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Case report

Esophagus impaction in a 4.5-yrs-old African elephant

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Place: Botswana
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"Choke" symptoms can be associated with:

- Congenital abnormalities of the upper digestive system; symptoms usually show up when the animal is weaned and starts eating solid food.
- Mega-esophagus, which can be congenital or acquired;
- Foreign bodies that get stuck in the upper digestive system;
- Impaction or blockage of the stomach/duodenum;
- Abnormal function of the cardiac sphincter which is the valve allowing food to flow into the stomach.

Blockage of the upper digestive system can cause damage to the esophagus resulting in strictures and narrowing, which makes the problem worse or even necrosis and rupture of the esophagus.

Regurgitation can result in food and liquid entering the trachea and the lungs. This causes a foreign body pneumonia, which in chronic cases can result in the eventual death of the animal.

History

A ±4.5-yrs-old male African elephant calf had been rescued from the wild after being injured in a bush fire. While in the rescue facility, it developed episodes of "choke", caused by impaction of the esophagus with ingested foodstuff. This resulted in regurgitation, and the inability to eat and drink without the food or liquid dribbling out of the animal's mouth.

Treatment

At first consultation, the elephant was not eating and was salivating.

Under standing sedation, a stomach tube was advanced into the esophagus which immediately created passage. Painkillers, antibiotics, and Buscopan were given. The cause of the blockage could not be determined.

Ten weeks later, the animal showed similar symptoms, however, they were more severe and they had possibly been going for longer than the previous episode. Awaiting the arrival of the vet, the animal was given

Buscopan, antibiotics and anti-inflammatories. As there was no improvement, the elephant was sedated again. This time, 4 sedations during 2 days were required before the blockage of the esophagus could be cleared. High volumes of fluids were given intravenously, tubes of different diameters were introduced (one for flushing with water and one for drainage), and foodstuff was removed mechanically. The impaction could be visualized with a three-meter endoscope as twigs and leaves jammed proximal to the cardiac sphincter. The esophagus appeared enlarged and flaccid. The wall of the stomach looked normal at endoscopic inspection.The animal was treated with a long course of antibiotics, liquid food slowly changing to solids over two months and he recovered well.

Four months later the animal once again showed signs of discomfort, inappetence, regurgitation and salivation. Finally he vomited chewed bark and milk. He did not respond to the treatment with antibiotics, NSAIDs and Buscopan. He was sedated again for three hours, initially standing and then recumbent. The esophagus was blocked and passage of a tube failed. However, large volumes of chewed bark could be removed manually from the oro- and nasopharynx. The pharynx and esophagus were completely blocked with chewed bark. A large-bore tube was introduced into the proximal esophagus and a small-bore tube within it attached to a hose pipe was used to flush the esophagus and pharynx. At the same time high volumes of intravenous fluids and rectal fluids were administered. After this procedure, antibiotics and NSAIDs, steroids were given for several days. Sildanefil rectally was used to try and relax the cardiac sphincter.

The following day the elephant was anesthetized and went into lateral recumbency. Medetomidine/butorphanol was used for induction of anesthesia, which was maintained with intravenous ketamine. A cuffed endotracheal tube was inserted into the trachea. The two tubes as described above were introduced into the esophagus. Large volumes of finely chewed bark were flushed from the esophagus, through the mouth and trunk. A handful of 12 mm gravel was also recovered. Eventually the esophagus and the stomach were cleared from these materials. No abnormalities gross lesions were seen with the endoscope. The esophagus was suspected to be flaccid and dilated, with possible damage at the level of the cardiac sphincter, possibly caused by impaction with gravel.

During anesthesia high volumes of intravenous and rectal fluids were given. Grey, hard stools were being passed irregularly. A secondary impaction as a result of pain and dehydration was suspected. No discomfort was observed. Fluid, intravenous and rectal continued during several days after anesthesia. Finadyne was given at lower dose to treat the impaction. Oral fluids were being swallowed and volumes were slowly increased. The administration of antibiotics was continued as complications in the form of a regurgitation pneumonia were anticipated.

The prognosis for full recovery was guarded as recurrence was anticipated. In the following 2 months, the elephant went through several similar episodes, which luckily resolved on their own. Repeated bouts of pneumonia were expected, which need to be treated aggressively each time they occur. This follows a classic congenital abnormality/mega-esophagus pattern but in this case the damage resulting in the impaction may be the result of injuries from the original fire, possibly by inhalation. However, it cannot be ruled out that this animal is just a naughty, hand-reared baby elephant eating strange objects.

Treatment results

Two months after the last treatment the elephant calf is recovering well, tired but improving. Showing some discomfort in one leg. Suspect an infection or damaged areas of skin with secondary infection. He showed ventral edema which improved with a course of antibiotics. It appears that areas of skin are sloughing off. Antibiotic treatment has been instituted. The skin problems are suspected to be part of a systemic infection originating from a regurgitation pneumonia. Another theory to explain the skin necrosis is the involvement of an autoimmune circulatory component as there had been peripheral lesions on the ears at the start of treatment, which later also showed edema.