Surgical treatment of progressive ethmoidal hematoma aided by computed tomography in a foal

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- Progressive ethmoidal hematoma (PEH) is uncommon in horses and unlikely to develop in foals.
- In foals, PEH may result in atypical clinical signs because of rapid enlargement of the mass, extent of facial deformity, and minimal epistaxis and intraoperative hemorrhage.
- Use of computed tomography may aid in successful removal by altering the approach for surgical treatment of PEH.

A 4-week-old 104-kg (229-lb) Belgian filly was admitted for evaluation of facial enlargement over the left paranasal sinuses, respiratory stridor, and epistaxis. At birth, a firm swelling was noticed over the left caudal maxillary sinus, which progressively enlarged. Two days before admission, the facial swelling rapidly enlarged and was associated with respiratory stridor, a scant serosanguineous discharge from the left nostril, and mucopurulent discharge from the right nostril.

Physical examination revealed that the foal was in good body condition, was suckling without difficulty, and heart rate (84 beats/min), respiratory rate (32 breaths/min), and rectal temperature (38.9 C [102 F]) were within reference ranges. Mucous membranes were pink, capillary refill time was < 2 seconds, and hydration status was considered normal. There was extensive bulging of the nasal and maxillary bones over the left paranasal sinuses (Fig 1) and reduced airflow through the left nostril. Values obtained from a CBC and measurement of serum creatinine and γ-glutamyltransferase concentrations were within reference ranges. The foal had moderate hyperfibrinogenemia (9.0 g of fibrinogen/L; reference range, 1.0 to 5.0 g/L).

Endoscopic examination of the nasal passages and nasopharynx revealed a smooth hemorrhagic mass 4 cm caudal to the nostril, completely obstructing the left nasal passage. The right nasal meatus was narrow because of deviation of the nasal septum. An increase in soft-tissue opacity was observed in the maxillary sinuses and nasal cavity on lateral, dorsoventral, and oblique radiographic views of the head and was associated with distortion of the surrounding bony structures, flattening and distortion of the roots of the maxillary premolar teeth, and deviation of the nasal septum to the right. Radiographic examination of the thorax revealed findings that were indicative of mild bronchopneumonia.

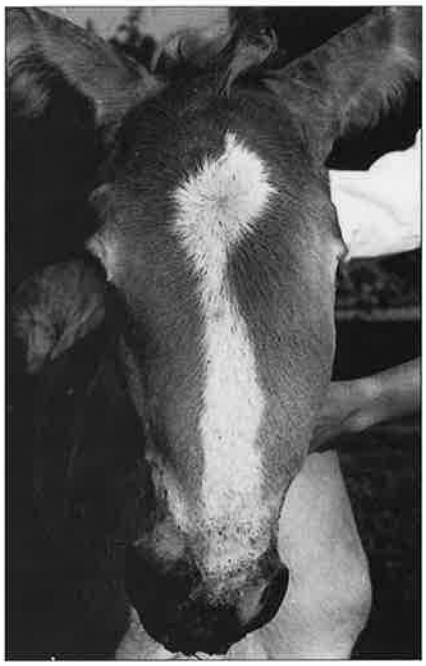

Figure 1—Photograph of a 4-week-old Belgian foal with prominent bulging of the nasal and maxillary bones over the left paranasal sinuses.

The foal was sedated with xylazine hydrochloride; general anesthesia was induced with ketamine hydrochloride and diazepam and was maintained with isoflurane and oxygen. Lactated Ringer's solution (25 ml/kg of body weight/h [55 ml/lb/h]) was administered IV before and during general anesthesia. **Computed tomographic (CT)** images of the head were obtained with the foal in right lateral recumbency. There was a large soft-tissue mass surrounding the left ethmoturbinates, occupying most of the left maxillary sinus and completely obstructing the left nasal passage. On progressive rostral images, lack of ethmoturbinates on the left side was observed, with distortion of the nasal septum and nasal turbinates and displacement of these structures to the right (Fig 2). A division was not found between the left nasal passage and left paranasal sinuses. Small pockets of gas and remnants of mineralized structures were observed in the soft-tissue mass. Fluid was observed in the left rostral and caudal maxillary sinuses and left frontal sinus surrounding the mass. There was osteolysis and dorsolateral displacement of the left nasal and maxillary bones and ventral displacement of the premolars and molars.

