Exigo™ H400 and Exigo C200

Case book

Boule

Acknowledgement

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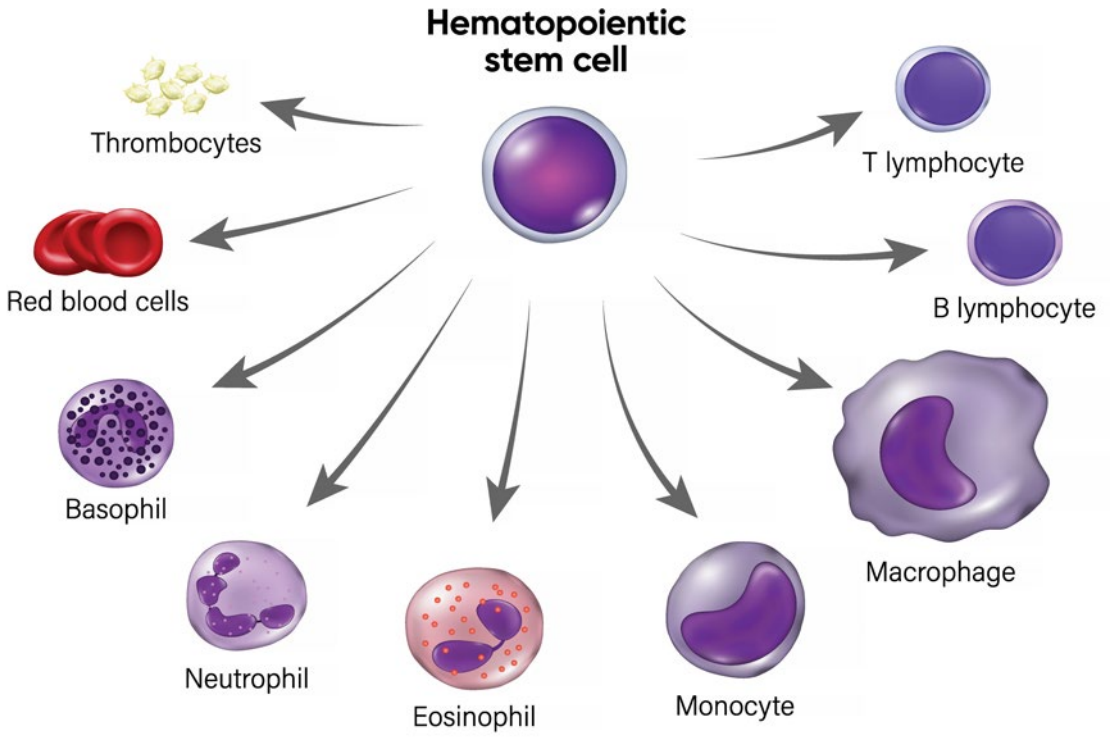
Content

Introduction

Hematology is the most common diagnostic test performed, tightly followed by clinical chemistry. To understand the results of a complete blood count (CBC) and a chemistry panel, it is important to understand the meaning of each parameter and what factors that might affect their concentration and production.

Hematology

All blood cells (white blood cells, red blood cells, and platelets) are produced in the bone marrow from pluripotent stem cells, from which they divide into various cell lineages and continue their maturation processes before being released into the blood stream.



Red blood cell and hemoglobin interpretation

The main task of red blood cells (RBCs), also called erythrocytes, is to deliver oxygen from the lungs to the body's tissues. Hemoglobin (HGB) is the oxygen carrying protein within the RBCs. A functional hemoglobin molecule consists of four subunits, each including an iron-containing heme group with the ability to bind to oxygen and carbon dioxide. Hemoglobin binds oxygen in the lungs where pH is high and carbon dioxide is low. The oxygen is released in the tissue where pH is low and replaced by carbon dioxide that is transported back to the lungs and exhaled. RBC count and HGB concentration in circulation therefore gives an indication of the oxygenation status. Anemia is characterized by poor oxygenation and is diagnosed as a low HGB concentration. Based on the hemoglobin concentration, anemias are divided into three different groups: hypochromic, normochromic, and hyperchromic.

