Clinical Cases in Avian and Exotic Animal Hematology and Cytology

TERRY W. CAMPBELL
and
KRYSTAN R. GRANT

WILEY-BLACKWELL
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Section 6: Avian Cytology Case Studies

Section 7: Herptile Cytology Case Studies

Section 8: Fish Cytology Case Studies

PREFACE

This book provides representative examples of hematology and cytology cases encountered in exotic animal practice. Cases in the book were selected based on the important role of cytodiagnosis or hematology in the medical management of the exotic animal patient. The cases in the book offer a variety of hematologic and cytodiagnostic interpretations.

Cases representing animals with anemia include blood loss, hemolytic, iron deficiency, Heinz body, and nonregenerative anemia. An example of polycythemia as well as the effects of lead toxicosis on the hemogram is provided. A variety of abnormal leukograms, such as leukocytosis, leukopenia, leukemia, and stress responses, are represented in the text. Representations of normal and abnormal hemic cytologies are provided. These include normal hemic cells, toxic neutrophils and heterophils, left shifts, and leukemia. Blood parasites, such as *Leukocytozoon*, *Hemoproteus*, *Plasmodium*, and *Hemogregarine*, and bacteremia are also represented.

Example cases of the basic cytodiagnosis interpretations are also represented. These include normal cytology, inflammation, hyperplasia or benign neoplasia, and malignant neoplasia. Inflammatory lesions are represented by neutrophilic or heterophilic, mixed cell, macrophagic, and eosinophilic inflammation. Along with these inflammatory lesions, a specific etiologic agent, such as bacterial, mycobacterial, fungal, viral, parasitic, or foreign body, is represented. Tissue hyperplasia or benign neoplasia is represented by epithelial hyperplasia, papilloma, adenoma, lipoma, mast cell tumor, and chondroma. Representations of malignant neoplasia include carcinomas, such as undifferentiated carcinoma, adenocarcinoma, and squamous cell carcinoma; sarcomas, such as undifferentiated soft tissue sarcoma, liposarcoma, hemangiosarcoma, and malignant melanoma; and discrete cell neoplasms, such lymphoma, histiocytoma, and mast cell tumor.

Effusions are also represented. These include transudate, modified transudate, exudate, and hemorrhagic effusion. Examples of specific fluid analysis include synovial fluid, such as articular gout and synovial cysts, and a salivary mucocele.

Guideline for Using the Clinical Cases Presented in this Book

This book is offered as a companion to TW Campbell and CK Ellis, *Avian and Exotic Animal Hematology and Cytology*, Ames, Iowa, Blackwell Publishing, 2007, and is designed to assess one's level of knowledge in the use of hematology and cytology in the diagnosis of health disorders involving exotic animal patients. The clinical cases presented were obtained from animal medical records, and each was chosen for its relevant hematology or cytology data. Although not a focus of the book, other clinical data, such as serum or plasma biochemistry profiles (presented in conventional units), imaging, and histology, are also presented with some case studies. Veterinarians, veterinary students, and veterinary technicians in clinical practice will find this additional information useful as an example of how each case was managed medically or surgically. Veterinary clinical pathologists and laboratory technicians will also find this added information beneficial in providing a complete overview of each case. Often the pathologist and laboratory technicians are exposed to only a small part of the clinical cases that they help to manage. Overall, this book is designed to test one's skills in the interpretation of laboratory data and cytology with the added benefit of providing self-assessment material for all aspects in the management of the exotic animal patient.

Results of the serum or plasma biochemistry profiles presented in these case studies were obtained using the Roche Hitachi 911 chemistry analyzer (Roche Diagnostics Corporation, Indianapolis, IN). Study cases that include mammalian blood cell counts were obtained using the Advia® 120 Hematology System (Siemens Medical

Solutions Diagnostics, Tarrytown, NY). Total leukocyte counts from lower vertebrates (birds, reptiles, and fish) in the case studies were obtained by manual methods using either the direct (Natt and Herrick's method) or semidirect (phloxine B method) manual method (Campbell and Ellis, 2007).

The cases are presented in a manner that allows the reader to learn by making his or her own description of microscopic images and interpretation of the data. Following each data set, an interpretive discussion and case summaries are provided to be used by the reader for self-assessment of proficiency in interpretation of the data.

It is possible that one may have managed a case differently from what was described in the text. Each case presented in this book follows the case management as it was described in the medical records including the outcome, when known. Any differences of opinion can be used as a comparison of clinical management styles.

The clinical case studies are organized according to the animal type and diagnostic focus (either hematology or cytology):

Section 1: Mammalian Hematology Case Studies
Section 2: Avian Hematology Case Studies
Section 3: Herptile Hematology Case Studies
Section 4: Fish Hematology Case Studies
Section 5: Mammalian Cytology Case Studies
Section 6: Avian Cytology Case Studies
Section 7: Herptile Cytology Case Studies
Section 8: Fish Cytology Case Studies

The reader should note that the term "herptile" used in the title for Sections 3 and 7 is an arcane lexicon, in this case, a word used only by those who deal with reptiles and amphibians. Thus, reptiles and amphibians are collectively known as herptiles. The term likely comes from the word herpetology, the study of reptiles and amphibians. The term "herp," another arcane lexicon used by this group, refers to an animal that is either a reptile or an amphibian.

The following questions are to be answered by the reader while navigating through the clinical cases and are designed to guide the reader in the management of real-life cases. Many quality reference texts on avian and exotic animal medicine are available to provide the reader with in-depth information on specific aspects in the management of real-life clinical cases and aid the reader in answering these questions:

1. What is the significant historical information needed in order to assess the husbandry provided to the patient? What husbandry advice would you give to the owner of this patient?
2. What historical information is needed in order to assess the cause of the primary complaint?
3. How would one perform a physical examination on this patient?
4. How does one determine the gender in this species?
5. On the basis of the historical information and physical examination findings, what are the likely rule-outs concerning this case?
6. What, if any, diagnostic tests are needed in order to evaluate the patient and arrive at a more definitive rule-out?
7. How would one obtain a blood sample from this patient and how much blood could one safely take? What is the best restraint method in order to do this?
8. What is the best way to handle the blood once it was obtained in order to perform a complete blood cell count and serum/plasma chemistry profile?
9. How would you interpret the complete blood count?
10. How would you interpret the plasma chemistry panel?
11. If needed, how would one obtain the cytologic sample for the assessment of this patient? How would you prepare this sample for cytological evaluation? What stain would you use?
12. How would you interpret the cytologic specimen?
13. How would you restrain and position the patient for a radiographic evaluation?
14. How would you interpret the radiographs, if available?
15. On the basis of the history, physical examination, blood profile, and radiographic evaluation, what is the most likely diagnosis?
16. What would you do next in the management of this case?
17. If needed, how would you anesthetize this patient?
18. If needed, how would you surgically manage this patient? How would you perform a surgical closure in this patient? When should one remove the skin sutures?
19. What instructions would you provide to the client?
20. What is the prognosis for this patient?

