Detachable latex balloon occlusion of an internal carotid artery with an aberrant branch in a horse with guttural pouch (auditory tube diverticulum) mycosis

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EQUINE

- Severe, often fatal, hemorrhage from the internal carotid artery is common in horses with guttural pouch (auditory tube diverticulum) mycosis.
- Guttural pouch mycosis affecting the internal carotid artery may be treated successfully with ligation of the proximal portion of the artery and occlusion of the distal portion of the artery with a detachable latex balloon.
- Aberrant branches of the internal carotid artery may originate more distally than expected; the use of angiography to identify these vessels prior to occlusion of the internal carotid artery is warranted.

A 5-year-old 480-kg (1,056-lb) Thoroughbred gelding was referred to the Marion DuPont Scott Equine Medical Center for evaluation and treatment of intermittent epistaxis of unknown cause. The horse had bled 6 times during the 2-week period prior to referral, including an episode of severe hemorrhage on the day of admission. Previous epistaxis ranged from mild to profuse and was unilateral, from the left nostril, or bilateral, predominately from the left nostril.

At referral, the gelding was calm, alert, and in good body condition. Rectal temperature was 37.3 C (99.2 F), heart rate was 44 beats/min, and respiratory rate was 24 breaths/min. Oral mucous membranes were pale, and capillary refill time was within reference range. Fresh and dried blood was observed in both nares. Endoscopic examination of the upper portion of the respiratory tract revealed a blood clot in the pharyngeal ostium of the left auditory tube. Examination of the left guttural pouch (auditory tube diverticulum) was prevented by a large blood clot within the pouch. Results of examination of the right guttural pouch were unremarkable. Red blood cell count (5.23×10^6 cells/µl [reference range, 6.7 to 10.0×10^6 cells/µl]), hemoglobin concentration (8.5 g/dl [reference range, 11.2 to 18.8 g/dl]), PCV (24.8% [reference range, 33.0 to 45.5%]), and plasma protein concentration (4.2 g/dl [reference range, 5.5 to 7.2 g/dl]) were less than reference values. Total WBC count (9,100 cells/µl [reference range, 5,000 to 13,500 cells/µl]) and differential counts were within reference limits. Blood from the gelding was crossmatched with that of resident blood donors.

On the basis of history, clinical findings, and results of endoscopic examination, a tentative diagnosis of mycosis of the left guttural pouch was made. Treatment options were discussed, and the owner elected initial medical stabilization, followed by surgical occlusion of the left internal carotid artery by ligation of the proximal portion of the artery and occlusion[1] of the distal portion of the artery with a detachable latex balloon. Medical treatment included administration of 2 L of equine plasma and aminocaproic acid (10 g in 1 L isotonic saline [0.9% NaCl] solution, IV, q 8 h until surgery). By 12 hours after admission, PCV had decreased to 13%, and 8 L of whole blood from a compatible donor was administered IV. The PCV increased slightly, to 17%, within 12 hours after the transfusion and remained stable over the next 12 hours. At this time, 42 hours after referral, the gelding's condition was considered stable enough for general anesthesia and surgical occlusion of the left internal carotid artery.

Preoperative treatment included administration of a single dose of flunixin meglumine (1.1 mg/kg [0.5 mg/lb] of body weight, IV) and ceftiofur sodium (4.4 mg/kg [2.0 mg/lb], IV, q 12 h, for 2 days). The gelding was sedated by administration of xylazine hydrochloride (0.5 mg/kg [0.23 mg/lb], IV). Anesthesia was induced with guaifenesin (10% solution, to effect, IV) and ketamine hydrochloride (2.2 mg/kg [1 mg/lb], IV) and maintained with halothane in oxygen. The horse was positioned in right lateral recumbency. Lactated Ringer's solution (11 ml/kg/h [5 ml/lb/h], IV) was administered during the anesthetic period.

The left parotid region was clipped and prepared for surgery. A 10-cm modified hyovertebrotomy incision was made cranioventral to the left wing of the atlas. The origin of the left internal carotid artery was identified and exposed by careful dissection. A Rumel tourniquet was placed 1 cm distal to the origin of the artery, occluding normograde blood flow. A loop of umbilical tape was placed around the artery, 3 cm distal to the tourniquet, to aid in exteriorization and stabilization of the vessel without occluding the vessel.

