Traumatic pneumocephalus in a dog

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Case Description—A 17-month-old dog was evaluated because of progressive tetraparesis. The dog had a history of craniofacial trauma at 2 months of age.

Clinical Findings—Results of a neurologic examination were suggestive of a lesion localized to the medulla. Computed tomography revealed extensive pneumocephalus extending throughout the ventricular system and into the cranial cervical subarachnoid space.

Treatment and Outcome—Because of the deterioration in the dog’s clinical condition, an emergency bilateral transfrontal craniectomy was performed. A large amount of pyogranulomatous material was found intraprotestively. Neurologic and computed tomographic abnormalities were no longer evident during a recheck examination 8 weeks after surgery.

Clinical Relevance—Findings suggested that pneumocephalus should be considered in the differential diagnosis for dogs with neurologic signs of an intracranial abnormality, particularly if the dog has a history of craniofacial trauma. (J Am Vet Med Assoc 2009;234:1295–1298)

A 17-month-old 35-kg (77-lb) castrated male Golden Retriever was referred to Gulf Coast Veterinary Neurology for evaluation of progressive tetraparesis. The dog had been examined at a local emergency clinic 3 days earlier because of anorexia, lethargy, hind limb lameness, and a reluctance to move, and radiography of the vertebral column at that time had not revealed any clinically important abnormalities. Treatment with tramadol (2.6 mg/kg [1.2 mg/lb], PO, q 8 to 12 h) and strict rest were prescribed. The following day, the dog was examined by the owner’s regular veterinarian, who administered a single dose of dexamethasone (0.67 mg/kg [0.3 mg/lb], IV) and prescribed dexamethasone (0.03 mg/kg [0.01 mg/lb], PO, q 12 h), methocarbamol (20 mg/kg [9.1 mg/lb], PO, q 12 h), and doxycycline (3 mg/kg [1.3 mg/lb], PO, q 8 h). The dog’s anorexia resolved by the third day, but the other problems persisted.

According to the owner, the dog had sustained a dog bite to the head and face at 2 months of age. Skull radiography at that time revealed a fracture of the frontal bone over the frontal sinus and associated subcutaneous emphysema. The wound had been cleaned, and the dog had been treated with cefazolin (22 mg/kg [10 mg/lb], IV, once), dexamethasone (0.2 mg/kg [0.09 mg/lb], IV, once), and carprofen (2.2 mg/kg [1 mg/lb], PO, q 24 h for 10 days). The dog had reportedly recovered without complications, and no nasal discharge or neurologic abnormalities had been identified in conjunction with this injury.

On initial examination at Gulf Coast Veterinary Neurology, heart and respiratory rate and rectal temperature were within reference limits. The dog was obtunded and had opisthotonus and vertical nystagmus with positional left ventral strabismus. Menace and pupillary light responses were present in both eyes. A gag reflex was present, but jaw tone was reduced. Proprioceptive placing was difficult to assess because the dog was unwilling to bear weight on any of its limbs. Segmental spinal reflexes were considered normal. The limbs, face, and nose had a generalized reduced response to superficial sensation. Signs of pain were evident during palpation of the cervical region. An IV catheter was immediately placed, and mannitol (0.5 g/kg [0.23 g/lb], IV) was administered.

Results of the neurologic examination suggested that the dog had a lesion localized to the medulla. Possibilities that were considered included infection, inflammation, a congenital malformation, and neoplasia. The diagnostic plan included a CBC, serum biochemical panel, and computed tomography of the brain followed by collection and analysis of CSF.

Results of the CBC and serum biochemical panel were unremarkable. Anesthesia was induced with propofol (6 mg/kg [2.7 mg/lb], IV) and maintained with isoflurane. Lactated Ringer’s solution was administered throughout the anesthetic period (20 mL/kg/h, IV, for the first hour; then 10 mL/kg/h [4.5 mL/lb/h], IV). Computed tomography of the brain revealed soft tissue densities in the frontal sinuses with a fracture of the left frontal sinus wall laterally and of the cribriform plate and frontal bone medially (Figure 1). Air opacity was seen within the CNS with associated dilatation of the lateral, third, and fourth ventricles of the brain (Figures 2 and 3) and compression of the cerebrum and cerebellum. Air was also observed within the cranial cervical subarachnoid space (Figure 4). Rostrally, air within the fourth ventricle had caused substantial compression of the brainstem. Decompression was attempted by inserting a 22-gauge, 1.5-inch spinal needle in the cisterna magna; 8 mL of air was obtained, but no CSF could be aspirated.