ACRONYMS AND ABBREVIATIONS

A/G	Albumin/globulin
ALT	Alanine aminotransferase
AP or ALP	Alkaline phosphatase
AST	Aspartate aminotransferase
BCS	Body condition score
BID	Twice daily
BUN	Blood urea nitrogen
CBC	Complete blood count
cc	Cubic centimeter
CK	Creatine kinase
cm	Centimeters
CNS	Central nervous system
CR	Computed radiography
CT	Computed tomography
dL	Deciliter
DV	Dorsoventral
E-collar	Elizabethan collar
EDTA	Ethylenediaminetetraacetic acid
F	Fahrenheit
fL	Femtoliters
FNA	Fine-needle aspirate
Ft	Feet
g	Gram
GFR	Glomerular filtration rate
GGT	γ-Glutamyltranserase
GI	Gastrointestinal
GMS	Gomori methenanime silver
Gy	Gray
Hb	Hemoglobin
HCO_3	Bicarbonate
IM	Intramuscularly
IO	Intraosseous
ISIS	International Species Information System
IU	International units
IV	Intravenously
kg	kilogram
LRS	Lactated Ringer's solution
Max	Maximum
MCHC	Mean cell hemoglobin concentration
mCi	Millicuries
MCV	Mean cell volume
M:E	Myeloid–erythroid
mEq	Milliequivalents
mg	Milligram
Min	Minimum
mL	Milliliters
mm	Millimeters
MPV	Mean platelet volume
MRI	Magnetic resonance imaging
N:C	Nucleus–cytoplasm
N/L	Neutrophils/lymphocytes
nmol	Nanomole
Oz	Ounce
PCV	Packed cell volume
PE	Physical examination
pmol	Picomole
PO	Per os
ppm	Parts per million
ppt	Parts per thousand
QID	Four times daily
q 24 hours	Every 24 hours
q 72 hours	Every 72 hours
RBC	Red blood cell
RDW	Red cell distribution width
SC	Subcutaneously
sp. or spp.	Species
Tc	Technetium
Tc-99m HDP	Technetium 99 high-density plasma
TIBC	Total iron-binding capacity
TID	Three times daily
μm	Micron
UIBC	Unsaturated iron-binding capacity
μL	Microliter
UV	Ultraviolet
VD	Ventral–dorsal
WBC	White blood cell

Clinical Cases in Avian and Exotic Animal Hematology and Cytology

Section 1
Mammalian Hematology Case Studies

1

A 6-Year-Old Otter Undergoing a Routine Physical Examination

Signalment

A 6-year-old intact North American male river otter (*Lontra canadensis*) was examined as part of a routine physical examination.

History

The patient was housed with two other male otters of the same age. No significant health problems had been observed in any of the otters. The otters were weighed weekly, and there had been no change in the appetite, behavior, or weight.

Physical Examination Findings

The 10 kg otter appeared healthy on physical examination (Figs. 1.1–1.4 and Tables 1.1 and 1.2).

Other Diagnostic Information

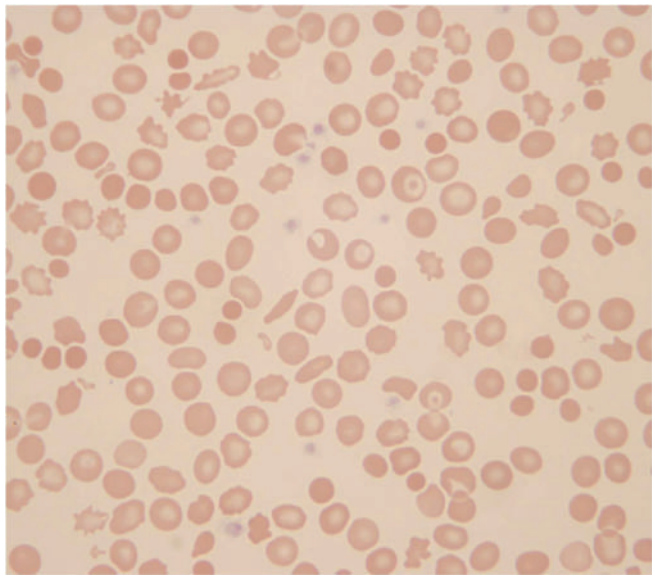
A fecal occult blood was positive; however, no red blood cells or other abnormalities were seen on a fecal cytology.

Whole body ventral–dorsal and lateral radiographs revealed no abnormalities in the abdominal organs. The T14-L1 intervertebral disk space was narrowed with sclerotic end plates and was indicative of spondylosis deformans.

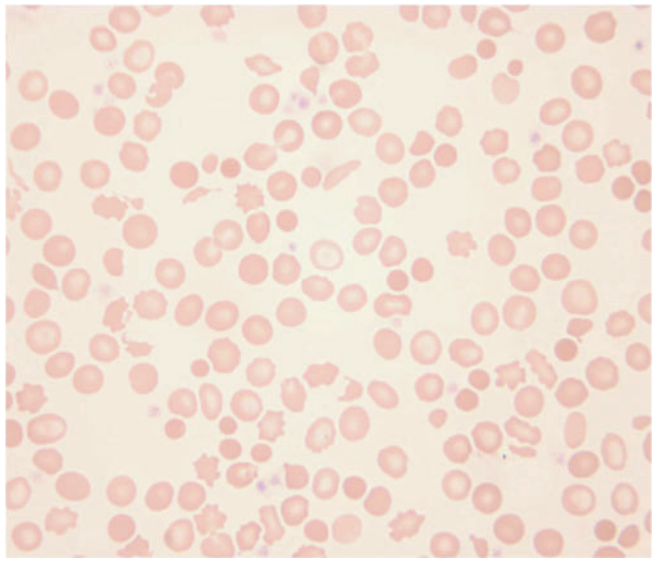
Endoscopic examination revealed evidence of fresh blood in the stomach and small punctate ulcers. Some shrimp tails remained in the stomach several hours after the last meal. The gastric mucosa was irregular, suggesting a possible infection associated with *Helicobacter* sp. The duodenum appeared normal. The esophagus was very long and the pylorus was open and easy to enter. Histopathologic examination of biopsies

Fig. 1.1. The North American river otter in an exhibit with his cage mate.