Access to the interior of the vessel was gained by use of a modified Seldinger technique.[1] An 8-F introducer sheath[a] was placed in a distal direction between

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the tourniquet and the distal loop of umbilical tape. Retrograde blood flow was observed through the introducer sheath, indicating patency of the distal portion of the internal carotid artery. An 8.5-mm detachable latex balloon[b] was mounted onto a 2-F carrier catheter[c] contained within an 8-F guide catheter.[d] The catheters were advanced through the introducer sheath to the proximal bend of the sigmoid flexure of the left internal carotid artery by use of fluoroscopic guidance. The carrier catheter was advanced 3 mm while the guide catheter was withdrawn 5 mm, thereby exposing the balloon within the lumen of the artery. The balloon was inflated with 0.5 ml of a 1:1 solution of isotonic saline solution and iohexol.[e] Appropriate positioning and inflation of the balloon were confirmed by fluoroscopy. The balloon was detached by applying gentle traction on the carrier and guide catheters. The catheters were withdrawn from the introducer sheath, and retrograde blood flow was assessed. Continued retrograde blood flow was identified by bleed-back through the introducer sheath.

Subsequent fluoroscopic examination confirmed complete inflation and correct positioning of the balloon within the sigmoid flexure of the internal carotid artery. An angiographic study confirmed complete occlusion of the distal portion of the internal carotid artery at the sigmoid flexure and revealed a small aberrant branch of the left internal carotid artery, which arose from the caudal aspect of the artery (**Fig 1**). The study was performed by injecting 30 ml of a 1:1 solution of iohexol[e] and isotonic saline solution into the introducer sheath within the internal carotid artery during fluoroscopy. The aberrant branch was located proximal to the suspected site of the mycotic lesion, approximately midway between the origin of the internal carotid artery and the sigmoid flexure. On the basis of results of the angiographic study, communication of the aberrant branch with the cerebral arterial circle was identified, and the aberrant branch of the internal carotid artery was determined to be the source of retrograde blood flow.

By use of fluoroscopic guidance and angiography, a second balloon was placed 1 cm distal to the aberrant branch (Fig 1). Angiography was performed after the balloon had been inflated to confirm effective occlusion of this segment of internal carotid artery, which was suspected of being affected by the mycotic lesion. The carrier, guide catheters, and introducer sheath were withdrawn. Ligatures of size-0 polypropylene[f] were placed proximal and distal to the site of vascular access, replacing the tourniquet and loop of umbilical tape. The incision was closed in routine fashion.

The gelding recovered from anesthesia without complications, but 1 hour after recovery, the gelding began pawing and sweating profusely. Physical examination revealed heart rate of 60 beats/min, hind limb stiffness, and moderately firm gluteal muscles. Serum activities of aspartate aminotransferase (685 U/L [reference range, 226 to 320 U/L]) and creatine kinase (16,348 U/L [reference range, 172 to 396 U/L]) were increased. Postanesthetic myopathy was suspected, and balanced polyionic fluids were administered IV (5 L/h for 4 hours, then 100 ml/kg/24 h [45.5 ml/lb/24 h]

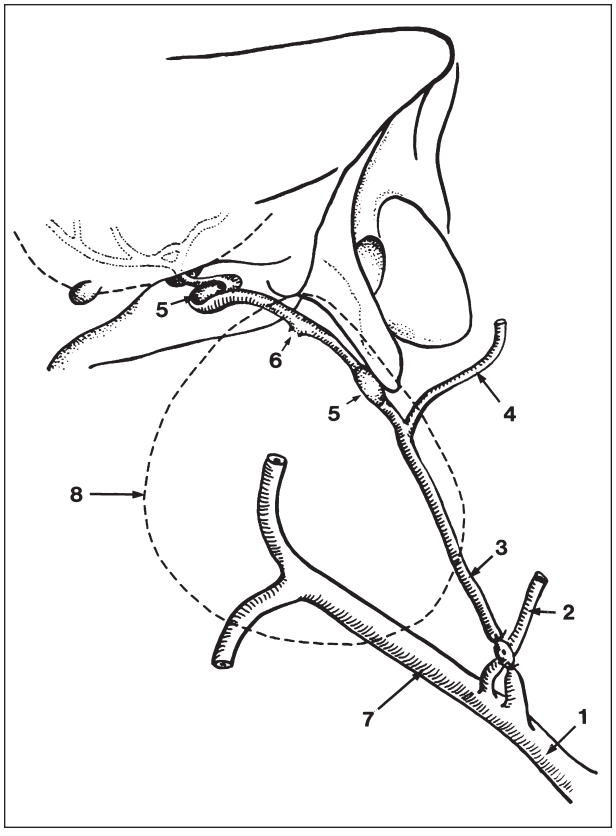

Figure 1—Illustration of the left internal carotid arterial tree of a horse with guttural pouch (auditory tube diverticulum) mycosis. Notice placement of 2 detachable balloons, the site of the mycotic lesion, and the origin of an aberrant branch of the left internal carotid artery. 1 = Left common carotid artery. 2 = Left occipital artery. 3 = Left internal carotid artery. 4 = Aberrant branch. 5 = Detachable balloons. 6 = Site of the mycotic lesion. 7 = Left external carotid artery. 8 = Outline of the guttural pouch.

for 8 hours). Acepromazine maleate was administered IV at 2 (25 mg), 7 (10 mg), and 11 (10 mg) hours after recovery. Dimethylsulfoxide (90 g in 1 L isotonic saline solution, IV) was administered 2 and 26 hours after recovery. Phenylbutazone (2.2 mg/kg [1.1 mg/lb], PO, q 12 h) was administered for 5 days. Twelve hours after recovery, all physical examination findings returned to reference limits, and clinical signs of postanesthetic myopathy were not observed.

