Complications and Solutions with Carotid Stenting

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KEYWORDS

• Carotid artery stenting • Complications • Embolic protection devices

KEY POINTS

- Complications of carotid stenting can be classified as neurologic, cardiovascular, death, carotid, access site, device malfunctions, and general and late complications.
- The risk of most complications is related to readily identifiable patient and anatomic factors.
- Management and outcome of complications require immediate recognition and a team-based approach to patient care.

INTRODUCTION

Patient selection for percutaneous and surgical revascularization for carotid artery stenosis is highly dependent on assessment of the risk/benefit ratio. Carotid artery stenting (CAS) may be associated with a variety of complications (**Box 1**); some are similar to complications arising after carotid endarterectomy (CEA), and some are unique to CAS. Since the introduction of CAS more than 20 years ago, there has been a decline in the risk of most complications due to improvements in operator experience, patient selection, technique, and equipment. Accordingly, this article focuses on the most mature CAS experience when possible, highlighting robust data from large randomized trials and registry studies that included more than 1000 patients, mandatory use of embolic protection devices (EPDs), independent neurologic assessment before and after CAS, and adjudication of all major events by an independent clinical events committee.

NEUROLOGIC COMPLICATIONS

Because the primary purpose of carotid revascularization is stroke prevention, it is compelling that the most important and dreaded complication is stroke. Most published studies of CAS enrolled specific patient populations designated as standard risk (patients considered reasonable candidates for CEA) or high risk (patients considered high risk for CEA on the basis of established clinical and anatomic criteria). The reported risk of stroke for standard and high-risk patients is 1.0% to 4.8%, including ipsilateral major stroke in 0.5% to 2%, ipsilateral minor stroke in 0.5% to 2.9%, and other types of stroke (contralateral or bilateral) in 0.3% to 0.4% (**Table 1**). In the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST), 2502 standard-risk patients were randomized to CAS with distal EPD (n = 1262 patients) or CEA (n = 1240 patients). The risk of procedure-related stroke after CAS (defined as stroke within 30 days) was 4.1%, which included major ipsilateral stroke in 0.9%, minor ipsilateral stroke in 2.9%, and other types of stroke in 0.3%.[1] In another study of 1300 unselected standard-risk and high-risk patients, the risk of procedure-related stroke after CAS with proximal EPD was 0.9%, equally divided between major and minor stroke.[2] Among 4 large registries of high-risk patients, the risk of stroke was 2.8% to 4.8%, including major ipsilateral stroke in 0.8% to 2.0%, minor ipsilateral stroke in 1.4% to 2.9%, and other strokes in 0.4%.[3–5]

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Box 1
Complications of carotid artery stenting

- Neurologic complications
  - Transient ischemic attack
  - Ischemic stroke
  - HPS
  - ICH
- Cardiovascular complications
  - VVRs
  - VDRs
  - AMI
- Death
- Carotid artery complications
  - Dissection
  - Perforation
  - Thrombosis
  - Vasospasm
  - Injury to ECA
- Device malfunction
  - Stent migration
  - Stent deformation
- General complications
  - Access site injury
  - Contrast nephropathy
- Late complications
  - Restenosis
  - TVR
  - Stent fracture
  - Death
  - Stroke
  - Cognitive dysfunction

Most neurologic complications associated with CAS and CEA can be broadly classified as ischemic injury, hyperperfusion syndrome (HPS), and intracranial hemorrhage (ICH), depending on the primary cause (Table 2).[6] This classification is somewhat arbitrary, however, because these conditions often coexist in the same patient. CREST investigators performed a detailed analysis of clinical and imaging characteristics of patients who developed procedure-related stroke (Table 3).[7] Clinical characteristics included major and minor strokes in 20.8% and 79.2% of patients, respectively, and 91.7% were identified as ischemic strokes and 8.3% as ICH. Although more than 90% of neurologic deficits were located in the ipsilateral cerebral hemisphere, 9.2% of patients had bilateral or contralateral deficits, suggesting that atheroembolism may originate from the aortic arch and brachiocephalic circulation. Furthermore, catheter or guide wire manipulation, placement and retrieval of EPDs, balloon inflation, and stent deployment may cause cerebral embolization. Imaging findings indicate that 83% of abnormalities involve the anterior circulation, 3% involve the posterior circulation, and 14% involve both circulations. Patterns of ischemic injury include scattered emboli in 38%, isolated cortical infarction in 31%, subcortical infarction in 17%, and bilateral infarction in 14%. These data indicate that most strokes after CAS are minor ischemic ipsilateral injuries, but there is a potentially broad spectrum of clinical and imaging abnormalities.

Approximately two-thirds of strokes after CAS occur during the day of the procedure[7]; most of these are minor and are due to atheroembolism. Complete or partial occlusion of a filter EPD is an important cause of acute neurologic injury that occurs during the CAS procedure and results from accumulation of atherosclerotic debris in the EPD. Clinically, filter occlusion may be indistinguishable from atheroembolic stroke, but this distinction is important because patients with filter occlusion usually respond immediately to filter aspiration and removal. Filter occlusion can usually be distinguished from other causes of cerebral ischemia by angiographic demonstration of impaired blood flow at the level of the filter itself.[8] In contrast, acute intracranial embolization during CAS is usually established by clinical examination of the patient and by anteroposterior and lateral projections of the intracranial circulation with digital subtraction angiography. Angiographic findings may include abrupt cutoff of one or more branches of the anterior or middle cerebral artery or more subtle wedge-shaped defects in the brain blush, visualized best in a lateral projection.

When acute neurologic injuries are recognized after CAS, activation of an institution's comprehensive stroke team is recommended, and individuals with expertise in the management of acute stroke should be consulted immediately. CT imaging of the brain is required to exclude ICH; further treatment recommendations depend on the imaging findings and on the extent of neurologic impairment.

Most strokes that occur on the day of CAS are minor and do not require acute stroke intervention. In contrast, major strokes, including ICH, tend to occur several days after CAS.[7] HPS and ICH are