Fig. 1.2. The otter under anesthesia for physical examination and blood collection.



(a)



(b)

Fig. 1.3. (a and b) Blood films from an otter (Wright–Giemsa stain, 50×).

taken during the endoscopic examination revealed no abnormalities.

Interpretive Discussion

Figures 1.3 and 1.4 reveal erythrocyte abnormalities. Many of the erythrocytes are hypochromatic as indicated by extended central pallor and thin rim of hemoglobin. There are many keratocytes and schistocytes present. The erythrocytes (blister cells) appear to be developing vacuoles or blisters that enlarge. These blisters eventually break open to form "apple stem cells" and keratocytes. Spiculated erythrocytes (those

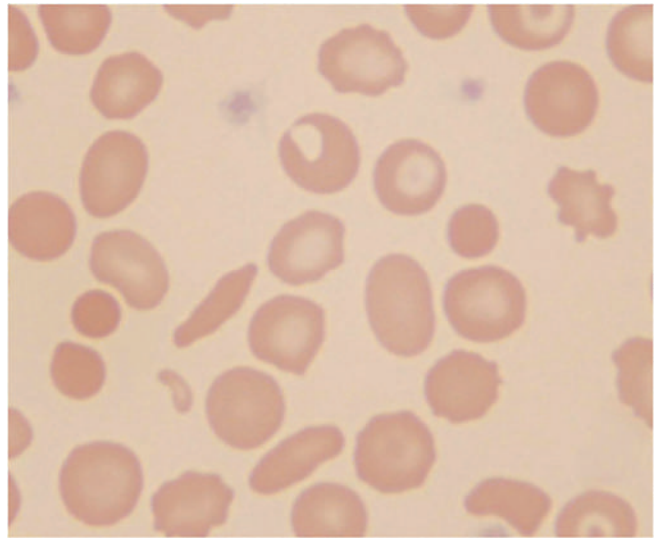


Fig. 1.4. A blood film from an otter (Wright–Giemsa stain, 100×).

with more than two pointed projections) are also seen. The projections fragment from the cells to form the schistocytes.

The packed cell volume (PCV), hemoglobin concentration (Hb), mean cell volume (MCV), and

Table 1.1. Hematology results.

	Day 1	Ranges for otters at aquarium
WBC (10^3/μL)	7.1	2.7–5.3 (3.8)
Neutrophils (10^3/μL)	4.4	1.6–3.9 (2.4)
Neutrophils (%)	62	38–73 (61)
Lymphocytes (10^3/μL)	2.4	0.7–1.6 (1.1)
Lymphocytes (%)	34	16–48 (31)
Monocytes (10^3/μL)	0.2	0–0.2 (0.1)
Monocytes (%)	3	1–5 (2)
Eosinophils (10^3/μL)	0.1	0–0.4 (0.2)
Eosinophils (%)	1	1–8 (4)
Basophils (10^3/μL)	0	0
Basophils (%)	0	0
Plasma protein (g/dL)	7.4	7.4–8.1 (7.7)
RBC (10^6/μL)	11.5	10.9–14.6 (12.3)
Hb (g/dL)	9.9	16.0–19.6 (17.0)
PCV (%)	38	48–60 (52)
MCV (fL)	33.0	39–45 (42)
MCHC (g/dL)	26.0	32–34 (33)
Reticulocytes per microliter		10,910–14,620 (12,673)
Reticulocytes (%)		0.1
RDW	8.8	13.6–18.0 (15.1)
Platelets (10^3/μL)	762	311–474 (371)
MPV (fL)	5.9	6.0–6.9 (6.6)
Clumped platelets	0	0
Keratocytes	Moderate	0
Echinocytes	Few	0 to few
Hypochromasia	Slight	0
Reactive lymphs	Few	0

Table 1.2. Plasma biochemical results.

	Day 1	Ranges for otters at aquarium
Glucose (g/dL)	109	91–136 (114)
BUN (mg/dL)	38	27–43 (36)
Creatinine (mg/dL)	0.4	0.4–0.7 (0.5)
Phosphorus (mg/dL)	4.6	2.2–4.8 (3.6)
Calcium (mg/dL)	8.9	8.5–9.4 (9.0)
Total protein (g/dL)	7.0	6.6–7.4 (7.0)
Albumin (g/dL)	2.9	2.6–3.2 (3.0)
Globulin (g/dL)	4.1	3.6–4.2 (4.0)
A/G ratio	0.7	0.7–0.9 (0.8)
Cholesterol (mg/dL)	177	88–235 (175)
Total bilirubin (mg/dL)	0.1	0.1–0.2 (0.2)
CK (IU/L)	149	148–588 (375)
ALP (IU/L)	61	60–118 (82)
ALT (IU/L)	104	91–127 (112)
AST (IU/L)	122	88–174 (125)
GGT (IU/L)	9	7–14 (10)
Sodium (mg/dL)	147	143–149 (146)
Potassium (mg/dL)	3.7	3.7–4.0 (3.9)
Chloride (mg/dL)	114	107–115 (112)
Bicarbonate (mg/dL)	20.8	17–25 (21)
Anion gap	15	10–20 (16)
Calculated osmolality	300	291–303 (297)
Lipemia (mg/dL)	9	—
Hemolysis (mg/dL)	9	—
Icterus (mg/dL)	0	—

mean cell hemoglobin concentration (MCHC) on the hemogram are decreased, which is indicative of an iron-deficiency anemia. The appearance of microcytic, hypochromic erythrocytes on the blood film is also indicative of an iron-deficiency anemia, a condition that is nearly always caused by chronic blood loss in an adult animal. The positive fecal occult blood is suggestive of gastrointestinal blood loss in this patient; however, the two healthy otters that share his habitat also exhibited positive fecal occult blood tests. Thus, it is likely that the results of the fecal occult blood testing are false-positive owing to the meat diet of the otters. The endoscopic examination suggested the possibility of blood being lost from the upper gastrointestinal tract as would be seen with *Helicobacter* involvement; however, histologic examination of biopsy samples failed to confirm pathology associated with that area (Table 1.3).

Table 1.3. Plasma iron profile results.

	Day 1	Ranges for otters at aquarium
Iron (μg/dL)	35	112–160 (135)
TIBC (μg/dL)	434	286–409 (320)
Saturation (%)	8	27–58 (44)
UIBC (μg/dL)	399	116–297 (186)

Variability in the normal serum iron, total iron-binding capacity (TIBC), and percent saturation of transferrin occurs among mammalian species; however, in general, healthy animals have an average serum iron concentration of 100 μg/dL, a TIBC of 300 μg/dL, and transferrin saturation of 33%. Using these values, this otter patient has a confirmed iron deficiency based on reduced serum iron concentration and transferrin saturation with an increased TIBC.