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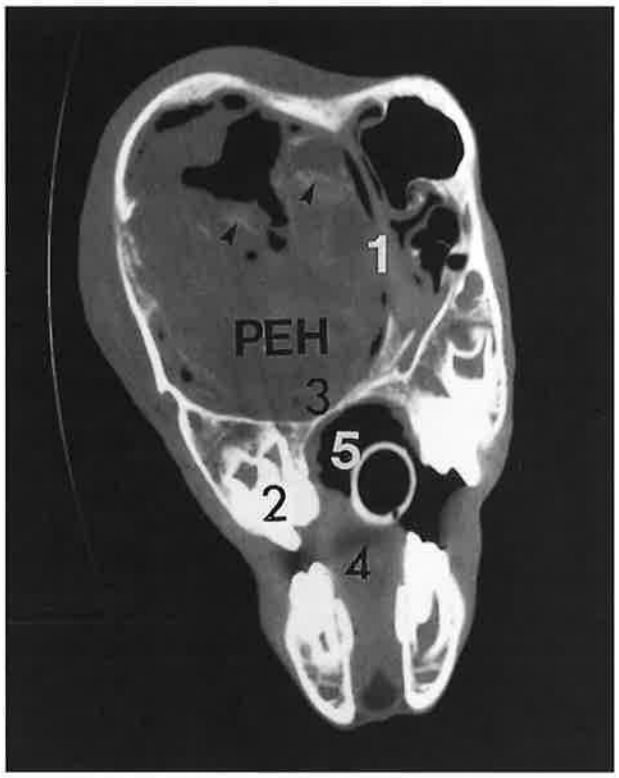

Figure 2—Transverse computed tomographic image of the head of the foal in Figure 1 obtained at the level of the second premolar. Notice the progressive ethmoidal hematoma (PEH) containing remnants of mineralized structures (arrowheads) in the left maxillary sinus, deviation of the nasal septum (1) to the right, dorsolateral displacement of the left facial bones and ventral displacement of the left upper cheek teeth (2). The hard palate (3), tongue (4), and endotracheal tube (5) are also indicated. Left side of image corresponds to left side of skull.

Potassium penicillin G (22,000 IU/kg [10,000 IU/lb], IV, q 6 h) and gentamicin (6.6 mg/kg [3.3 mg/lb], IV, q 24 h) were administered in preparation for surgical exploration. With the foal positioned in right lateral recumbency, the skin over the left paranasal sinuses was prepared for aseptic surgery. A 6-cm-wide by 10-cm-long frontonasal bone flap was made by incising the skin, subcutaneous tissues, and periosteum in the same plane and reflecting the edges of the periosteum for a width of 5 mm to expose bone, which was cut along 3 sides by use of an oscillating bone saw. The bone flap was based at the dorsal midline and elevated until it fractured.

Mucoid, serosanguineous fluid was aspirated from the sinus, and a soft, hemorrhagic mass, approximately 8 cm in diameter, originating from the sinus portion of the ethmoid labyrinth and extending into the adjacent sinuses and nasal passage was observed. The mass was freed from its attachments to the mucosal lining of the sinus by digital dissection and was removed with Foerster sponge forceps. The thickened, discolored mucosal lining of the caudal maxillary sinus was excised. The maxillary sinuses were distorted, and the nasomaxillary ostium formed a large fistula to the nasal cavity. Hemorrhage during surgery was minimal, and the sinuses were lavaged with sterile lactated Ringer's solution and packed with sterile gauze bandage, which was passed through the nasomaxillary opening, exteriorized, and sutured at the nostril.

Interrupted sutures of size-2 polydioxanone were placed to close the 2 free corners of the bone flap; the periosteum and subcutaneous tissues were closed in 2 layers with simple continuous sutures of 2-0 polydioxanone; and the skin was closed with staples. A tracheostomy tube was placed before recovery from general anesthesia. Butorphanol (0.1 mg/kg [0.045 mg/lb], IV) and flunixin meglumine (0.5 mg/kg [0.23 mg/lb], IV) were administered during and after surgery, respectively.

Potassium penicillin G (22,000 IU/kg [10,000 IU/lb], IV, q 6 h), flunixin meglumine (0.5 mg/kg [0.23 mg/lb], IV, q 24 h), cimetidine (8 mg/kg [3.6 mg/lb], PO, q 6 h), and sucralfate (3 g, PO, q 6 h) were administered for 3 days. Gentamicin (6.6 mg/kg [3.3 mg/lb], IV or IM, q 24 h) administration was continued for 10 days. The gauze packing was removed 24 hours after surgery. A small amount of serosanguineous discharge was observed draining from the left nostril, but this resolved 3 days after surgery. At that time, endoscopic examination revealed that the nasal passages were clear and the large nasomaxillary fistula was easily observed. The tracheostomy tube was removed, and the foal was discharged from the hospital without respiratory stridor.

