Internal Carotid Artery Dissection After Remote Surgery

Iatrogenic Complications of Anesthesia

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Background
Subintimal dissection with acute occlusion of the internal carotid artery resulting in acute cerebral infarction has not been reported as an iatrogenic complication of general anesthesia.

Case Descriptions
An anesthetist stretched the neck of a 44-year-old man by anchoring an anesthesia mask posterior to the angle of the jaw with overlying fingers as the patient struggled during an insufficient inhalational mask anesthetic. A 33-year-old man received an endotracheal anesthesia without struggle, but as he was turned from a supine to a prone position, his head and neck were not immobilized to rotate with his torso during the move. The next day both patients suffered acute cerebral infarctions secondary to ICA dissections and occlusions (angiographically demonstrated).

Conclusions Stretching the soft neck tissues of anesthetized patients can cause internal carotid artery dissection and acute cerebral infarction. (Stroke. 1994;25:1276-1278.)

Key Words • anesthesia • carotid arteries • cerebral infarction • dissection

No report in the literature links the position of an anesthetized patient's head and neck or the forcible holding of an anesthesia mask to a struggling (insufficiently anesthetized) patient with the complication of carotid artery intimal tearing, dissection, and occlusion. The complication of carotid artery occlusion and cerebral infarction typically is delayed after the initial carotid artery injury and the beginning of dissection and may therefore divert attention from the physical rendering of a general anesthetic as the cause of such events. The following accounts describe the circumstances preceding these adverse events.

Case Reports

Case 1
A 44-year-old man weighing 185 lb, normotensive and without systemic disease, underwent surgical removal of a cactus thorn from his hand. An initial 5 mg atracurium and 350 mg thiopental followed by 100 mg succinylcholine was administered intravenously. An anesthesia mask was held in place on the patient's face by the anesthetist's left hand. Laryngoscopy, attempted once, could not expose the larynx, although the patient's anatomy was normal. Tracheal intubation was not pursued. Oxygenation was well maintained, as confirmed by pulse oximetry. Nitrous oxide (50%) and inspired enflurane (2%) were administered through the mask, with an oral airway in place. Upon surgical incision the patient moved strenuously. Assisted ventilation through the mask was maintained, while the patient produced high-pitched vocalization, another manifestation of a light plane of anesthesia. The patient's head (ie, occiput) was firmly restrained on the operating table under the anesthesia mask held by the anesthetist's left hand. Sufentanil and more atracurium were given intravenously to deepen the anesthesia.

After surgery the patient awakened and complained of pain on the left side of his neck. No external abnormalities were apparent, and he was released from the hospital. Eleven hours later he suddenly became aphasic and had right hemiparesis and right central facial palsy. On his immediate return to the hospital, carotid angiography was performed that demonstrated an occluded left internal carotid artery. The pattern of a conical, tapering lumen produced by subintimal dissection started at the level of the third cervical vertebra; flow ceased at C-2 (see Figure). No filling of the left carotid siphon was seen from antegrade flow or from collateralized filling retrograde to the ophthalmic artery. The aortic arch and other cervical vessels were normal; computed tomography (CT) of the head also appeared normal.

Heparin therapy was given after transfer to another hospital. Right-sided hemiparesis resolved in 2 days; intellectual function and fluent speech did not recover. CT of the brain then revealed acute infarction of the left temporal and parietal lobes in the territory supplied by the middle cerebral artery. Repeat carotid arteriography 6 days after the acute event demonstrated persistent occlusion of the left internal carotid artery and collateral blood supply to the left cerebral hemisphere from the anterior and posterior communicator branches. The patient requires continuous custodial care.

Case 2
A 33-year-old normotensive man, 6' 6" tall, with unconfirmed suspicion of Klinefelter syndrome (XXY chromosome and variants) and symptomatic lumbar disc herniation, was anesthetized for lumbar laminectomy in the prone position. Laryngoscopy and tracheal intuba-
tion were unremarkable; hemodynamics remained stable throughout the anesthesia. The patient’s recovery from the anesthetic was uneventful until the next day, when he suddenly developed right hemiparesis.