The platelet count is greater than expected. This is a common finding associated with iron-deficiency anemia in other mammalian species. The exact cause of this is unknown.

The otter had a mild leukocytosis, mature neutrophilia, and lymphocytosis, which are suggestive of a physiological leukocytosis. This is not surprising owing to the nature of capture and delivery of a chemical restraint needed in order to obtain the blood sample.

Summary

The otter underwent a 4-month treatment for a presumed chronic blood loss anemia resulting in the loss of iron from the gastrointestinal tract in association with a *Helicobacter* sp. infection. He was also treated with injectable supplemental iron. Because the otter never appeared weak or ill from his anemia, a reevaluation examination was performed 4 months following the initial examination. The erythrocyte parameters had returned to normal by that time.

2 A 10-Year-Old Ferret with Lethargy and Anorexia

Signalment

A 10-year-old castrated male Fitch ferret (*Mustela putorius furo*) was presented for anorexia and lethargy (Fig. 2.1).

History

The ferret recently exhibited bouts of intermittent melena. The ferret is housed with two other ferrets that appear healthy. Other pets in the household include two dogs and two cats. The client lives on a small farm where a small number of livestock (cattle and chickens) are kept. The ferrets are fed a commercial kibbled diet.

Physical Examination Findings

A geriatric ferret was 10% dehydrated and was moderately lethargic. A small amount of watery discharge was noted from his left eye. There was also a significant amount of debris in both ears that contained a mixed yeast and bacterial infection based on cytological examination. See Figures 2.2–2.5 and Tables 2.1 and 2.2.

Interpretive Discussion

In Fig. 2.2, the dark tarry stool is representative of melena. Figure 2.3 represents ear mites (*Otodectes*). Figure 2.4 shows the Wright–Giemsa stained blood film, which reveals numerous echinocytes and a schistocyte. An erythrocyte in the center as well as a few others appears to contain a pale structure, suggestive of Heinz bodies. Figure 2.5 shows staining of the blood with a stain used to detect reticulocytes and reveals blue structures within the erythrocytes, indicative of Heinz bodies.

On day 1, the ferret appears to be exhibiting a stress leukogram; however, considering the geriatric status of the ferret, it is also likely that the neutrophils/lymphocytes ratio has changed with age, resulting in decreased lymphocytes and increased neutrophils compared to younger ferrets. The ferret has a significant anemia based on the low PCV, RBC, and hemoglobin concentration. The cause of the anemia is likely in part related to blood loss in the gastrointestinal tract as indicated by melena. The refractometric plasma protein is low, which supports blood loss; however, this finding is not supported by the normal protein or perhaps elevated value found in the biochemical profile. Because the ferret appears clinically dehydrated, the anemia may actually be worse than it appears and the red cell indices and total protein values would expect to decrease with fluid replacement therapy. The anemia may also be related in part to a hemolytic anemia associated with Heinz body formation in the erythrocytes in which a moderate number of large Heinz bodies and a few small Heinz bodies were reported on the hemogram. At this time, the anemia appears to be poorly regenerative as indicated by the lack of a significant polychromasia and reticulocyte count. The platelet count is low owing to either excessive peripheral utilization of platelets associated with gastrointestinal hemorrhage or perhaps as an analytic artifact associated with clumping of platelets as indicated by the interpretation of the blood film. The presence of echinocytes is typically an artifactual finding.

The plasma biochemical profile on day 1 indicates a possible hyperproteinemia with a hyperglobulinemia suggestive of an immune response based on the first set of reference values but not supported by the second set of reference values.

The hemogram on day 6 indicates no significant change in the leukogram; however, there is a marked improvement to the erythrocyte parameters. The ferret is exhibiting a significant regenerative response to his erythrocytes, platelets, and refractometric total protein. He is no longer anemic and appears to be recovering from a blood loss anemia. The unexplained presence of

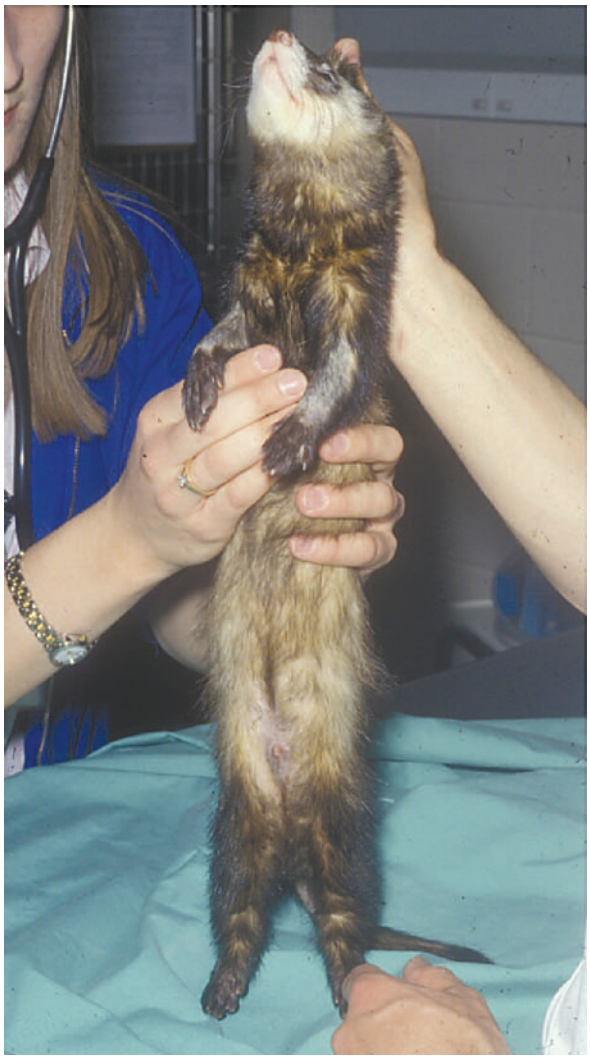


Fig. 2.1. The 10-year-old ferret during physical examination that presented with anorexia and lethargy.

Fig. 2.2. The gross appearance of the ferret's feces.