Histologic examination of the mass that had been removed from the sinus revealed extensive areas of hemorrhage bordered by prominent areas of granulation tissue with neovascularization. Osteoblasts and osteoclasts were observed around focal spicules of mineralized bone, indicating bone remodeling. The thin capsule of the mass consisted of normal appearing respiratory epithelium and submucosal stroma. The histopathologic diagnosis was **progressive ethmoidal hematoma (PEH)**.

Thirty days after surgery, the maxillary sinus was examined endoscopically through the nasomaxillary fistula and evidence of recurrence of the PEH was not detected. Distortion of the facial bones had decreased. Three months after surgery, the referring veterinarian reported that the facial bones and nasal septum had returned to a normal contour and abnormal respiratory noise was not apparent during exercise. Twelve months after surgery, the facial appearance was normal and the abnormal endoscopic appearance of the ethmoid region was less obvious with return of the nasal septum to a normal position.

Progressive ethmoidal hematoma is a slowly enlarging, encapsulated, hemorrhagic mass that originates from the mucosal lining of the ethmoidal labyrinth in horses. The cause is unknown. Progressive ethmoidal hematoma is not neoplastic, but enlarges by fibrotic reaction in zones of repeated hemorrhage. Local pressure by the mass can cause erosion and resorption of bone. Progressive ethmoidal hematoma is uncommon and is reported to be more prevalent in Thoroughbreds and Arabians.[1] The lesion has been reported as developing in horses 3 to 20 years of age,[2-6] but, to our knowledge, has not previously been reported as devel-

oping in a neonate. Progressive ethmoidal hematoma can be unilateral or bilateral and can be located in the paranasal sinuses and nasal cavity.[7] When clinically apparent, PEH commonly fills the frontal and caudal maxillary sinuses with a smaller nasal component.[7] In the foal of our report, PEH involved the frontal and maxillary sinuses, nasal turbinates, and nasal passage. Compared with the effect of PEH in an adult horse, the effect of an enlarging mass in the skull of a foal is likely to result in greater deformity of the surrounding area because of the relatively softer bones.

Epistaxis is the most common clinical sign of PEH and is typically mild, intermittent, unilateral, and not associated with exercise.[8] Epistaxis results from hemorrhage caused by ulceration of the respiratory epithelium that covers the lesion and may be noticed as a sanguineous component of a serous or mucopurulent discharge.[1] Other clinical signs include abnormal respiratory noise resulting from nasal passage or pharyngeal obstruction, mucopurulent nasal discharge, coughing, facial deformity, malodorous breath, and head shaking.[2,4,6] Facial deformity and respiratory stridor were the predominant clinical signs in the foal of this report, and epistaxis was evident only as a scant serosanguineous discharge.

A tentative diagnosis of PEH can be made on the basis of history, clinical signs, findings on endoscopic examination, and radiographic findings. Definitive diagnosis is made on the basis of histologic appearance of the lesion. On endoscopic examination, the nasal portion of PEH may be seen protruding into the nasal passage or nasopharynx from the ethmoid area. Lesions vary from red-purple to green-yellow and have an irregular, bulb-shaped rostral surface that protrudes into the nasal cavity.[1] Progressive ethmoidal hematoma of the paranasal sinus, without an endoscopically visible nasal lesion, has also been described but is less common.[9-11] In the foal of our report, the mass extended far rostrally into the nasal passage and destroyed the ethmoid region.

On radiographic evaluation of the head, PEH may have the appearance of a soft-tissue opacity that involves the area of the ethmoturbinate, paranasal sinus, and nasal passage.[10] Positive-contrast sinusography has been described and may allow more complete evaluation of the extent of the lesion in the paranasal sinuses.[12] Obtaining a biopsy sample of the lesion by use of an endoscope or a small sinus trephine can assist in diagnosis, but the amount of tissue that can be obtained is often too small to be of diagnostic use. In a conscious horse, trephination into the paransal sinuses may allow direct examination of the ethmoid labyrinth by use of an arthroscope or a flexible pediatric endoscope.[13]

Computed tomography[14,15] and nuclear scintigraphy[11] have been used to assist in the evaluation of PEH. Computed tomography of the head has considerable advantages over other techniques, because structures are viewed without superimposition.[15] In the foal of this report, CT images of the lesion provided excellent contrast, which was used to discriminate between different tissue types. The CT images were used to determine the extent of the soft-tissue mass. On the basis of these images, the bone flap was much larger and more axially located than that typically made in an adult horse. This surgical approach permitted an opening that accommodated the surgeon's hand, which made PEH removal and debridement much easier.

