

Investigation and treatment of anaemia and suspected gastric ulceration in a juvenile Eurasian otter (*Lutra lutra*)

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Introduction:

A juvenile female Eurasian otter (*Lutra lutra*), from Somerset, was found collapsed and was submitted to the RSPCA West Hatch Wildlife Hospital. Following a clinical examination she was found to be anaemic. This case report details the investigation and treatment of this anaemia and discusses a number of important rehabilitation issues that arose as a result of this treatment.

Case History:

Day 1:

A juvenile female otter was presented for treatment in a state of collapse. The otter weighed 1006g and had a full set of deciduous teeth. On initial examination her nose and eyes were found to be dull and her mucous membranes pale pink. Her body temperature was 36.5°C. There was no evidence of any bite wounds around her tail or anus and no other injuries were found.

Intravenous fluid therapy was administered into the cephalic vein at approximately 60ml/kg/day (500ml Hartmans (Aqupharm No.11; Animalcare) + 20ml of an electrolyte, vitamin and amino acid solution (Duphalyte: Fort Dodge)). Broad spectrum antibiotic cover was provided with clavulanate-amoxicillin (Synulox; Pfizer) at 12.5mg/kg subcutaneously.

Day 2:

A marked improvement was evidenced and the otter chose to discontinue fluid therapy herself! Blood was taken for a small mammal profile and for Aleutian disease serology. A low packed cell volume (PCV) and low total protein (TP) (albumin and globulin) levels were recorded (Figure 1). An anthelmintic was administered: Fenbendazole at 100mg/kg (Panacur 2.5% Liquid; Intervet). Fresh trout was offered, and taken, together with some artificial milk (Esbilac; Kruuse). The otter was housed in a large in-door pen, bedded on shavings and blankets, with access to a dog basket filled with water.

The otter's PCV and TP were monitored over the following two weeks. On day 9 the PCV had fallen to 26%; TP 72 g/l.

Day 15:

The otter had gained 400g and was eating well. The otter's temperature was 38.2°C. Her PCV, however, had still not improved. An in-house PCV measurement

revealed a PCV of 28% and a buffy coat of 2%. Blood was taken for a repeat small mammal profile and treatment was instituted with clavulanate-amoxicillin (Synulox; Pfizer) at 12.5mg/kg subcutaneously once daily for seven days. A single dose of carprofen (Rimadyl; Pfizer) was administered at 4mg/kg subcutaneously. Vitamin and iron supplementation (Equisup-i; Vétquinol) was provided: 0.6ml orally once daily for seven days. The haematology results confirmed a leucocytosis (WBC $19.3 \times 10^9/l$), consisting primarily of a lymphocytosis ($8.88 \times 10^9/l$) with a slight monocytosis ($1.16 \times 10^9/l$) and a very slight eosinophilia ($1.16 \times 10^9/l$).

Day 21:

A further 400g weight gain had been obtained and the otter now weighed 1808g. Another blood sample was taken following antibiotic and vitamin treatment. The results revealed a worsening anaemia with low haemoglobin, PCV and RBC readings (Figure 1). A reticulocyte count of 17% confirmed the presence of large numbers of young erythrocytes. This was consistent with the hypochromasia reported in all three samples to date. The anaemia was therefore regenerative. The leucocytosis was still in evidence, having dropped only slightly to $16.0 \times 10^9/l$. Vitamin and iron supplementation was continued.

Day 30:

The otter continued to gain weight (2586g) and now measured 69cm nose to tail (tip). Her permanent upper canines had broken the gum.

The deteriorating RBC picture report (PCV 22%; TP 66g/l) suggested a continuous source of bleeding. The possibility of gastric bleeding from a stomach ulcer was raised. This prompted a trial therapy of sucralfate (Antepsin; Chugai) (250mg/otter orally bid, equivalent to 100mg/kg) and Ranitidine (Zantac; Glaxo-Wellcome) (2mg/kg orally tid). Vitamin and iron supplementation was continued.

Day 37:

Following a seven-day course of anti-ulcer treatment, blood was again taken for a repeat small mammal profile. Some weight loss was recorded (2280g) but this was put down to the treatment and the necessary food restriction that was necessary to ensure that the treatment was taken. The blood results showed little improvement in the otter's PCV but some improvement in the white blood cell picture (Figure 1). Treatment was continued awaiting the blood results and prolonged for a further seven days once they had been obtained.

Day 42:

A routine faecal analysis was performed and revealed the presence of a large number of nematode eggs. Selamectin (Stronghold 15mg Spot on Solution; Pfizer) was administered topically at a minimum dose rate of 6mg/kg.