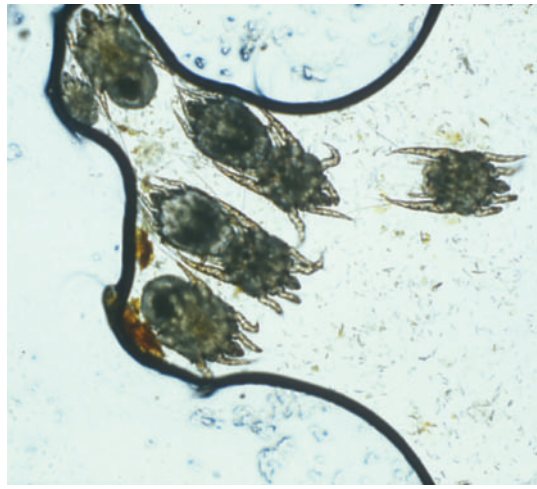


Fig. 2.3. A microscopic image of the material collected from the ferret's ear.

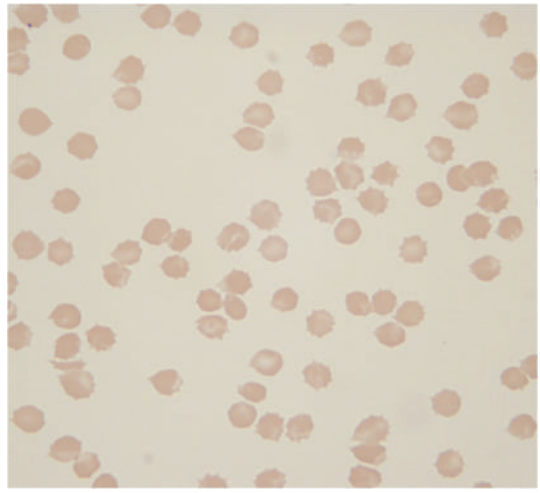


Fig. 2.4. A microscopic image of the erythrocytes on the blood film from the ferret (Wright–Giemsa stain, 100×).

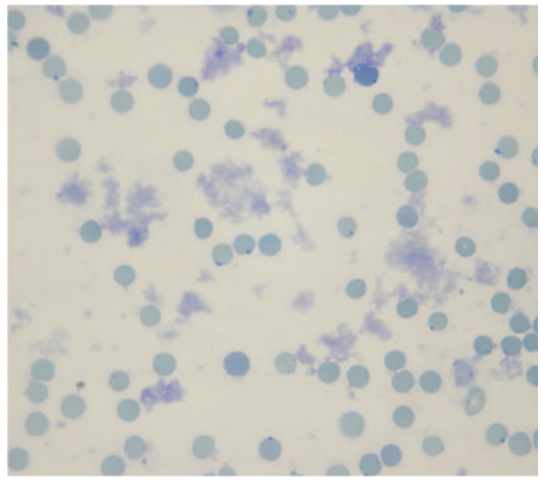


Fig. 2.5. A microscopic image of the erythrocytes on the blood film from the ferret (methylene blue stain, 100×).

Table 2.1. Hematology results.

	Day 1	Day 6	Reference[a]	Reference[b]	Reference[c]
WBC ($10^3/\mu L$)	5.2	5.2	4.0–9.0	4.4–19.1	7.7–15.4 (11.3)
Neutrophils ($10^3/\mu L$)	4.5	4.6	1.5–3.5	—	—
Neutrophils (%)	86	88	—	11–82	24–78 (40)
Lymphocytes ($10^3/\mu L$)	0.6	0.5	0.5–5.0	—	—
Lymphocytes (%)	11	9	—	12–54	28–69 (50)
Monocytes ($10^3/\mu L$)	0.2	0.3	0–0.5	—	—
Monocytes (%)	3	3	—	0–9	3.4–8.2 (6.6)
Eosinophils ($10^3/\mu L$)	0	0	0–0.5	—	—
Eosinophils (%)	0	0	—	0–7	0–7 (2)
Basophils ($10^3/\mu L$)	0	0	0	—	—
Basophils (%)	0	0	—	0–2	0–2.7 (0.7)
Plasma protein (g/dL)	3.5	7.7	5.0–6.5	—	—
RBC ($10^6/\mu L$)	3.7	9.4	7.0–11.0	7.3–12.2	—
Hb (g/dL)	7.1	18.1	12–18	16.3–18.2	12.0–16.3 (14.3)
PCV (%)	20	54	35–53	44–61	36–50 (43)
MCV (fL)	54.0	58.0	47–52	—	—
MCHC (g/dL)	36	33.0	33–55	—	—
Reticulocytes per microliter	7,360	46,110	—	—	—
Reticulocytes (%)	0.2	4.9	—	1–12	—
RDW	12.5	12.3	—	—	—
Platelets ($10^3/\mu L$)	73.0	475	—	297–730	—
MPV (fL)	9.3	8.5	—	—	—
Clumped platelets	Yes	No	—	—	—
Howell–Jolly bodies	Few	Few	—	—	—
Echinocytes	Moderate	Few	—	—	—

[a]Colorado State University reference ranges.
[b]Fox (1988).
[c]Carpenter (2005).

Table 2.2. Plasma biochemical results.

		Reference[a]	Reference[b]	Reference[c]
Glucose (mg/dL)	132	95–140	94–207	63–134 (101)
BUN (mg/dL)	23	10–26	10–45	12–43 (28)
Creatinine (mg/dL)	0.3	0–0.5	0.4–0.9	0.2–0.6 (0.4)
Phosphorus (mg/dL)	3.8	3.0–5.5	4.0–9.1	5.6–8.7 (6.5)
Calcium (mg/dL)	8.8	8.0–9.7	8.0–11.8	8.6–10.5 (9.3)
Total protein (g/dL)	7.4	5.0–6.4	5.1–7.4	5.3–7.2 (5.9)
Albumin (g/dL)	3.0	2.9–4.1	2.6–3.8	3.3–4.1 (3.7)
Globulin (g/dL)	4.4	1.8–3.0	—	2.0–2.9 (2.2)
A/G ratio	0.7	1.0–2.2	—	1.3–2.1 (1.8)
Cholesterol (mg/dL)	259	70–200	64–296	—
Total bilirubin (mg/dL)	0.3	0–0.3	<1	—
CK (IU/L)	136	80–400	—	—
ALP (IU/L)	25	10–60	9–84	30–120 (53)
ALT (IU/L)	207	80–270	—	82–289 (170)
AST (IU/L)	68	30–75	28–120	—
GGT (IU/L)	5	1–15	—	5
Sodium (mg/dL)	142	147–153	137–162	146–160 (152)
Potassium (mg/dL)	3.5	3.3–4.5	4.5–7.7	4.3–5.3 (4.9)
Chloride (mg/dL)	109	114–120	106–125	102–121 (115)
Bicarbonate (mg/dL)	16.3	15–23	—	—
Anion gap	20	14–21	—	—
Calculated osmolality	28.5	—	—	—
Lipemia (mg/dL)	0	—	—	—
Hemolysis (mg/dL)	13	—	—	—
Icterus (mg/dL)	0	—	—	—

[a]Colorado State University reference ranges.
[b]Fox (1988).
[c]Carpenter (2005).