Radiographic examination of the guttural pouch region 2 days after surgery revealed that both balloons were inflated and in their original location (**Fig 2**). Endoscopic examination of the left guttural pouch revealed a mycotic plaque in the caudodorsal aspect of the medial compartment, which involved the left internal carotid artery. The left internal carotid artery lacked a visible pulse; a small dilation of the artery was visible on the caudal aspect of the medial compartment at the site of the proximal (second) balloon. Further complications did not develop, and the gelding was discharged 6 days after surgery.

Endoscopic examination of the left guttural pouch 30 days after surgery revealed that the mycotic plaque was decreased in size by approximately 85%. Minimal inflammation within the pouch was observed; the left internal carotid artery was yellow-gray and reduced in

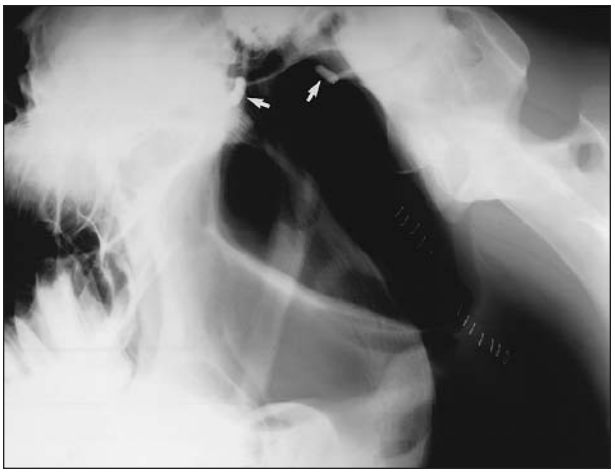

Figure 2—Lateral radiographic view of the parotid region of a horse with guttural pouch mycosis. Radiography was performed 2 days after surgery to confirm inflation and position of 2 detachable balloons (arrows) that were placed within the left internal carotid artery. The rostral balloon is located between the proximal and distal bends of the sigmoid flexure, and the caudal balloon is located between the mycotic lesion and an aberrant branch of the left internal carotid artery.

size. Radiographic examination confirmed that both balloons were inflated and in their original positions. Six months after surgery, endoscopic examination revealed complete resolution of the mycotic plaque. Follow-up 1 and 2 years after surgery did not reveal complications or adverse effects of balloon placement, and the horse was showing successfully.

Severe, often fatal, hemorrhage from the internal carotid artery is common in horses with guttural pouch mycosis.[2-5] The objective of recommended treatment is physical occlusion of the internal carotid artery. Simple ligation of the proximal portion of the artery at its origin[4,6,7] and a combination of proximal ligation and balloon-tipped catheter occlusion of the distal portion of the artery have been described in affected horses.[8,9] A combination of proximal ligation and detachable latex balloon occlusion of the distal portion of the artery has also been described in clinically normal horses.[1] To our knowledge, successful occlusion of an internal carotid artery with an aberrant branch has not been reported.

Typically, the internal carotid artery in horses originates as 1 of the 3 terminal branches of the common carotid artery and terminates as a portion of the cerebral arterial circle, giving rise to the caudal intercarotid artery, caudal communicating branch, middle cerebral artery, and rostral cerebral artery. The occipital artery originates just proximal to the origination of the internal carotid artery and anastomoses with the vertebral artery. The internal carotid and occipital arteries may arise as a common trunk that usually divides within a short distance[10]; however, anatomic anomalies of the internal carotid artery are generally thought to be uncommon.

Although the internal carotid artery in horses is commonly described as an unbranched vessel throughout its extracranial course, it may have a branch (corticobasilar artery) that arises from the distal bend of the sigmoid flexure and passes through the foramen lacerum to communicate with the basilar artery.[10,11] A suspected aberration of this branch that resulted in treatment failure was identified in a mare with guttural pouch mycosis affecting the internal carotid artery.[12] The mare began to hemorrhage during recovery from occlusion of the distal portion of the internal carotid artery with a balloon-tipped catheter and was subsequently euthanatized because of uncontrollable hemorrhage. At necropsy, the catheter was found to have entered the aberrant branch that originated proximal to the sigmoid flexure and mycotic lesion, and thus prevented occlusion of the distal portion of the artery.