Day 44:

The otter was again re-blood-sampled. Blood clotting tests were requested to exclude rodenticide toxicity as a possible differential. This was very much precautionary, as no evidence of any haemorrhaging had ever been detected. The blood results showed little improvement in the red blood cell picture. Even more alarmingly the WBC count had jumped to $25.2 \times 10^9/l$, consisting primarily of a lymphocytosis ($15.12 \times 10^9/l$) (Figure 1). The blood clotting times were normal: Prothrombin time 8.5sec; Activated Prothrombin time 15.5sec.

A urine sample was collected and examined to rule out renal or bladder blood loss. The urine had a pH of 5 and a specific gravity of 1.039. No blood, protein or glucose was detected.

Another faeces sample was collected and revealed large numbers of hookworm-like eggs. A sample was sent to the Natural History Museum for identification. Once weekly treatment with praziquantel and pyrantel (Drontal Plus; Bayer) was prescribed: 1/3 tablet orally on day 55, then 1/2 tablet orally on day 62 and again on day 69.

Day 56:

The otter was pre-medicated with 0.25mg/kg midazolam (Hypnovel; Roche) and 5mg/kg ketamine (Vetalar; Pharmacia and Upjohn) *i/m*. Pre-operative analgesia was provided with an *i/m* injection of 0.02mg/kg Buprenorphine (Temgesic; Schering-Plough). Anaesthesia was induced seven minutes later with 4% halothane (Halothane Vet; Merial) and oxygen administered by facemask. The otter was then intubated with a size 4 ET tube and anaesthesia was maintained with 1-2% halothane. A dextrose saline infusion was administered into the cephalic vein, using a syringe driver, at 5ml/kg/hr.

A gastroscopy was performed per os. Large quantities of shavings were found in the otter's stomach. These had to be removed with basket forceps to allow detailed visualisation of the gastric mucosa and facilitate access to the pylorus. A lesion was found on the gastric mucosa that looked like an old ulcer; deposits of old blood were also observed on the gastric mucosa. The overall appearance of the gastric mucosa suggested an inflammatory process. A number of biopsies were taken. The duodenum was then examined and the mucosa found to have a rough appearance. Biopsies were taken from the proximal duodenum. The otter was then allowed to recover from anaesthesia.

Treatment with ranitidine and sucralfate, together with iron and vitamin supplementation, was continued. The otter was taken off a shavings based bedding and put onto sheets and blankets to eliminate shavings as a possible aggravating factor.

Day 63:

The otter's weight now approached 3kg (2994g) and she measured 79cm nose to tail (tip). Blood was again taken for a repeat profile. No improvement was seen in the blood picture (Figure 1). No nematode eggs were seen on faecal examination.

Day 71:

Blood was again taken for a small mammal profile. A faecal examination revealed no evidence of the eggs previously seen. The sample sent to the Natural History Museum had not conclusively identified any helminth eggs or adult worms. Treatment was discontinued.

Discussion:

Otter cubs may be born during any month of the year (Simpson and King, 2003) and the time of year therefore gives little indication of a cub's age. Cubs do not normally leave the holt until about eight weeks old, by which time they should weigh

around 1.5kg (Simpson and King, 2003). Permanent incisors erupt at approximately 13 weeks and the permanent canines at approximately 15 weeks (Heggberget, 1996).

This particular cub was found in the first week of February and weighed 1006g. She had a full set of deciduous teeth. The adult canines erupted somewhere around day30 after admission, at which point she weighed 2586g and measured 69cm nose to tail tip. Based on the little data available (Heggberget, 1996), she would probably have been 14-16 weeks old at this point, and 8-10 weeks old on admission.

Her low admission weight suggested that she may have been orphaned or abandoned (Simpson and King, 2003). She was found to be both hypothermic and dehydrated on admission, which is again consistent with her becoming separated from her mother (Simpson and King, 2003). No wounds or injuries were found that might have suggested a traumatic episode.

The otter's anaemia was evident on day 2. Dehydration may still have been present at this point and the initial PCV reading of 28% may actually have been artificially raised as a result. The red blood cell picture showed good signs of regeneration. The findings were generally consistent with a period of anorexia and malnutrition. In view of these findings and the otter's excellent appetite, it was felt that things should correct themselves naturally over the following week. A single high-dose of anthelmintic was administered but was not repeated due to staff objections that the otter should deal with its parasite burden itself.

The otter's anaemia did not improve. Over the following two weeks the PCV remained stable at 28%, before then dropping to 18% on day30. The anaemia showed every sign of being regenerative but has never climbed much above 20%. A leucocytosis, with a lymphocytosis predominating, became apparent and this became a consistent feature of the white blood cell picture. No evidence of reactivity, toxicity or vacuolation was seen in any of the leucocyte series. The otter's globulin levels were never raised.