Although the rate of progression is unknown, complete surgical removal or laser ablation of PEH is recommended.[11,16,17] Repeated partial removal of lesions to maintain an airway may be appropriate when there are economic constraints or short-term control is adequate. Surgical exploration via a frontonasal bone flap is recommended but may not allow accurate assessment of the origin and extent of the lesion, particularly with respect to the degree of involvement of the sphenopalatine sinus. Cryotherapy of the entire lesion, if small, or freezing the base of a lesion after debulking has been used as an adjunct to surgical removal of PEH.[2] Cryotherapy decreases intraoperative hemorrhage and may decrease the incidence of regrowth.[18] Successful treatment and decreased incidence of recurrence of the lesion depend on complete removal of the lesion along with any diseased sinus or ethmoid tissue. Use of a large frontonasal bone flap in the foal of this report allowed good exposure of the frontal sinus, ethmoid labyrinth, and maxillary sinus; therefore, adjuvant treatment was not needed.

Excessive hemorrhage is the main complication during surgery. Temporary bilateral occlusion of the common carotid arteries as a method of reducing hemorrhage during nasal and paranasal sinus surgery has been described[19]; however, it prolongs surgery time and has not been effective during surgical excision of PEH in some horses.[3,4] Use of electrocautery, gauze packing, cold saline (0.9% solution of NaCl)-soaked sponges, saline ice slush, epinephrine-soaked sponges, cryotherapy, and vascular clamps are other methods described to control hemorrhage during surgery of the paranasal sinuses.[6] Surgical removal of PEH in our foal was unusual, because hemorrhage during surgery was minimal. The possibility that the mass had outgrown its major blood supply was considered. The short surgical time was considered invaluable in minimizing blood loss, and gauze packing controlled postoperative hemorrhage.

The main postoperative complication is recurrence of PEH, which may follow incomplete or radical excision of the lesion.[2] A recurrence rate of 40 to 50% has been reported as regrowth, development of a contralateral PEH, or both.[2,3,6] Periodic endoscopic examination of nasal passages after surgery is important for early detection of lesion recurrence. Evidence of recurrence of PEH on radiographic and endoscopic examination was not apparent 12 months after surgery in the foal of this report. Surgical treatment of PEH was uncomplicated, and resolution of swelling and remodeling of the facial bones and nasal septum resulted in a good cosmetic result 3 months after surgery.

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Book Review:
Avian Medicine and Surgery. *By Robert B. Altman, Susan L. Clubb, Gerry M. Dorrestein, and Katherine Quesenberry. 1,070 pages; illustrated. WB Saunders Co, The Curtis Center, Independence Square West, Philadelphia, PA 19106–3399. 1997.*

The goal of the editors of this hardbound volume was to provide a user-friendly guide to avian medicine and surgery. More than 45 expert contributors helped achieve that goal. The book—divided into 9 sections—contains 58 chapters and 4 appendices. In section 1, authors discuss clinically normal birds, with chapters pertaining to systems, reproductive physiology, nutrition, laws and regulations, embryology, pediatric husbandry, behavior, aviculture medicine and flock health management, and nonsurgical methods of avian sex identification. Additional sections detail diagnostic procedures and techniques, emergency and supportive care, infectious and noninfectious diseases, pharmacologic considerations and therapeutics, surgery, specific species, and the human-avian bond. The most current information dealing with avian medicine and surgery is discussed and referenced, with numerous and appropriate references provided for each chapter.

There are a few grammatical errors and inconsistencies. For example, some reference names are misspelled, but these are minor problems in an otherwise good textbook. A few of the labeling schemes are a bit overwhelming in the chapter on radiology, which may confuse some readers.

Each chapter in the section on noninfectious diseases is designed to familiarize readers from a practitioners' standpoint with the anatomy, physiology, and disorders of the system being discussed. Numerous black-and-white photographs and line drawings compliment the text and generally provide sharp images of the material. The text is easy to read and interpret. Color figures are formatted at the beginning of the book and are of excellent quality. More than 120 color figures of ophthalmic conditions, endoscopic views of normal and abnormal findings, cytologic preparations, pathologic conditions, and candled-egg images are provided. It would have been nice to include color photographs with the text, but this would certainly have increased the price of the book.

Other chapters are equally informative and provide readers with appropriate information necessary to initiate or maintain quality veterinary care for avian species. In this regard, the book is a valuable reference for any avian veterinarian.

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