Heinz bodies in this case has disappeared. Heinz bodies are caused by oxidative damage to hemoglobin. A common cause for this condition in domestic cats is ingestion of onions or onion products. Other plants, such as garlic, *Brassica*, and red maple (*Acer rubrum*) leaves, may also cause Heinz body formation. Drugs such as acetaminophen, phenazopyridine, phenothiazine, and propylene glycol, to name a few, will also cause Heinz body formation. No history of exposure to any of these materials was revealed in this case. Heinz body formation can occur without exposure to oxidant chemicals or drugs with medical conditions, such as lymphoma, diabetes mellitus, and hyperthyroidism.

Summary

The ferret's overall condition improved after 5 days of treatment for *Helicobacter*-induced gastrointestinal ulcers and hemorrhage, which continued for a total of 21 days. This treatment consisted of amoxicillin (20 mg/kg PO BID), doxycycline (5 mg/kg PO BID), omeprazole (0.7 mg/kg PO daily), and sucralfate (25 mg/kg PO every 8 hours). He was also successfully treated for an ear mite infestation using ivermectin (0.3 mg/kg) subcutaneously once every 10 days for three treatments.

3 A 6-Year-Old Ferret with Anorexia and Lethargy

Signalment

A 6-year-old female ferret (*Mustela putorius furo*) was presented for anorexia and lethargy.

History

This adult female ferret had a 2-day history of anorexia. The client had not observed her eating or drinking during this period and reported that the ferret has been sleeping more than usual. The owner did observe that although her stool production was less than normal, it appeared dark. During the past year, the ferret has been given oral melatonin for suspected adrenal disease. The ferret was normally fed a diet of kibbled ferret food.

Physical Examination Findings

The ferret was thin (body score of 3/9) and weighed 500 g. She was lethargic and at least 10% dehydrated. She was weak and reluctant to move. Her body temperature was 98°F and she had tachypnea. Black tarry stools were found adherent to the hair around the anus. A large ulcer was found during examination of the oral cavity. See Figures 3.1 and 3.2 and Tables 3.1 and 3.2 .

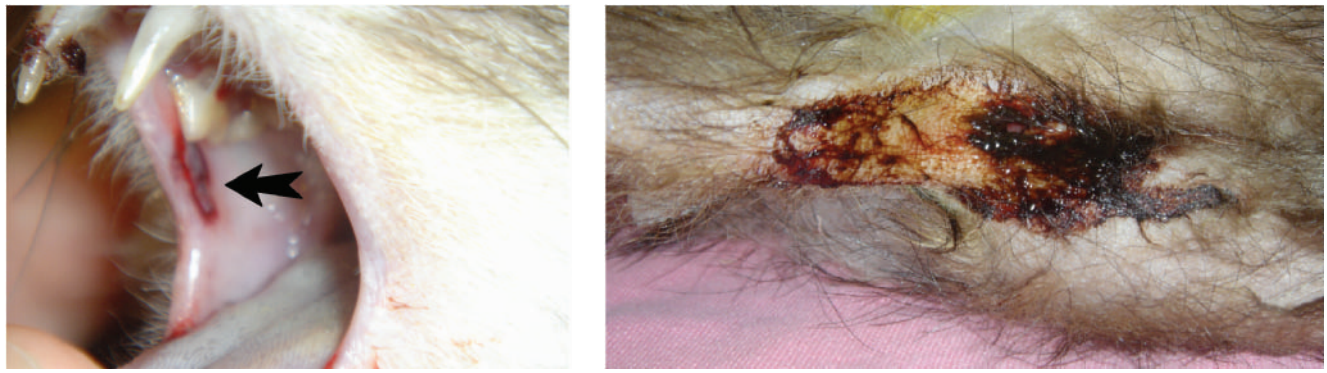
Other Diagnostic Findings

A radiographic evaluation of the ferret (Fig. 3.3) indicated a diffuse, unstructured interstitial to alveolar pattern in the caudal dorsal lungs. Because the radiographs were not centered over the thorax, specific thoracic radiographs were recommended for further evaluation. The cardiac silhouette appears to be within normal limits and the pulmonary vasculature appears to be within normal limits. The serosal detail of the abdomen is poor. There is very little falciform fat (back fat), suggesting an overly thin animal. What can be visualized in the abdomen appears to be otherwise normal. On the lateral image, the abdomen appears mildly pendulous. There is a large spleen, although this is typical for a ferret; however, splenomegaly cannot be entirely ruled out. The findings of the thorax and lungs could be indicative of hematogenous pneumonia in the caudal dorsal lungs. Alternatively, diffuse neoplasia cannot be entirely ruled out. Repeat imaging with computed radiography would be recommended to try to further evaluate these caudal dorsal lung lobes. The appearance of the loss of serosal detail in the abdomen could be the result of poor body condition score, although peritoneal effusion or carcinomatosis cannot be entirely ruled out. The remainder of the abdomen is unremarkable. An ultrasound examination was recommended, but declined by the client owing to the cost of the procedure.