The RBC and HGB reference intervals (normal range) can differ between species and even between breeds. For example, greyhounds have been shown to have relatively higher RBC counts and HGB concentration than other dog breeds. Younger animals also tend to have slightly lower RBC counts than adults.

Mean cell volume (MCV) represents the average RBCs size. Based on the MCV, anemias are divided into three different groups: microcytic, normocytic, and macrocytic.

Hematocrit (HCT) is a value calculated by the analyzer, this is the product of the mean cell volume (MCV) and the red blood cell count (RBC).

Red blood cell distribution width (RDW) is an index of variation in red blood cell volume within the red cell population. A high RDW indicates high variability in RBC size in a blood sample and could be seen with regenerative anemia or iron deficiency anemia.

Mean cell hemoglobin concentration (MCHC) is the calculated average concentration of HGB in the total RBC mass. A low MCHC can be observed in strongly regenerative anemias, because of an increased population of reticulocytes (immature RBCs) with low HGB content.

Mean cell hemoglobin (MCH) represents the calculated absolute amount of hemoglobin in the average RBC in a blood sample. MCH can be used to confirm different types of anemia (microcytic, normocytic, and macrocytic).

Platelet interpretation

Platelets (PLTs), or thrombocytes, are important in the blood clotting mechanism. To minimize blood loss when a blood vessel is damage, PLTs clump together, forming a thrombus. PLTs are also vital in many inflammation and healing processes, as they release various signaling substances and interact with many other types of cells.

High PLT counts, or thrombocytosis, can occur with drug administration (corticosteroids and β-adrenergic drugs), post-splenectomy, reactive conditions (neoplasia, chronic inflammatory diseases, immune-mediated diseases, trauma) and neoplasia. Young animals normally have higher platelet counts than adults.

Low PLT count, thrombocytopenia, can be due to a decreased PLT production (caused by infectious agents, immune-mediated inhibition of megakaryocytes, neoplasia, drugs), increased platelet consumption (acute, severe hemorrhage, platelet activation and aggregation) or through increased PLT destruction or clearance (immune-mediated thrombocytopenia, increased platelet sequestration). Patients with low platelet counts are at risk of bleeding or can have difficulty stopping the bleeding. Thrombocytopenia can also be inherited. Cavalier King Charles Spaniels and Akitas have a higher prevalence of thrombocytopenia due to a genetic mutation that affects early platelet formation.

Mean platelet volume (MPV) represents the average PLT size. Young platelets are often larger, although individual platelets can vary markedly in size within a given sample, even in healthy animals.

A common issue when counting platelets is pseudo-thrombocytopenia, a falsely low platelet count. Pseudo-thrombocytopenia can be caused by platelet clumping, and in such a case, often accompanied with an increase in MPV.

White blood cell interpretation

The white blood cells (WBCs), also known as leukocytes, are the body's defense against foreign matter and microbes. WBCs are important in response to infection and inflammation and are the "cleaners", removing dead or damaged cells and tissue.

In general, an inflammation, infection, or malignancy in the body will activate the defense and increase the WBCs (leukocytosis), whereas too few WBCs (leukopenia) is seen with viral infections, sepsis, or certain drug administrations (chemotherapy). As leukopenia poses an increased risk of severe infection, the state is detrimental and can be deadly.

The WBC count can also vary with age, for example, kittens have normal count at birth, but by 3 to 4 months of age, neutrophil (NEU) and lymphocyte (LYM) counts can exceed the adult levels and decrease to within the adult range by 5 to 6 months of age. Senior cats (> 10 years) have lower WBC counts, due to lower LYM and eosinophil (EOS) counts, than younger adults.

For dogs, the total WBC counts are higher at 3- to 8 weeks of age than at any later stage in life and are elevated compared to adult ranges until 4 months of age. Lymphocytosis is common in dogs younger than 6 months. WBC count tends to decrease from 2 years of age.

A high NEU count, neutrophilia, is commonly seen in various conditions. Physiologic neutrophilia is commonly seen with emotional distress, fear, and vigorous exercise. Neutrophilia as response to stress or steroids is seen with hyperadrenocorticism and exogenous corticosteroid administration. Neutrophilia as a manifestation of an acute inflammatory response is seen with a variety of diseases, for example, bacterial or other infections, immune-mediated diseases, neoplasia, or tissue necrosis.

