Management of Acute Ischemic Thrombosis

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KEYWORDS

• Acute ischemic thrombosis • Occlusive cerebrovascular disease • Stroke
• Intracranial atherosclerotic disease • Perfusion imaging

KEY POINTS

- Noninvasive perfusion imaging has become important for the management of stroke in evaluating time-to-peak, cerebral blood volume, and cerebral blood flow in acute ischemic stroke and penumbra regions.
- Endovascular techniques for acute ischemic thrombosis include aspiration through a large-bore catheter (known as the A Direct Aspiration first Pass Technique [ADAPT]), the use of a stent retriever, and a combination of the 2 techniques (known as Solumbra or Trenumbra).
- Management of acute ischemic thrombosis in neurosurgical patients requires early detection and aggressive intervention. The use of perfusion imaging studies to discern viable tissue is important in understanding the risk–benefit analysis.

INTRODUCTION

The brain is the organ in the body that is most vulnerable to hypoperfusion. Ischemic changes happen quickly because of the demand of the brain for oxygen and glucose. Exclusive aerobic metabolism and high metabolic rates are needed to maintain ionic gradients and constant synaptic activity. This organ comprises only 2% of the human body weight but uses 18% of the cardiac output.[1] Because of this, even small fluctuations generate significant changes in electrochemical gradients and neuronal cell functioning.

Several mechanisms that protect cerebral blood flow (CBF) include multiplicity of supply via collateral blood vessels and the Circle of Willis and physiologic local perfusion matching and autoregulation to areas of increased metabolism. In settings of acute ischemic thrombosis, understanding the dynamic nature of CBF and its relationship to cerebral blood volume (CBV) is crucial in identifying viable brain tissue (salvageable penumbra) that is at risk.[2] In cases of acute ischemic thrombosis, the interventionist must consider physiologic responses of blood vessels and compensatory mechanisms such as autoregulation while trying to improve CBF and CBV.

PATHOPHYSIOLOGY

The 2 most significant pathophysiological causes of acute ischemia are atherosclerotic disease

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and thromboembolism. Low-density lipoproteins (LDLs) and triglycerides play a large role in atherosclerotic disease. The plaque initially forms as a fatty streak secondary to elevated LDL cholesterol deposition that initiates the immune response of macrophages, leading to a proinflammatory state. LDL oxidation leads to incorporation of cholesterol deposits into the subendothelial layer of the blood vessels. Injury to the intimal layer from accumulation of these deposits can cause platelet aggregation and thrombus formation that may lead to stenosis of the vessel.[3] Rupture of plaques can lead to acute ischemic events as well as occlusion at the site of the stenosis.

Another common cause of acute ischemia involves thromboemboli leading to acute occlusions. Atherosclerotic disease of proximal vessels and atrial fibrillation are typical causes of acute ischemia. Both of these causes can potentially lead to thromboemboli, which when distributed more distally, can impede blood flow in the cranial circulation. In addition, other sources of thromboemboli include fusiform aneurysms, arterial wall dissections, trauma-causing blunt vessel injury, traumatic injury to vessels, hypercoagulable states, and postoperative hypercoagulability, all of which contribute to higher risks of acute arterial intracranial thrombosis.[4] Appropriate postoperative anticoagulation for known hypercoagulable states is recommended, typically focusing on antithrombin and antifactor Xa therapies, discussed in other articles in this edition.[5]

In addition to these causes of acute ischemia, surgical procedures that involve manipulation of arteries and endovascular procedures can increase the risk of thromboembolic complications. Surgical bypass procedures, endarterectomies, and other procedures involving manipulation of the intracranial vasculature predispose vessels to arterial thrombosis. Vessel injury, flow stasis, turbulent blood flow, and immobility of the patient can also predispose vessels to thrombosis. Surgeons performing open vascular procedures need to consider the contact of suture lines or surgical material including patch grafts and other grafts with the circulation. Endovascular procedures have an inherent risk of arterial thrombosis of nearly 1% per procedure and up to 5% to 7% for interventions.[6] Antiplatelet therapy can help prevent intraprocedural thrombosis, because the mechanism for thrombosis is believed to be secondary to platelet aggregation. Anticoagulation therapy, including systemic heparinization during procedures, is standard for most endovascular interventions and helps to reduce the risk of thrombosis by acting on the intrinsic coagulation cascade, which may be activated when the plastic or metal components of the endovascular catheters and devices are in contact with blood for extended periods of time.

DIAGNOSIS

Patients with acute arterial thrombosis tend to present with significant symptomatic findings on clinical examination. Symptoms referable to motor and sensory areas of the cortex indicate risk for cerebral ischemia or cerebral hypoperfusion.[7] Even in neurosurgical patients, clinical findings can be localized, and prognosis can be partially understood by determining the National Institutes of Health Stroke Scale (NIHSS) for symptoms that last more than 15 minutes. A baseline neurologic examination and NIHSS score are paramount for patients who undergo neurosurgical procedures, because understanding postoperative changes in clinical findings and the score may play a role in determining whether surgical intervention is warranted.[8]

Initial imaging modalities include noncontrast computed tomography (CT) of the head to exclude intracranial hemorrhage or postoperative causes for the changes in the neurologic examination. Early changes may be noted near the gray–white matter junction, basal ganglia, and insular cortex in nearly 50% of cases.[9,10] The Alberta Stroke Programme Early CT Score (ASPECTS) was established to guide revascularization efforts in acute stroke patients.[9] The ASPECTS investigators found that patients with scores less than 7 were noted to have a higher risk of symptomatic hemorrhage with thrombolytic therapy and lower modified Rankin Scale (mRS) scores at 3 months. Further studies demonstrated the potential importance of CT angiography to demonstrate arterial anatomy and suggest potential targets for revascularization.[2] Thrombolytic therapy for treatment of acute ischemic thrombosis is not a common option in postsurgical patients because of the risk of hemorrhage.

The capabilities of CT angiography were further extended with CT perfusion imaging technology. At the authors' institute, CT perfusion imaging is used to guide endovascular and open vascular revascularization decisions.[11] Determining whether increased time to peak correlates with areas of decreased CBF and CBV can delineate areas of brain that are salvageable versus areas that demonstrate core infarct.[12] The risks of endovascular or surgical revascularization in the presence of a core infarct are concerning because of a higher risk of reperfusion injury and hemorrhage.[13,14] Patients with small areas of core infarct but with a large ischemic penumbra can benefit from prompt surgical intervention.