Interpretive Discussion

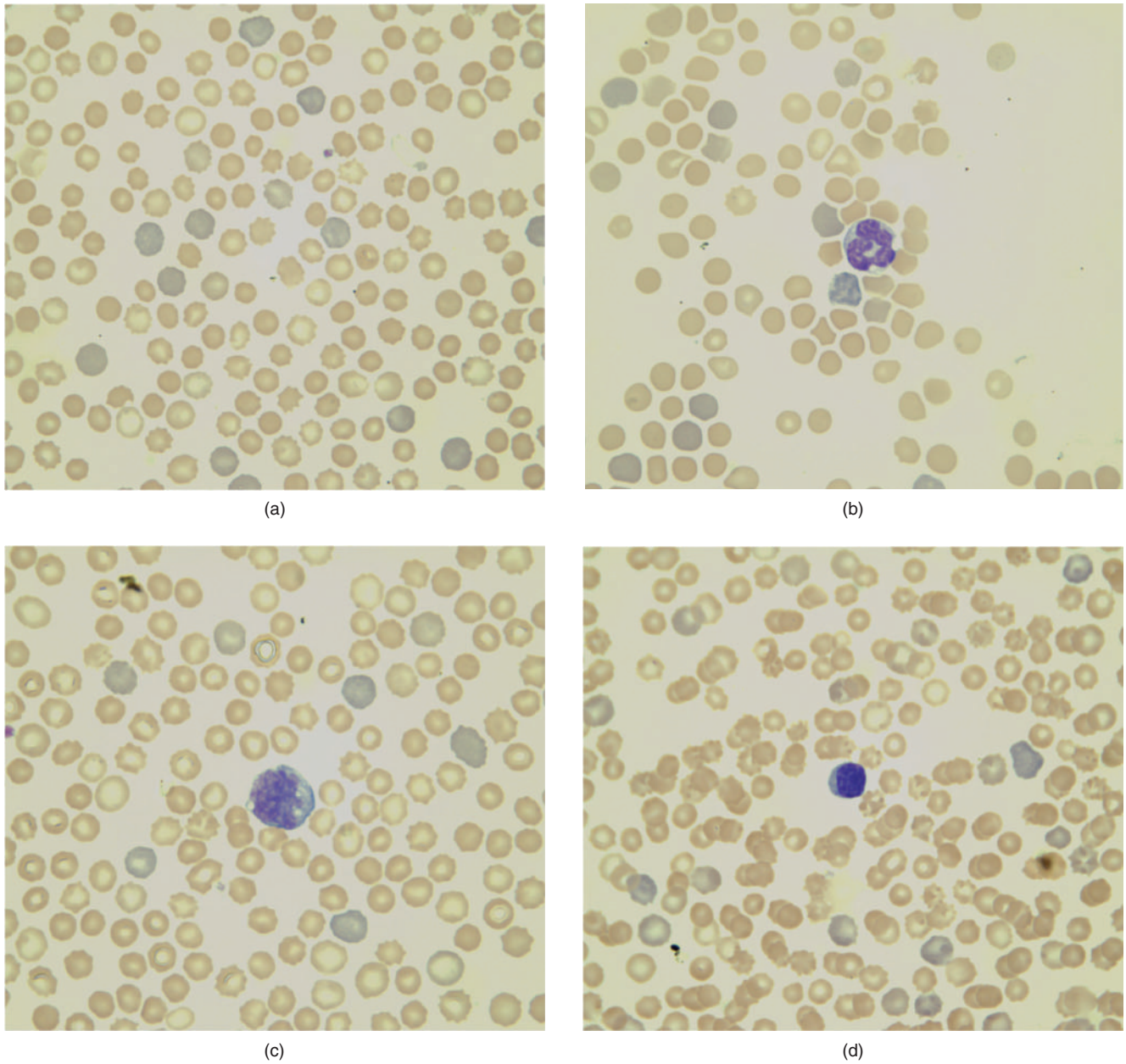
Figure 3.2a shows a marked number of polychromatophilic erythrocytes and echinocytes. Figure 3.2b shows a toxic neutrophil among erythrocytes, exhibiting significant polychromasia. Figure 3.2c shows a monocyte among erythrocytes, exhibiting significant polychromasia and many echinocytes. Figure 3.2d shows a lymphocyte with a moderate amount of dark blue cytoplasm, indicating a reactive lymphocyte as well as a significant polychromasia and many echinocytes.

In general, the hematology of ferrets resembles that of domestic carnivores. In this case, the ferret has a marked regenerative anemia based on the marked polychromasia on the blood film, presence of nucleated erythrocytes, and marked number of reticulocytes. The cause of the anemia is likely to be associated with blood loss as indicated by the low total protein and a low platelet count that indicates excessive consumption of platelets. The blood loss is likely from gastrointestinal



(a) (b)

Fig. 3.1. The 6-year-old ferret during physical examination that presented with anorexia and lethargy. (a) Oral cavity examination reveals an ulcer (arrow). (b) The Ferret in left lateral recumbency (the tail is on the left side of image and legs are on the right side). Melena seen on perianal region.



(a) (b)

(c) (d)

Fig. 3.2. (a–d) Microscopic images from the blood film (Wright–Giemsa stain, 100×).

Table 3.1. Hematology results.

		Reference[a]	Reference[b]	Reference[c]
WBC ($10^3/\mu L$)	2.4	4.0–9.0	4.4–19.1	2.5–8.6 (5.9)
Neutrophils ($10^3/\mu L$)	1.6	1.5–3.5	—	—
Neutrophils (%)	67	—	11–82	12–41 (31)
Band cells ($10^3/\mu L$)	0	0.5–5.0	0	—
Band cells (%)	1	—	0	0–4.2 (1.7)
Lymphocytes ($10^3/\mu L$)	0.3	0–0.5	—	—
Lymphocytes (%)	14	—	12–54	25–95 (58)
Monocytes ($10^3/\mu L$)	0.1	0–0.5	—	—
Monocytes (%)	4	—	0–9	1.7–6.3 (4.5)
Eosinophils ($10^3/\mu L$)	0	0	—	—
Eosinophils (%)	0	—	0–7	1–9 (4)
Basophils ($10^3/\mu L$)	0	5.0–6.5	—	—
Basophils (%)	0	7.0–11.0	0–2	0–2.9 (0.8)
nRBC ($10^3/\mu L$)	0.3	12–18	0	—
nRBC (%)	14	35–53	0	—
Plasma protein (g/dL)	4.4	47–52	—	—
RBC ($10^6/\mu L$)	6.5	33–55	7.3–12.2	—
Hb (g/dL)	11.1	—	16.3–18.2	15.2–17.4 (15.9)
PCV (%)	34	—	44–61	47–51 (48)
MCV (fL)	53	—	—	—
MCHC (g/dL)	33	—	—	—
Reticulocytes ($10^3/\mu L$)	712	—	—	—
Reticulocytes (%)	11	—	1–12	—
RDW	17.2	—	—	—
Platelets ($10^3/\mu L$)	6.9	—	297–730	—
MPV (fL)	7.9	—	—	—
Polychromasia	Marked			
Howell–Jolly bodies	Few			
Echinocytes	Moderate			

[a]Colorado State University reference ranges.
[b]Fox (1988).
[c]Carpenter (2005).

Table 3.2. Plasma biochemical results.