A low NEU count, neutropenia can be seen with per-acute bacterial infections, extreme pyrexia, initial period of endotoxic and anaphylactic shock, retroviral infections, parvovirus infection, myelodysplastic disease, leukemia, cytotoxic drug therapy, to mention a few.

A high LYM count, lymphocytosis can accompany physiological states (emotional distress, fear, vigorous exercise) but can also be seen in reactive states (young animals, post vaccinations, prolonged immune stimulation, hyperthyroidism), hypoadrenocorticism, and neoplastic diseases (lymphoma).

A low LYM count, lymphopenia, can be a response to stress/steroid, an acute inflammatory response (any inflammatory response, viral infection, sepsis, or endotoxemia), lymphocyte loss (chylothorax, lymphangiectasia), decreased production (immunosuppressive drug therapy, feline immunodeficiency virus infection), and obstruction of lymph flow (inflammation, neoplasia).

A high EOS count, eosinophilia, is seen with ectoparasites and endoparasites, allergic diseases (feline asthma, eosinophilic broncho-pneumopathy, food hypersensitivity), inflammatory diseases (inflammatory bowel disease, eosinophilic myositis), neoplasia (mast cell tumor, lymphoma), infectious diseases (feline panleukopenia virus, feline infectious peritonitis, toxoplasmosis, upper respiratory tract infection, pyometra), hypoadrenocorticism.

A low EOS count, eosinopenia, can be a response to corticosteroid and as part of the stress leukogram.

A high monocyte (MON) count, monocytosis, is typically associated with chronic inflammation (infection, malignancy, tissue necrosis, internal hemorrhage, and pyogranulomatous inflammation), but can also be seen with acute inflammation (trauma, immune-mediated hemolytic anemia, and other immune-mediated states), a stress and steroid response, or compensatory secondary to neutropenia, and leukemia.

Biochemistry interpretation

Urea nitrogen is synthesized by hepatocytes from ammonia, generated by catabolism of amino acids derived from digestion of proteins in the intestines or from endogenous tissue proteins, and is excreted by the kidneys, colon, saliva, and sweat. Increased urea nitrogen is seen with increased catabolism (fever, severe burns, corticosteroid administration, starvation), increased protein digestion (upper gastrointestinal tract hemorrhage, diets high in protein and raw meat) or decreased glomerular filtration rate (prerenal, renal. or postrenal). Decreased urea nitrogen is seen with decreased protein intake or protein anabolism (dietary restriction of protein, in young animals), increased excretion (any cause of polyuria), and decreased production (liver disease, enzyme deficiencies in urea cycle).

Creatinine is produced as the result of normal metabolism of muscles and is filtered through the glomerulus. Increased creatinine can be seen with increased production (after recent cooked meat meal), decreased glomerular filtration (prerenal, renal, postrenal), and release from the muscle. Creatinine can be higher in premature and in new-born foals and heavily muscled horses. Creatinine concentrations are higher in greyhounds compared with other dog breeds. Decreased creatinine concentration can be seen with decreased muscle mass, increased glomerular filtration rate (pregnancy, portosystemic shunt), decreased production (loss of muscle mass).

Total protein is the summed concentration of all individual serum proteins and should be interpreted regarding albumins and globulins.

Albumin is synthesized in the liver and is catabolized in various tissues and makes a large contribution to plasma colloid osmotic pressure and as a carrier protein for many insoluble organic substances and drugs. Increased albumin concentration is seen with fluid loss, prednisolone administration, hyperadrenocorticism, and hepatocellular carcinoma. Hypoalbuminemia is seen with decreased production (liver failure, acute phase reaction response, increased oncotic pressure), loss (protein-losing enteropathies, protein-losing glomerulopathy, hemorrhage, severe exudative dermatopathies), and sequestration (protein-rich effusions).