Normal values for juvenile Eurasian otters have been established (Lewis and others, 1998). The mean PCV for 16 otters, of both sexes, aged less than 1 year was 46%. Nine of the sixteen juvenile otters were female; their mean PCV was 45%. A range was only provided for the forty-one otters, of both sexes and all ages, this ranged from 28% to 74%. The full set of results are presented in figure 2.

The persistent anaemia, together with chronic inflammatory picture, was of great concern. From a rehabilitation point of view, the otter urgently needed socialisation with other otters and there was considerable concern that she was becoming too tame. Concerns over her condition, however, prevented her being mixing with other otters; there were two considerations:

- (i) Her own health status and need for further treatment.
- (ii) The risk she might pose to the health of other otters.

Repeated efforts were made to identify the source of blood loss. The otter showed no signs of jaundice. The anaemia was therefore assumed to be extra-vascular rather than intra-vascular. Anti-coagulant rodenticide toxicity was not suspected because of the absence of any mucosal, scleral or retinal haemorrhaging. The normal clotting times obtained would tend to confirm this. Renal and bladder blood loss was

discounted as there was never any evidence of haematuria and urine analysis was unremarkable. Consideration was given to a parasitic aetiology: The otter did not have an obvious ecto-parasite burden, but was treated for fleas and ticks anyway on day 42. A high dose of praziquantel (100mg/kg) had been administered on day 2. The possibility of an endo-parasite burden was therefore not properly considered until it became clear that the anaemia was not responding to treatment. Faecal analysis on day 42 revealed large numbers of nematode eggs. Selamectin was therefore administered to deal with any ecto- and endo-parasites. Subsequently repeat doses of praziquantel and pyrantel were administered. No intra-cellular parasites were seen on any of the blood smears. Aleutian disease serology was negative. No serology was performed to test for feline infectious peritonitis or any other viruses that may possibly affect otters (Simpson and King 2003).

The possibility was raised of some gastric bleeding / ulceration as this has been recorded in otters (Vic Simpson personal communication). A trial treatment with anti-ulcer drugs was decided upon. An improvement in the otter's PCV was not recorded and it was therefore decided to perform an endoscopic examination of her stomach and duodenum. The gross findings of this examination were strongly suggestive of a gastritis. A healing gastric ulcer was identified but no significant histopathological results were reported following biopsy of the gastric and duodenal mucosa.

Gastric ulceration is often associated with stress and this is believed to be the most probable underlying aetiology for its presence in otters (Simpson and King, 2003). Gastric ulceration has been recorded in male otters with extensive bite wounds (Simpson, 1997). Similar mucosal ulceration has also been recorded in orphaned otter cubs (Simpson and King, 2003). The persistent anaemia and persistent lymphocytosis seen in this otter cub can only be explained by the continuous loss of blood from the gastro-intestinal tract and the resulting chronic inflammation. A very heavy endo-parasite burden could be responsible for this sort of blood loss but one would have expected to find obvious evidence of adult worms in the faeces. *Pseudoterranova decipiens*, a nematode known as the "cod worm" has been recorded in British otters (Jefferies and others, 1990). There are, however, no records in British otters of the hookworms (*Uncinaria* spp.) commonly recorded in British foxes (*Vulpes vulpes*). The precise identification of the eggs detected in the otter's faeces samples remains unclear, as does the significance of this finding. It is suggested that gastric ulceration and an endo-parasite burden are responsible for this otter's anaemia.

Worryingly, the otter has failed to respond to anti-ulcer treatment and anthelmintic treatment. The otter has also become increasingly tame and its viability as a wild otter is in doubt. On a more positive note the otter has more than trebled in weight since admission and retains a good appetite.

A number of important clinical and rehabilitation issues and questions have arisen as a result of this case:

- (1) An abandoned cub may well have been abandoned by its mother because it was sick and unlikely to survive. A full health check is therefore indicated to establish the otter's health status.

- (2) All wild animals, taken into care, should be wormed and / or should have a faecal sample examined for endoparasites. Repeat faecal examination is indicated to ensure response to treatment.
- (3) The need to treat a young wild animal in isolation needs to be carefully balanced against its developmental needs. A compromise may have to be negotiated but each case should be assessed on its merits.
- (4) A veterinary pre-release health check of all otters should be performed.
- (5) Decisions must be based on as much objective information as possible. The scarcity of any data on otters, and many other wild species, makes it all the more important that data be recorded accurately and systematically. Improved decision-making will hopefully result from this data and from reports such as this.

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