		Reference[a]	Reference[b]	Reference[c]
Glucose (mg/dL)	88	95–140	94–207	63–134 (101)
BUN (mg/dL)	50	10–26	10–45	12–43 (28)
Creatinine (mg/dL)	0	0–0.5	0.4–0.9	0.2–0.6 (0.4)
Phosphorus (mg/dL)	7.2	3.0–5.5	4.0–9.1	5.6–8.7 (6.5)
Calcium (mg/dL)	8.1	8.0–9.7	8.0–11.8	8.6–10.5 (9.3)
Total protein (g/dL)	4.2	5.0–6.4	5.1–7.4	5.3–7.2 (5.9)
Albumin (g/dL)	2.6	2.9–4.1	2.6–3.8	3.3–4.1 (3.7)
Globulin (g/dL)	1.6	1.8–3.0	—	2.0–2.9 (2.2)
A/G ratio	1.6	1.0–2.2	—	1.3–2.1 (1.8)
Cholesterol (mg/dL)	112	70–200	64–296	—
Total bilirubin (mg/dL)	0.7	0–0.3	<1	—
CK (IU/L)	820	80–400	—	—
ALP (IU/L)	26	10–60	9–84	30–120 (53)
ALT (IU/L)	290	80–270	—	82–289 (170)
AST (IU/L)	346	30–75	28–120	—
GGT (IU/L)	49	1–15	—	5
Sodium (mg/dL)	145	147–153	137–162	146–160 (152)
Potassium (mg/dL)	4.7	3.3–4.5	4.5–7.7	4.3–5.3 (4.9)
Chloride (mg/dL)	115	114–120	106–125	102–121 (115)
Bicarbonate (mg/dL)	12.4	15–23	—	—
Anion gap	22	14–21	—	—
Calculated osmolality	301	—	—	—
Lipemia (mg/dL)	14	—	—	—
Hemolysis (mg/dL)	24	—	—	—
Icterus (mg/dL)	0	—	—	—

[a]Colorado State University reference ranges.
[b]Fox (1988).
[c]Carpenter (2005).

hemorrhage as indicated by the presence of melena on the physical examination.

Neutrophil concentrations are generally higher than lymphocyte concentrations in normal ferrets and they tend to increase in concentration, while lymphocytes decrease in concentration with increasing age. The total leukocyte count of healthy ferrets can be as low as 3,000/μL; therefore, ferrets are unable to develop a marked leukocytosis with inflammatory disease, and concentrations greater than 20,000/μL are unusual and a left shift is rare. In this case, the ferret has a leukopenia with slightly toxic neutrophils and a left shift indicative of a degenerative left shift. She has also a severe lymphopenia.

The increased serum blood urea nitrogen (BUN) concentration can be associated with dehydration, renal failure, or gastrointestinal hemorrhage. The nonexistent creatinine supports the idea of gastrointestinal hemorrhage; however, one must consider that in normal and azotemic ferrets, the plasma creatinine concentration is lower than that in dogs and cats. The mean plasma creatinine concentration of healthy ferrets is 0.4–0.6 mg/dL with a range of 0.2–0.9 mg/dL. As a result, a moderate increase in the plasma creatinine concentration (i.e., 1–2 mg/dL) in a ferret is significant and suggestive of renal disease. This, however, is not an issue in this case.

Evaluation of the liver in ferrets by laboratory testing is the same as that for those in dogs and cats. The plasma alanine aminotransferase (ALT) activity, which appears elevated in this case, is a sensitive and specific test for hepatocellular disease in ferrets. Ferrets with hepatocellular disease commonly have increased aspartate aminotransferase (AST) activity as well. Those with cholestasis likely have increased plasma alkaline phosphatase and γ-glutamyl transferase (GGT) activities. Ferrets rarely become icteric or have plasma bilirubin concentrations greater than 2.0 mg/dL, even when hepatobiliary disease is severe. In this case, the ferret likely has hepatocellular disease.

The causes of hypoproteinemia in ferrets are the same as those in dogs and cats. In this case, it is likely associated with significant blood loss from gastrointestinal hemorrhage.

The prognosis for survival in this ferret based on the physical examination, hemogram, and plasma biochemical profile is poor.

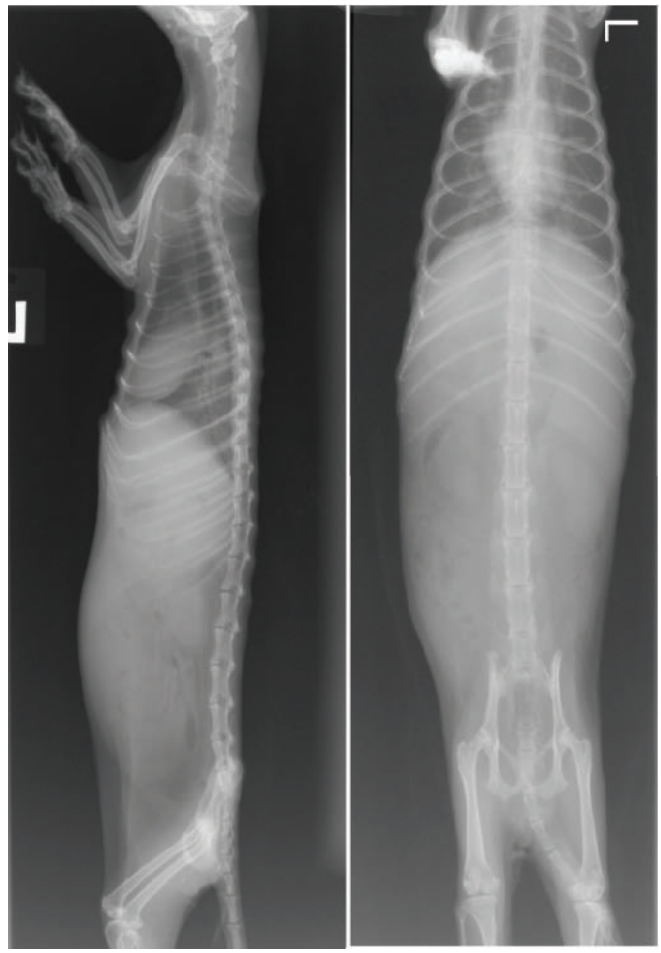


Fig. 3.3. The radiograph of dorsoventral position (right image) and left lateral position (left image).

Summary

The ferret was immediately transferred to the critical care unit for intravenous fluid therapy, correction of hypothermia, and treatment for *Helicobacter*-induced gastrointestinal ulcers. The treatment plan included doxycycline (2.5 mg PO BID), amoxicillin (11 mg PO TID), sucralfate (125 mg PO QID), and two beads from a 20 mg omeprazole capsule (PO daily). The ferret died within 12 hours following presentation. Gross necropsy findings revealed a large perforated ulcer at the gastric pylorus, associated with a marked amount of hemorrhage.