Globulins consist of all non-albumin proteins and constitute a specific group of proteins that are produced in response to inflammatory stimuli, acute phase proteins, and immunoglobulins. Increase in total globulins can be seen with acute phase reactant response, inflammation, active immune response to antigenic stimulation (polyclonal gammopathy, monoclonal gammopathy, neoplasia, and non-neoplastic disorders like occult heartworm disease, lymphoplasmacytic enteritis, amyloidosis). Decreased globulin production is seen with acquired hypogammaglobulinemia, acquired immunodeficiencies, infectious diseases (feline leukemia virus, feline immunodeficiency virus, toxoplasmosis, canine distemper, demodicosis), and neoplasia.

Routine laboratory testing in liver disease includes measuring of activities of liver enzymes and bilirubin.

Alanine aminotransferase is increased with liver disease (e.g., hepatocellular carcinoma, hepatic insufficiency, advanced cirrhosis, chronic hepatitis), muscle disease, and hyperthyroidism.

Aspartate aminotransferase is increased with myopathies (muscle trauma, rhabdomyolysis), liver disease, and hyperthyroidism.

Alkaline phosphatase (AP) is increased with drugs (phenobarbitone, primidone, corticosteroids), hepatobiliary disease (structural and functional cholestasis, neoplasia, acute hepatocellular injury), hyperadrenocorticism, adrenal dysfunction, non-hepatic neoplasia, increased osteoblastic activity. AP is increased in young, growing animals of all species and is about 2 to 10 times higher than in adults. Some Siberian huskies and scottish Terriers have been reported to have higher AP values.

γ-Glutamyl trasferase (GGT) is increased with primary liver disease (cholangiohepatitis, cholelithiasis), gastrointestinal issues (right and left colonic displacement), and in racehorses (high GGT syndrome in racehorses). GGT is increased with biliary hyperplasia and structural cholestasis, renal disease, and hyperadrenocorticism.

Bilirubinemia can be seen in fasting horses and in young animals, hemolytic anemia, liver disease, and cholestasis.

Hyperglycemia can be seen post-prandially, with stress, pregnancy, diabetes mellitus, hyperadrenocorticism, acromegaly, hyperglucagonemia, hyperpituitarism in horses, transient hyperglycemia (hyperthyroidism, acute pancreatitis, sepsis, proximal duodenal obstruction).

Hypoglycemia can be seen with decreased production (glycogen storage disease, liver disease, juvenile hypoglycemia in toy and small breed dogs), decreased intake (starvation, malabsorption, high grain diet in horses), increased use (hypoglycemia of endurance horses and hunting dogs, insulinoma, leiomyoma, leiomyosarcoma, hepatica, renal tumors, hypopituitarism, hypoadrenocorticism).

Hypertriglyceridemia can be seen post-prandially, with certain drugs (corticosteroids), inflammation, hyperadrenocorticism, pancreatitis, excessive energy negative balance (starvation, anorexia, especially during pregnancy and lactation, hyperlipidemia syndrome in horses, familial hyperlipidemias in Siamese, domestic shorthair, and Himalayan cat breeds, familial hyperlipidemias in Miniature schnauzer, beagle, Brittany spaniel dog breeds).

Hypercholesterolemia can be seen with certain drugs (corticosteroids), nephrotic syndrome, hypothyroidism, cholestasis, hyperadrenocorticism, diabetes mellitus, pancreatitis, familial hypercholesterolemia in Briard, Rottweiler, Shetland sheepdog, and doberman dog breeds.

Hypocholesterolemia is seen with decreased absorption (protein-losing enteropathies, exocrine pancreatic insufficiency), decreased production (chronic liver disease, liver failure, portosystemic shunt), increased uptake of lipoproteins (rapidly proliferating tumor cells), and hypoadrenocorticism.

Hypernatremia can be seen with hypertonic fluid administration, water deficit (inadequate intake, hypotonic fluid loss with panting, vomiting, and diarrhea, third space loses) and increased sodium intake.

Increased lipase can be seen with acute pancreatitis, chronic renal insufficiency, decreased glomerular filtration, intestinal disease, and obstruction.

Hyponatremia is seen with diuretic therapy, hypotonic fluid administration, hyperosmolar states, volume overload, excessive water intake, hypertonic fluid losses, and hypotonic fluid losses (renal losses, gastrointestinal losses, third space losses).

