Acute spontaneous otorrhagia resulting from a ruptured petrous carotid aneurysm

Case report

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A 35-year-old man experienced sudden spontaneous arterial bleeding from his left ear. Arteriography demonstrated a left petrous carotid aneurysm. The bleeding was initially controlled with packing and ultimately by left common carotid ligation and transection.

Key Words • temporal bone • carotid aneurysm • carotid occlusion • carotid artery • petrous carotid

Aneurysms of the petrous portion of the internal carotid artery are rare lesions. Approximately 20 cases have been reported in the literature and include fatal results from exsanguinating hemorrhage. Reports on the management of these cases are necessary to establish the most direct and safest therapy.

Case Report

This 35-year-old right-handed iron foundry worker was seen in the emergency room. He was bleeding profusely from his left ear. The bleeding was initially controlled by packing Surgicel deep into the external auditory canal followed by Vaseline gauze. Compression was maintained with a tight mastoid dressing. Transfusion of four units of whole blood was required to reverse the hypotension, after which his hematocrit was 34%.

He had noted a loss of hearing in his left ear for more than 17 years. In the 3 months before admission he vigorously irrigated the external canal with peroxide in an attempt to “remove the wax.” On the evening of admission he was seated watching television when he heard a loud “popping sound” in his left ear and then noticed blood spurting out “like a water pistol.” His efforts to control the bleeding with his finger in the external canal or by covering the ear with a towel were futile. Approximately 20 minutes after the bleeding started, it was controlled in the emergency room. At that point all features of his neurological profile were normal with the exception of audition on the left, which could not be satisfactorily evaluated.

Examination. The general physical examination was similarly unremarkable except for a well healed fracture deformity of the left ankle. Further questioning disclosed that a 25-year-old brother had died of an intracerebral hemorrhage. There was a personal history of excessive alcohol consumption and hypertension.

Plain radiographs of the skull showed clouding of the petrous portion of the left temporal bone. Tomography demonstrated that the carotid canal coursed lateral to its normal position, and delineated a 5-mm ovoid lucency (Fig. 1) compatible with the angiographic finding of an aneurysm at the genu of the left petrous carotid artery protruding into the middle ear (Fig. 2, left). Irregularity of the arterial wall in the carotid canal was suggestive of the presence of granulation tissue and/or periadventitial infection (Fig. 2). Cross compression on selective right carotid injection showed a hypoplastic left A1 segment, but excellent filling of the candelabra branches of the left middle cerebral artery. Both anterior cerebral arteries filled only on right carotid injection. An aberrant occipital artery arose from the left internal carotid artery just above the bifurcation, and cephalad narrowing of that vessel suggested spasm (Fig. 2). No other aneurysms were apparent.

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Fig. 1. Tomography of the right and left petrous bones in the anteroposterior projection. The letters "ICA" (internal carotid artery) delineate the carotid canal. The ovoid lucency (arrows) represents the bone defect housing the petrous carotid aneurysm.

Computerized tomography (CT) scan demonstrated changes in the left petrous bone which represented either extravasated blood or granulation tissue.

A second episode of bleeding occurred when the patient coughed violently and dislodged the packing. This was controlled with Vaseline gauze tightly packed into the external canal and a mastoid dressing. Two additional units of packed red blood cells were needed to bring his hematocrit to 38%. He was begun on ampicillin, 500 mg/6 hours. His hypertension was well controlled with Inderal (propranolol hydrochloride) and Aldomet (methyldopa). His tendency to bleed, evidenced by hematoma formation following transfemoral angiography, was attributed by the hematologist to folate deficiency associated with consumption of ethyl alcohol, and platelet deficiency following multiple transfusions. A Matas test was performed three times daily for 10-minute intervals during the 4 days before surgery. He remained completely asymptomatic during the externally applied common carotid compression.

Operation. Left common carotid ligation and transection was carried out. After exposure of the carotid artery under general anesthesia, he was awakened. Heparin, 2500 units, was given intravenously. The common carotid was then clamped for 30 minutes during which there were no neurological abnormalities. When it was certain that he tolerated the clamping without incident, anesthesia was restarted, and the common carotid was doubly ligated and transected. When he awoke, there was no deficit of motor or sensory function, and his speech was normal.

Postoperative Course. In the evening after surgery, there was another episode of otorrhagia, probably due to dislodgement of the pack during coughing at the time of extubation. This was readily controlled with a tight mastoid dressing. When the packing was removed on the seventh postoperative day, no further bleeding occurred. Microscopic visualization of the middle ear showed destruction of the tympanic membrane and small amounts of granulation tissue. There was a nubbin of clotted blood which was thought to be associated with the aneurysm. This was not disturbed. The patient was begun on a second 10-day course of ampicillin, and Cortisporin drops were instilled into the canal daily. Audiometric testing showed a conductive hearing loss without evidence of sensory neural damage.

Discussion

Aneurysms of the petrous portion of the internal carotid artery may be congenital, mycotic, or posttraumatic. Rupture of the aneurysms may occur spontaneously, presenting with profuse epistaxis via the eustachian tube, or with otorrhagia. Rupture may also be the result of manipulation as during suboccipital exploration, or tympanotomy for middle ear masses presumed to be glomus tumors.

Congenital aneurysms could be expected to arise at the junction of the internal carotid artery, and any of its branches in the carotid canal which include the caroticotympanic, the Vidian, the periosteal, and the persistent stapedial artery. A direct origin from the side of the internal carotid artery was seen by Guirguis.
FIG. 2. Left common carotid subtraction angiograms. Left: Anteroposterior projection demonstrating the aneurysm (white arrows) and irregularity of the petrous carotid (black arrows) suggesting periadventitial infection. Right: Lateral projection showing the aneurysm (white arrow) and irregularity of the petrous portion of the carotid artery (black arrows).

and Tadros, and they speculated that it was the result of incomplete involution of an embryonic branch. Mycotic aneurysms in this location are probably not of hematogenous origin, but rather secondary to periadventitial infection of the carotid artery due to communication with the middle ear or the eustachian tube; reported cases had evidence of purulent otorrhea or cholesteatoma antedating the aneurysm. Post-traumatic aneurysms of the internal carotid artery are associated with basal skull fractures in most instances.

There is strong presumptive evidence for a congenital origin of our patient's aneurysm in view of the family history of intracerebral hemorrhage and the specific denial of head trauma. In light of the irregularity of the petrous portion of the internal carotid artery on angiography, it is conceivable that an infectious process might have converted a berry aneurysm into a mycotic aneurysm, thereby predisposing it to rupture.

Control of the hemorrhage was achieved with common carotid ligation and transection. We favor this procedure over gradual occlusion with a Crutchfield or Selverstone clamp. Common carotid ligation was used successfully in five previous instances. We believe that it carries a lower morbidity than internal
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carotid occlusion, and it affords an opportunity at a later date to perform muscle embolization and entrapment procedures should they be required.

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References


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