Endovascular Management of Intracranial Atherosclerosis

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KEYWORDS

• Intracranial atherosclerotic disease • Endovascular • Stroke • Stenosis • Angioplasty • Stenting
• Percutaneous transluminal angioplasty and stenting

KEY POINTS

- Intracranial atherosclerotic disease (ICAD) is responsible for a considerable proportion of ischemic strokes worldwide.
- The clinical presentation of ICAD is heterogeneous and may involve more than 1 mechanism.
- Delineating the mechanism of ischemia requires careful clinical analysis, and usually necessitates multimodal imaging.
- Conservative medical management is the appropriate first step in the treatment of ICAD.
- An endovascular treatment approach based on the mechanism of stroke may be beneficial for select patients.
- Patient selection will be a critical factor in the design of future ICAD clinical trials.

INTRODUCTION

Epidemiology and Natural History

A common cause of stroke worldwide, intracranial atherosclerotic disease (ICAD) is most prevalent in Black, Asian, and Hispanic populations.[1] In the United States, ICAD was found in an estimated 10% of stroke patients, whereas in Asia ICAD accounts for approximately 30% to 50% of all strokes.[2] Risk factors for ICAD include age, hypertension, smoking, diabetes mellitus, hypercholesterolemia, and metabolic syndrome.[3] Although the high rate of certain uncontrolled risk factors, such as diabetes mellitus, hypertension, and hyperlipidemia, may partially account for the increased incidence of ICAD in African Americans,[4,5] the rates of these risk factors do not differ significantly in the Chinese population in comparison with Caucasians, and thus do not account for the significant burden of ICAD in this population.[6]

Data from the randomized, double-blind, controlled trial Warfarin versus Aspirin for Symptomatic Intracranial Disease (WASID) revealed that patients with symptomatic ICAD carry a high risk of subsequent stroke.[7] Despite the use of aspirin and management of risk factors, patients with a recent transient ischemic attack (TIA) or stroke and a stenosis of 70% or greater had a 23% risk of stroke at 1 year.[7,8]

Clinical Manifestations

Intracranial atherosclerotic disease presents with ischemic stroke or TIA, which may be single or recurrent.[9] Depending on the stroke location, there

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can be various clinical presentations including isolated motor or sensory involvement and/or cortical function impairments.[10–12] In addition, cognitive deficits, such as impairment of executive function and anterograde amnesia, can occur especially with infarcts involving the anterior-medial thalamus, caudate nucleus, or areas of cerebral cortical or white matter.[13,14] White matter degeneration, hypoperfusion, and hypometabolism can lead to cognitive changes in the absence of infarcts.[15]

Mechanisms of Ischemia

Downstream ischemia from ICAD can be due to hypoperfusion, in situ thromboembolism, perforator orifice occlusion, or a combination of these mechanisms.[16,17] In situ thrombosis followed by distal arterial embolism, in addition to delayed washout of emboli resulting from hypoperfusion, can be present at the same time. Similarly, a combination of local branch occlusion and embolism, with or without hemodynamic compromise, can occur concurrently.[10,18]

Neuroimaging may aid in delineating the stroke mechanisms, although sometimes one imaging pattern can be produced by a combination of mechanisms. In general, border-zone infarcts are suggestive of hypoperfusion, territorial infarcts point to peripheral embolism, and deep subcortical infarcts indicate perforator artery orifice occlusion.[19,20] In a study investigating lesion patterns on diffusion-weighted imaging (DWI) for middle cerebral artery atherosclerotic disease, 15 (83.3%) of 18 patients with border-zone infarcts had concomitant infarcts suggestive of either peripheral embolism (territorial infarcts) or perforator artery involvement (subcortical infarcts), indicating the coexistence of multiple mechanisms.[10] Inferring the initial stroke mechanism is important, as it could be predictive of the risk of recurrent stroke or the mechanism of the next ischemic event. In an analysis of patients presenting with an index stroke in the WASID trial, the risk of recurrent stroke was similar in patients who presented with lacunar and nonlacunar strokes, and recurrent strokes in patients presenting with lacunar stroke were typically nonlacunar.[21]

DIAGNOSIS

The goals of imaging are: to detect intracranial stenosis with high sensitivity; to ascertain the degree and length of stenosis; to differentiate the atherosclerotic stenosis from mimics such as a recanalized partial thrombus, intracranial dissection, or nonatherosclerotic vasculopathy; and to assess the state of collateral circulation.

Detection and Quantification of Stenosis

Digital subtraction angiography (DSA) is considered the reference standard for the evaluation of intracranial stenosis. The high resolution of DSA allows for excellent quantification of luminal stenosis. Calculation of the degree of stenosis on DSA uses the equation $[1 - (D_{stenosis}/D_{normal})] \times 100$, where $D_{stenosis}$ is the diameter of the artery at the site of most severe degree of stenosis and D_{normal} is the diameter of the proximal normal artery (Fig. 1).[22]

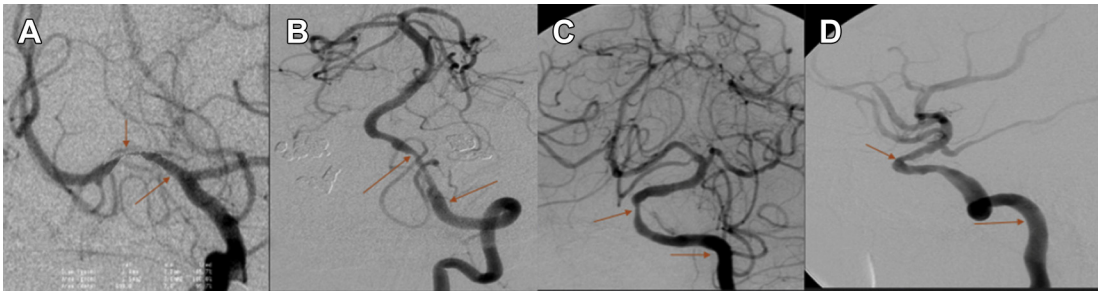

Fig. 1. Measurement of intracranial stenosis using the Warfarin versus Aspirin for Symptomatic Intracranial Disease (WASID) method. The diameter of the proximal part of the artery at its widest, nontortuous, normal segment is chosen (first choice), as shown in *A* and *B* (proximal *arrows* reference the normal vessel diameter; distal *arrows* reference the area of stenosis). If the proximal artery is diseased, the diameter of the distal portion of the artery at its widest, parallel, nontortuous normal segment is substituted (second choice; not shown). If the entire intracranial artery is diseased, the most distal, parallel, nontortuous normal segment of the feeding artery is measured (third choice), as in *C* (the proximal *arrow* denote the normal vessel diameter to compare the stenotic diameter to). For internal carotid artery disease involving the precavernous, cavernous, and postcavernous segments, the petrous carotid segment with parallel margins is measured at its widest, nontortuous, normal portion, as shown in *D* as the reference diameter (the proximal *arrow* in *D*). If the entire petrous carotid is diseased, the most distal, parallel part of the extracranial internal carotid artery is substituted (second choice; not shown). The distal most *arrows* in all figures denote the area of maximum narrowing.