Hyperkalemia is seen with hemolysis, leukocytosis, transcellular shifts (hyperkalemic poly-myopathy of horses), decreased renal excretion (uroabdomen, hypoaldosteronism, acidosis).

Hypokalemia is seen with decreased intake, transcellular shifts (insulin release, endotoxemia), and increased loss and (vomiting, abdominal stasis, gastric outflow obstruction, diarrhea, third space losses, renal losses).

Hyperchloremia can be seen with metabolic acidosis (renal failure, proximal and distal tubular acidosis) and compensatory metabolic acidosis.

Hypochloremia can be seen with drug administration (sodium rich fluids, diuretics), gastrointestinal and renal losses.

Hypercalcemia is seen with drug administration (thiazide diuretics, osteolysis (primary hyperparathyroidism, humoral hypercalcemia of malignancy), increased intestinal absorption (hypoadrenocorticism, hypervitaminosis D), decreased renal excretion, increased protein binding (hyperalbuminemia), and idiopathic hypercalcemia.

Hypocalcemia is seen with decreased protein binding (hypoalbuminemia), abnormal parathormone (primary hypoparathyroidism, pseudohypoparathyroidism, resistance to parathormone), decreased absorption of calcium (nutritional secondary hyperparathyroidism, hypovitaminosis D, renal secondary hyperparathyroidism, protein-losing enteropathy, hyperadrenocorticism), losses of calcium (renal losses, pregnancy or lactational), and sepsis.

Hyperphosphatemia can be seen with administration of phosphate containing compounds, increased intake (hypervitaminosis D, excess phosphate in diet), transcellular shifts (acute tumor lysis, severe soft tissue trauma), and decreased excretion (decreased glomerular filtration rate due to renal or post-renal azotemia, hypoparathyroidism, acromegaly, hyperthyroidism).

Hypophosphatemia can be seen with phosphate-binding antacids, deficient intake, and absorption (vitamin D deficiency), transcellular shifting (alkalemia due to respiratory alkalosis, catecholamines, insulin or glucose administration), increased losses (hyperparathyroidism, renal disease, hyperadrenocorticism, vomiting, diarrhea), and hepatic lipidosis.

Hypermagnesemia can be seen with excessive supplementation, reduced glomerular filtration in renal and post-renal azotemia, and massive tissue necrosis.

Hypomagnesemia is seen with decreased albumin, decreased intake, and excess loss (malabsorption, diarrhea, diuresis of any cause).

Normal

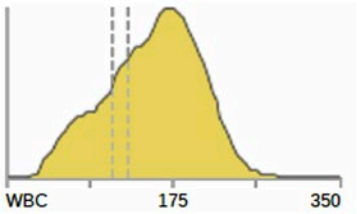
Case A01

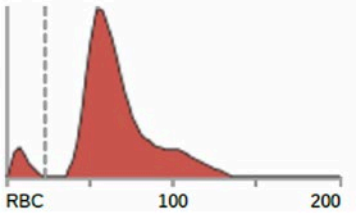
Healthy dog

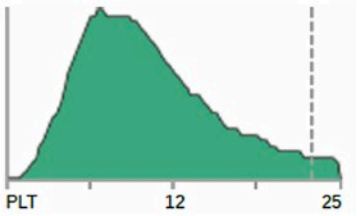
A healthy, 4-year-old male intact Belgian shepherd was admitted for a health examination prior to castration. Clinical examination was without remark. Thoracic X-ray was without remark.

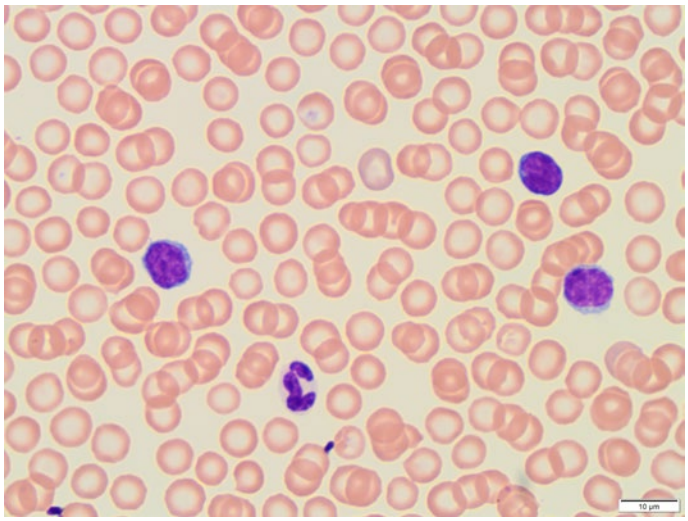
A complete blood count and biochemistry serum profile were performed.

