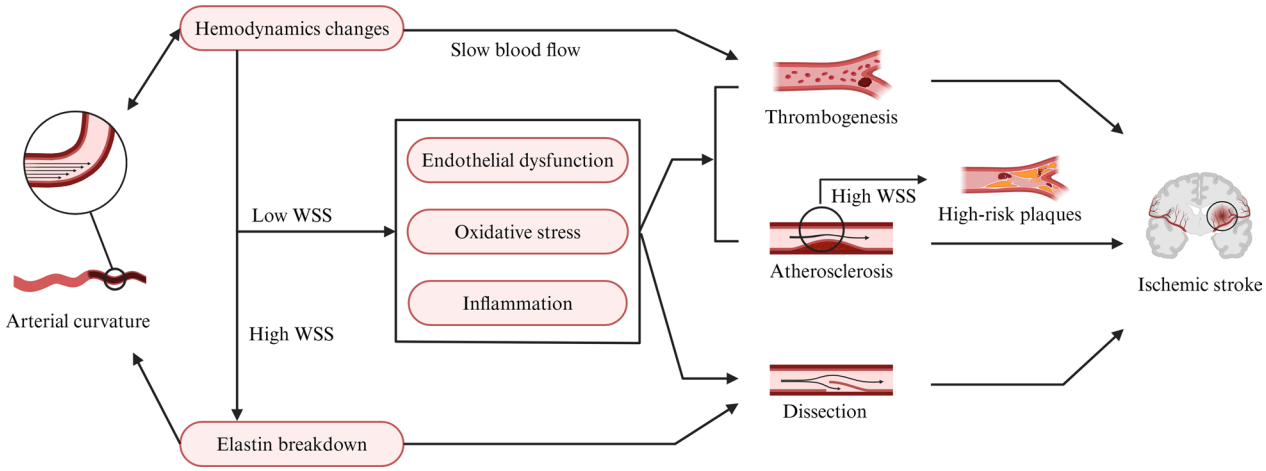

Fig. 3 Pathophysiological mechanisms linking arterial curvature and stroke. Arterial curvature results in haemodynamic changes, causing slowed blood flow in the curved artery. This can lead to inadequate blood supply to brain tissue and an increased risk of intravascular thrombus formation. Changes in wall shear stress (WSS), particularly low WSS, can result in endothelial dysfunction, oxidative stress, and inflammatory responses, all of which contribute to thrombosis and atherosclerosis. High WSS associated with atherosclerosis-related arterial stenosis may destabilize atherosclerotic plaques, potentially triggering acute vascular events. Additionally, elevated WSS may enhance fibrin degradation, contributing to the development of arterial dissection. Ultimately, thrombogenesis, atherosclerosis, and arterial dissection can lead to acute ischemic stroke. (Created with BioRender.com.)

hypertension as a risk factor, should be categorised as high-risk for stroke. Moreover, patients with stroke and a high degree of arterial curvature should be evaluated for the potential causes of arterial dissection. Further exploration of arterial curvature could provide valuable insights for clinicians managing patients with curved vessels.

Abbreviations

ICA	Internal carotid artery
BA	Basilar artery
VA	Vertebral artery
DFM	Distance factor metric
SOAM	Sum of angular measures
PAD	Product of angle and distance
ICM	Inflection point counting metric
TI	Triangular index
RL	Relative length
BL	Band length
ATS	Arterial tortuosity syndrome
LDS	Loeys-Dietz syndrome
LOPD	Late-Onset Pompe disease
MCA	Middle cerebral artery
WSS	Wall shear stress
MMP	Metalloproteinases
TIMP	Tissue inhibitor of matrix metalloproteinases

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L.Z. performed the literature search and wrote the first draft of manuscript and drew the figures and generated table. F.C. performed the literature search and supervised the writing and edited the manuscript. All authors have read and agreed to the published version of the manuscript.

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The authors declare no competing interests.

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