Because of the rapid deterioration in the dog’s clinical condition, an emergency bilateral transfrontal craniectomy was performed. Phenobarbital (3 mg/kg, IV)
and methylprednisolone sodium succinate (30 mg/kg [13.6 mg/lb], IV) were given prior to surgery, and manni- nol (0.25 g/kg [0.11 g/lb], IV), cefazolin (22 mg/kg, IV, q 2 h), and metronidazole (14 mg/kg [6.4 mg/lb], IV) were administered during surgery. A modified bi-

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lateral transfrontal approach was used, as described,\(^1\) exposing both frontal sinuses and the frontal bone over the olfactory and frontal lobes of the brain. A large amount of brown-tinged pyogranulomatous material was identified within the left frontal sinus and removed with rongeurs, lavage, suction, and thumb forceps. The nasal turbinates, sinus mucosa, and dorsal aspect of the cribriform plate were also removed. Samples were sub-

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**Figure 1**—Transverse computed tomographic image at the level of the frontal sinus in a 17-month-old dog examined because of progressive tetraparesis. The dog had a history of craniofacial trauma at 2 months of age. There are soft tissue opacities in both frontal sinuses (asterisks) that are more severe on the left. There is a fracture of the left frontal sinus wall laterally (arrow) and the frontal bone medially (arrowhead) and erosion of the cribriform plate (not shown). The image is oriented so that the left side of the dog’s head is on the left side of the image.

**Figure 2**—Transverse computed tomographic image at the level of the thalamus in the dog in Figure 1. Notice the hypoattenuation and dilatation of the lateral ventricles bilaterally (asterisks) consistent with pneumocephalus.

**Figure 3**—Reconstruction of a sagittal computed tomographic image of the dog in Figure 1. Notice the areas of hypoattenuation throughout the ventricular system and extending into the cervical subarachnoid space (asterisk) and associated compression of the surrounding neural parenchyma. The areas of hypoattenuation are consistent with air.

**Figure 4**—Transverse computed tomographic image at the level of the first cervical vertebra in the dog in Figure 1. Notice the hypoattenuation within the subarachnoid space (asterisk) and severe compression of the spinal cord.
mitated for aerobic and anaerobic bacterial culture but did not yield any growth.

Exposure of the brain revealed that the left olfactory lobe was malacic, and affected brain tissue was removed until grossly normal brain tissue within the olfactory lobe was evident. All grossly abnormal and soft bone was removed with rongeurs, and the area was copiously lavaged. Hemostasis was achieved through a combination of bipolar electrocautery, absorbable gelatin compressed sponges, and polysaccharide powder. A fascial graft was obtained from the left temporal muscle fascia and placed over the frontal bone defect, with the edges tucked under the frontal bone. A second fascial graft was placed over this and ventral to the craniectomy site to seal the area and prevent air leakage. The frontal sinus cavity was then packed with absorbable gelatin sponges and autogenous fat obtained from a small incision in the dorsal cervical region. Polymethyl methacrylate was used to mold a diamond-shaped frontal plate to close the craniectomy defect and was secured in place with 2-0 nylon sutures at each corner.

A small incision was made at the rostral third of the temporal muscle, medial to the zygomatic process, and muscle was dissected away, allowing exposure of a small area of the frontal bone. A burr hole craniotomy was performed with a small bit attached to a pneumatic drill, and a 22-gauge, 1.5-inch needle was inserted. A total of 6 mL of air was suctioned from the ventricle, and the surgical site was closed in 3 layers.

The dog recovered from anesthesia without complications. Pain was managed with hydromorphone (0.1 mg/kg [0.045 mg/lb], IV, at the time of extubation, then 0.05 mg/kg [0.023 mg/lb], IV, q 4 h for 24 hours) and a fentanyl patch (30 µg/h, transdermal). Antimicrobial treatment was considered warranted on the basis of the gross appearance of the material in the left frontal sinus, and treatment with metronidazole (14 mg/kg, IV, q 12 h) and enrofloxacin (5 mg/kg [2.27 mg/lb], IV, q 12 h) was begun. Phenobarbital (3 mg/kg, IV, q 12 h) was administered prophylactically because of the possibility of postoperative seizures.

The dog’s neurologic condition improved daily after surgery, and results of a neurologic examination were normal with the exception of a slight left head tilt and a tendency to circle to the left at the time of discharge 4 days after surgery. Metronidazole (14 mg/kg, PO, q 12 h for 30 days), enrofloxacin (5 mg/kg, PO, q 12 h for 35 days), and phenobarbital (3 mg/kg, PO, q 12 h) were prescribed.