Bifurcation of the internal carotid artery was detected in 5 of 37 horses undergoing carotid or cerebral angiography.[13] Bifurcation was also detected in 4 horses by endoscopic examination of the guttural pouches.[13] Two authors (HSC, RSP) of the study reported here have observed branching of the internal carotid artery as it coursed through the guttural pouch, during terminal carotid angiography or complete dissection of the internal carotid artery at necropsy.

Complete absence of the normal left internal carotid and occipital arteries was identified in a gelding with guttural pouch mycosis that affected what appeared to be an aberrant branch of the left common carotid artery.[14] The aberrant branch originated slightly more proximally on the common carotid artery, compared with the typical site for origination of the right internal carotid and occipital arteries. The aberrant vessel coursed along the caudal aspect of the medial compartment of the guttural pouch and terminated on the basilar artery just proximal to the origin of the caudal cerebellar arteries. During occlusion of the anomalous vessel, the catheter made contact with the base of the cerebellum and resulted in apparent cerebellar artery vasospasm and cerebellar necrosis, which led to respiratory failure and death.

The successful outcome of treatment for the horse reported here can be partly attributed to intraoperative identification of the aberrant branch of the internal carotid artery. Continued retrograde blood flow was identified, and subsequent angiography revealed the aberrant branch. A second balloon was easily placed between the branch and the suspected site of the mycotic lesion, which eliminated blood flow to that segment of artery. We chose to place a second balloon to complete occlusion of the internal carotid artery rather than attempt to identify and ligate the aberrant branch by further dissection. If angiography is not available and retrograde hemorrhage is identified, further dissection of the internal carotid artery may be beneficial for identifying an aberrant branch. However, further dissection risks entering the guttural pouch or disrupting the internal carotid artery at the site of the lesion.

The aberrant branch identified in the horse of this report was not the occipital artery branching from a common trunk, because the origin of the occipital artery was identified during the initial surgical dissection. Also, the aberrant branch likely did not represent an anomaly of the corticobasilar artery, although this possibility could not be excluded with certainty, because hard copies of the angiograms were not made,

and a postmortem examination was not performed. Using real-time angiography, we clearly identified the origin of the aberrant branch but could not determine the exact terminal course of the vessel. The aberrant branch did not appear to pass through the foramen lacerum but seemed to course caudal to the occipital condyles; however, angiography performed after the initial balloon occlusion did reveal cerebral vasculature, indicating the branch communicated with the cerebral arterial circle. We suspect this communication involved an extracranial anastomosis with the basilar artery.

The horse of this report developed mild postanesthetic myopathy that resolved completely with treatment. Factors thought to contribute to development of postanesthetic myopathy include duration of the surgical and anesthetic period, anesthetic drugs selected, body weight and nutritional status, position and padding during surgery, dehydration, hemorrhage, hypoxemia, hypotension, and poor tissue perfusion.[15] Hemorrhage and duration of anesthesia may have contributed to development of postanesthetic myopathy in the horse reported here. The PCV before induction of anesthesia was 17%, duration of anesthesia was 185 minutes, and duration of surgery was 135 minutes; difference between duration of surgery and anesthesia was largely related to initial fluoroscope setup. Approximately 40 minutes of the surgical time was attributable to the unanticipated finding of the aberrant arterial branch and preparation for placement of a second balloon. Angiography accounted for approximately 5 minutes of surgical time. Had the aberrant branch been identified prior to placement of the first balloon, approximately 30 minutes of surgical time may have been eliminated by preparing for placement of 2 balloons.

Because aberrant branches of the internal carotid artery may be more common than previously thought, we recommend performing intraoperative angiography prior to vascular occlusion for treatment of guttural pouch mycosis. Angiography is simple to perform with or without fluoroscopic capabilities, and when planned for, the time required for angiography is minimal. To expedite angiography performed with conventional radiographic techniques, an 11 × 14-in radiograph cassette, centered under the guttural pouch region, should be placed prior to draping; the radiograph machine should be ready in the surgical suite. The angiogram should be performed as soon as vascular access is gained, and the occlusion procedure should proceed while the radiograph is processed. This angiographic technique is efficient and may eliminate potentially fatal complications such as catheter malpositioning or postoperative hemorrhage caused by incomplete occlusion of the distal portion of the artery.

[a]Pinnacle Introducer Sheath, Medi-tech, Natick, Mass.
[b]Gold Valve Balloon, GVB 17, Laboratoiries Nycomed SA, Paris, France (North American distributor: Yocan Medical Systems, Thornhill, ON, Canada).
[c]Mini-Tourquer, CIFN 135, Laboratoiries Nycomed SA, Paris, France (North American distributor: Yocan Medical Systems, Thornhill, ON, Canada).
[d]Cyber, 8-F Gen 55 cm, Medi-tech, Natick, Mass.
[e]Omnipaque 240, Sanofi-Winthrop, New York, NY.
[f]Prolene, Ethicon, Somerville, NJ.

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