Left internal carotid artery dissection was confirmed by immediate angiography; CT of the brain showed ischemic infarction of the territory of the left middle cerebral artery. The patient has a permanent right hemiparesis and Broca’s aphasia.

Discussion

The mechanism of hyperextension of the neck with axial rotation of the head and neck is the proposed mechanism of injury to the carotid artery that causes the dissections described above. In case 1 the forceful restraint of the patient’s head as his torso strained against the operating table may well have stretched the internal carotid artery “across the transverse process of the third cervical vertebra or the bony mass of the first and second cervical vertebrae. This results in rupture of the intima and media at the level of the third cervical vertebra with preservation of the more elastic adventitia.”2 The fourth and fifth fingers of the left hand holding the anesthesia mask might also directly traumatize the artery, as they anchor the mask by extending over the mandible and encroaching on the carotid bifurcation.

In case 2 the tracheal intubation entailed no apparent difficulties, and an adequate depth of anesthesia was maintained. Unrecognized movement of the neck when the patient was turned to the prone position or when the head and neck were positioned with the patient prone (such as 90° axial rotation or extension instead of flexion) may have caused internal carotid artery damage. The potential for injury to the carotid artery exists from “cervical rotation and hyperextension,”3 perhaps enhanced for anesthetized patients unprotected by normal muscle tone. Reports of patients with Klinefelter syndrome with ruptured cerebral aneurysms,4,5 coupled with a propensity for gravitational leg ulcers at an early age, “lend support to the concept of a more fundamental vascular abnormality” in this disorder.6 The second patient might have been at increased risk from anesthesia positioning because of this constitutional factor.

The possibility that tracheal intubation might produce carotid artery dissection9 or vertebral artery dissection10 also has been suggested. The case report of a 44-year-old woman with left internal carotid artery dissection and cerebral infarction attributes the catastrophe to childbirth.7 However, her hemiparesis occurred 3 hours after general anesthesia for cesarean section.

A wide variety of physical stresses on the carotid artery capable of causing its dissection have been identified.11,12 Manual manipulation of the neck has caused carotid artery9 and vertebral artery10 dissection. Volitional strenuous neck movement also can produce carotid artery dissection.11 The complaint of neck pain, voiced by the patient described above in case 1, is infrequent in this entity; headache is a commonly experienced symptom.12 Although one survey of carotid dissection reports “an excellent or complete recovery” in 85% of the cases,12 a larger series emphasizes the likelihood of significant morbidity.13 Spontaneous dissections of cervical internal carotid arteries, causing cranial nerve palsies, have recently been reported.14,15 Catastrophic cerebral infarction was not a feature of these patients.14,15 The yearly incidence of spontaneous internal carotid artery dissection is estimated to be 2.6 per 100 000.16 Only one third of those affected in the survey16 had ischemic strokes, and the majority of these patients made a complete neurological recovery.16

Intraoperative brain infarctions carry a graver prognosis. Postsurgical infarctions of the brain stem and cerebellum arising in cardiovascular surgical subjects from cardiac or aortic embolization, or in noncardiovascular surgical subjects from “position-related vertebral artery thromboses,” produced severe neurological impairment in all cases.17

Two examples of acute dissection of left internal carotid arteries with irreversible ischemic cerebral infarction from occlusion following general anesthesia are newly presented. Vigilance against operating room conditions capable of causing carotid artery intimal tears recommends itself. Surveillance for the briefly delayed clinical presentation of this complication will define its true incidence as a complication of mask anesthesia administration and the turning and malpositioning of an anesthetized patient (possibly with an additional risk factor, as in case 2) lying prone on the operating room table.

References

Internal carotid artery dissection after remote surgery. Iatrogenic complications of anesthesia.
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