Anesthesiology
1998; 89:1273–5

A Devastating Complication of Inadvertent Carotid Artery Puncture

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CAROTID artery puncture is a recognized complication of internal jugular vein catheterization. We report a rare case of fatal thromboembolism of the internal carotid artery resulting from this fairly common complication.

Case History

A 59-yr-old obese man was admitted to hospital with a history of sudden onset of retroorbital pain and signs consistent with subarachnoid hemorrhage. Medical history was unremarkable, as were laboratory investigations. Computed tomography of the head showed diffuse subarachnoid hemorrhage, and angiography showed a 1-cm right posterior communicating artery aneurysm. Mild atherosclerotic disease of the basilar artery, the left vertebral artery, and the cavernous portion of the right internal carotid artery also was noted. The patient proceeded to surgery for clipping of the right posterior communicating artery aneurysm.

After induction of anesthesia and intubation, a 7-French polyurethane, two-lumen indwelling catheter was inserted into the right internal jugular vein using the Seldinger technique. This procedure was technically difficult because of a large roll of adipose tissue around the patient's neck. The internal carotid artery was punctured on two occasions using a 20-gauge needle. Firm pressure was applied to the carotid puncture site for 3–5 min on each occasion, which prevented hematoma formation. The operation then proceeded without difficulty, and the aneurysm was clipped successfully. During the procedure, the internal carotid artery was noted to be atheromatous and calcified at the level of the clinoid process. Postoperatively, the patient was extubated within 30 min of the end of surgery and was obeying commands. Over the next 3–4 h, the patient's neurologic condition deteriorated, and he became unconscious, responding only to painful stimuli. Computed tomography of the head showed early signs of an extensive infarction within the distribution of the internal carotid and right posterior communicating arteries, which subsequent computed tomography scans over the next 24 h showed to evolve. Subsequent angiography 12 h postoperatively showed occlusion of the distal right internal carotid artery and the middle cerebral artery with intraluminal thrombus (fig. 1). A major right cerebral hemisphere infarction developed subsequently in the patient. His condition declined rapidly, and he died 72 h postoperatively.

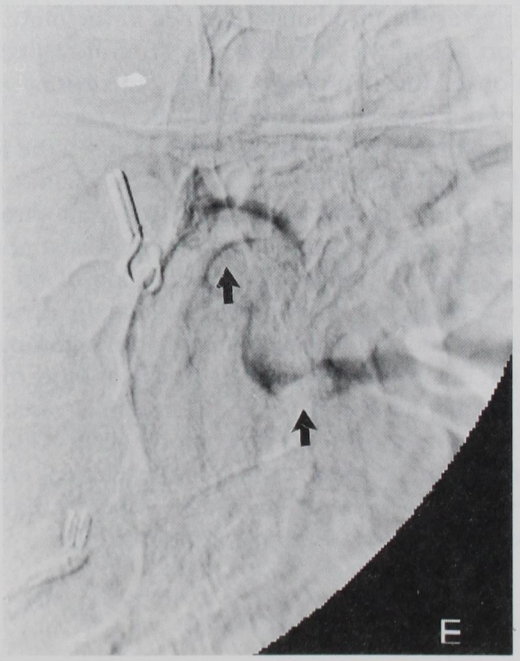

Fig. 1. Right common carotid arteriograms taken 12 h postoperatively. Anterioposterior views show complete occlusion of the supraclinoid internal carotid artery with tubular intravascular filling defects in the cervical and cavernous segments representing intraluminal thrombus (arrows).

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Received from the Departments of Anesthesia, Pathology, Neurosurgery, and Diagnostic Radiology, University Hospital, London, Ontario, Canada. Submitted for publication January 22, 1997. Accepted for publication June 11, 1998.

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Key words: Carotid artery thrombosis; central venous catheter; cerebral aneurysm surgery.

Pathologic Findings

Inspection of the circle of Willis revealed a 1.0-cm ruptured aneurysm at the junction of the right internal carotid and posterior cerebral arteries. A straight Sugita aneurysm clip was noted across the neck of the aneurysm. During gross inspection, the right frontal lobe was soft and dusky, and microscopic inspection confirmed the existence of a massive recent infarct.