At the time of suture removal 2 weeks after surgery, the only neurologic abnormalities that were evident were a mild delay in proprioceptive placing of the left hind limb and an apparent reluctance to move the cervical portion of the spine. Results of a neurologic examination performed 1 month after surgery were normal. Computed tomography of the brain 8 weeks after surgery did not reveal any evidence of pneumocephalus, and the ventricular system of the brain appeared to be of normal size and shape with a normal CSF opacity. The polymethyl methacrylate plate appeared intact and in proper position.

**Discussion**

Pneumocephalus is an accumulation of gas within the cranial cavity. Areas where gas can accumulate include the epidural, subdural, subarachnoid, cerebral, and intraventricular regions. Pneumocephalus typically does not cause any clinical signs, unless the accumulated gas is extensive enough to cause an increase in intracranial pressure sufficient to affect the brain parenchyma. In human medicine, pneumocephalus has been associated with craniectomy, craniotomy, trauma, otitis media, evacuation of a subdural hematoma, cerebral laminectomy, ventriculoperitoneal shunting, and radiotherapy. Previously published reports of pneumocephalus in veterinary medicine all involved animals that had recently undergone cranial surgery, and to our knowledge, the present case represents the first report of tension pneumocephalus in an animal that was not a result of cranial surgery.

In humans, the incidence of post-traumatic pneumocephalus is far higher when there is a fracture involving the paranasal sinuses. Pathologic mechanisms that have been proposed for development of pneumocephalus following trauma and a fracture include ball-valve and hydrodynamic mechanisms. The ball-valve theory maintains that air is forced into the cranium through a defect in the sinus when air pressure in the sinus is higher than the intracranial pressure, such as during sneezing or coughing. The air then becomes trapped within the cranium when pressure within the sinus is less than the intracranial pressure. The hydrodynamic theory suggests that changes in posture result in pressure changes within the skull, allowing air to be sucked into the skull through a fracture line. A valve mechanism, like a flap of dura mater or brain tissue itself, prevents air from escaping. The high intracranial pressure results in reabsorption of CSF, allowing more air to enter as the cycle is repeated.

The exact cause of tension pneumocephalus in the dog described in the present report was not clearly understood. Possibly, there was a fracture of the frontal bone or cribriform plate that developed a thin callous of bone or scar tissue, and this covering was destroyed by an infectious process, allowing tension pneumocephalus to occur. Interestingly, the time from trauma to the onset of clinical signs of pneumocephalus was 15 months, suggesting that there was a slow leakage of air throughout this period or a sudden onset of leakage possibly associated with secondary sinus infection. A fracture of the frontal bone over the frontal sinus was diagnosed at the time of the dog bite; however, the true extent of the damage may not have been defined, because only skull radiography was performed. Computed tomography or magnetic resonance imaging of the skull is the preferred diagnostic modality to define the extent of injury after head trauma, but was not initially pursued in this dog because of the rapid clinical improvement and absence of neurologic abnormalities.

Management of tension pneumocephalus depends on the severity of clinical signs and the underlying cause. In human patients, the most common treatment is surgical decompression with correction of the underlying defect. Administration of oxygen at high concentrations prior to surgery and maintaining the patient’s head in a slightly lowered position have been recommended. The theory behind oxygen administration is that most of the gas trapped intracranially is nitrogen, making the return of gas to the paranasal sinuses more rapid.
and that increasing the inspired oxygen concentration should create a gradient that allows removal of nitrogen from the cranium, thereby reducing the volume of the trapped gas and the intracranial pressure. However, the possibility of oxygen toxicosis limits the time this technique may be used. Keeping the patient’s head in a slightly lowered position is theorized to reduce CSF drainage, which may decrease continued gas trapping. In human medicine, dural defects are typically repaired with muscle, fat, or mucoperichondrial flaps. Decompression can be achieved via needle aspiration, ventricular puncture, or catheter drainage. However, it is not likely that all the air in the CNS can be evacuated during craniectomy. Over time, air that is reabsorbed is replaced with CSF.

The case described in the present report was unique in that the pneumocephalus was not associated with a surgical procedure and that the dog had a prolonged period without neurologic abnormalities following the initial trauma. In humans, clinical signs typically develop within 1 month after an injury, with drowsiness, headache, and vomiting being the earliest signs. In animals, identifying headaches or drowsiness is difficult, and thus, the diagnosis is often delayed until more severe neurologic abnormalities develop.

Findings for the dog described in the present report illustrated that emergency craniotomy may be appropriate in instances of severe, progressive neurologic abnormalities. Favorable outcomes may be achieved if the underlying disease is surgically amenable and extensive permanent damage to the CNS has not occurred.

References