Focal hemorrhage was noted in the adventitia of the right cervical internal carotid artery immediately distal to the carotid bifurcation. Microscopic inspection of transverse sections through the artery at this level revealed a radially oriented needle track associated with adherent thrombus on the luminal aspect of the vessel in the vicinity of this lesion. Notably, the thrombus was covered by a single layer of endothelial cells, confirming its occurrence well before death. It did not occlude the arterial lumen at this level and could be traced cephalad for approximately 2 cm. The more distal aspect of the cervical internal carotid was patent and free of adherent thrombus. In contrast, immediately distal to the neck of the aneurysm, transverse sections through the internal carotid artery displayed a large thrombus, also covered by a single layer of endothelial cells. Histologic appearance was identical to and, by inference, occurred at the same time as the thrombus described in the more proximal portions of the carotid artery. The most likely explanation of the neurologic events culminating in the death of this patient was that direct percutaneous injury to the proximal internal carotid artery caused the formation of a mural thrombus in the immediate vicinity of the lesion. Part of this remained as an adherent thrombus immediately cephalad to the bifurcation. Distal portions of the thrombus embolized to the region of the aneurysm, where they became impacted along the right distal internal carotid and middle cerebral artery causing massive right hemispheric infarction. The possibility that the thromboembolic material in the intracranial vasculature arose as a consequence of flow abnormalities related to the aneurysm clipping cannot be excluded entirely. However, this is extremely unlikely in the context of an identical-type lesion at the carotid bifurcation.

Discussion

At our institution, an internal jugular catheter is commonly used during cerebral aneurysm surgery to allow intraoperative monitoring and also to facilitate fluid-loading therapy should postoperative vasospasm occur. Carotid artery puncture is one of the most common technical complications during internal jugular vein cannulation, with a frequency varying between 2% and 14%, depending on the technique and the experience of the operator.[1,2] However, most of the literature discusses the hemorrhagic sequelae of this complication.

Anagnou[3] compiled data regarding six cases of hemiparesis after carotid puncture during internal jugular vein catheterization. The mean age of these patients was 68 years, and manual compression was applied to the neck in all to prevent hematoma formation; five of six cases were fatal. In all but one patient, there was evidence of arteriosclerosis.

The current case is similar to the six cases compiled by Anagnou[3] in that there was evidence of atherosclerosis in the cerebral vessels. Although the cervical internal carotid artery was free from atherosclerosis, there was disease affecting the carotid artery at the site of the fatal embolus. This was noted angiographically and clinically during the operation. Studies in dogs[4] found that, where arterial injury was superimposed on arterial stenosis, platelet deposition and thrombosis were enhanced compared with vessels in which there was no stenosis. It is possible therefore that patients with atherosclerosis have an increased risk of cerebral artery thrombosis or embolism, or both, associated with carotid artery puncture.

Blunt and penetrating trauma to the neck and pharynx both can cause thrombosis of the internal carotid artery.[5–8] Martin *et al.*[9] reported a series of eight patients with cervical injury, and, of these, four had thrombotic occlusion of the carotid artery. Two of these patients were treated successfully by arterial reconstruction, and the others recovered with nonoperative treatment. The authors suggested carotid ultrasonography as a useful noninvasive imaging technique for assessing these patients.

Manual strangulation also was reported as a rare cause of carotid thrombosis,[10] and it is possible that excessive pressure applied to the patient's neck to prevent hematoma formation may have contributed to the thrombotic process.

The consequences of inadvertent carotid artery puncture may therefore be devastating, and it is worth considering the course of action that should be taken to reduce the risk of thromboembolic complications.

In the current case, trauma to the artery with a small needle initiated the thrombotic process. It is possible that excessive manual external compression and the presence of atherosclerotic disease all contributed to the

acute pathologic process. Patients undergoing cerebral aneurysm surgery also are often fluid depleted, and this may also have contributed to a hypercoagulable state.

Removal of the needle from the carotid artery and manual compression of the puncture site is the correct procedure to follow because this reduces the risk of hematoma formation.[11] After thromboembolism was diagnosed, the use of thrombolytic agents or heparinization may have been considered. However, in this case, by the time the diagnosis was made, cerebral infarction was well established; and these agents may not be recommended after cerebral aneurysm surgery. Surgical exploration and removal of the thrombus would have constituted a major risk for the patient.

Thromboembolism may occur up to 48 h after trauma to the carotid artery.[7] When inadvertent carotid puncture occurs, firm but not excessive manual compression is essential to prevent the more common hemorrhagic complications. However, patients with an increased risk of thromboembolic complications, such as those with preexisting atherosclerosis, *e.g.*, a carotid bruit, and those with a hypercoagulable state, may also be at risk of thromboembolism. In these cases, close monitoring for neurologic sequelae for up to 48 h is suggested, and carotid ultrasonography may also be a useful noninvasive method of assessing these patients.

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