Parameter	Results	Unit		Reference range		
WBC	7.9	10^9/l		6.0	17.0	10^9/l
LYM	1.5	10^9/l	20.0 %	0.9	5.0	10^9/l
MON	0.8	10^9/l	11.2 %	0.3	1.5	10^9/l
GRA	5.6	10^9/l	68.8 %	3.5	12.0	10^9/l
HGB	152	g/l		120	180	g/l
MCH	25.3	pg		19.5	25.5	pg
MCHC	362	g/l		320	385	g/l
RBC	6.04	10^{12}/l		5.50	8.50	10^{12}/l
MCV	69.8	fl		60.0	72.0	fl
HCT	42.2	%		37.0	55.0	%
RDW	13.0	%	53.1 fl	12.0	17.5	%
PLT	368	10^9/l		200	500	10^9/l
MPV	10.4	fl		5.5	10.5	fl









Microscope image

Blood smear from healthy dog.

Case A01

Parameter	Result	Indication	Reference interval	Unit
ALB	30.9		23.0–40.0	g/L
TP	59.7		49.0–82.0	g/L
GLOB	28.8		19.0–45.0	g/L
A/G	1.07			
TB	2		0.0–15.0	µmol/L
GGT	<2		0–10	U/L
AST	85	H	0–50	U/L
ALT	85		5–125	U/L
ALP	68		17–212	U/L
AMY	1251		400–1500	U/L
Crea	93.8		28.0–159.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	4.05		2.50–9.60	mmol/L
BUN/CREA	43.21		16.000–218.000	g/dl
GLU	5.53		4.11–7.94	mmol/L
TC	4.86		2.84–8.27	mmol/L
TG	0.37		0.00–1.13	mmol/L
tCO2	17.7		12.0–27.0	mmol/L
Ca	2.44		1.98–3.00	mmol/L
PHOS	1.67		0.81–2.19	mmol/L
Mg	0.79		0.68–1.09	mmol/L
K^+	4.55		3.50–5.80	mmol/L
Na^+	146.1		136.0–156.0	mmol/L
Cl^-	109.6		95.0–119.0	mmol/L

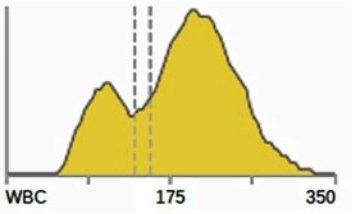
Normal

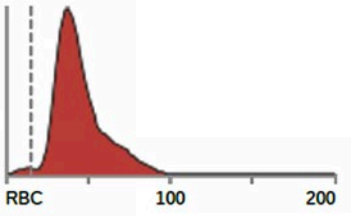
Case A02

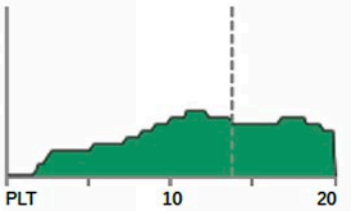
Healthy cat

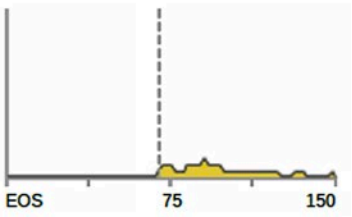
A healthy, 4-year-old male intact domestic shorthair was admitted for a health examination prior to castration. Clinical examination was without remark. Thoracic x-ray was without remark. A complete blood count and biochemistry profile were performed and showed no abnormalities. Low thrombocyte number was explained by macro-thrombocytes.

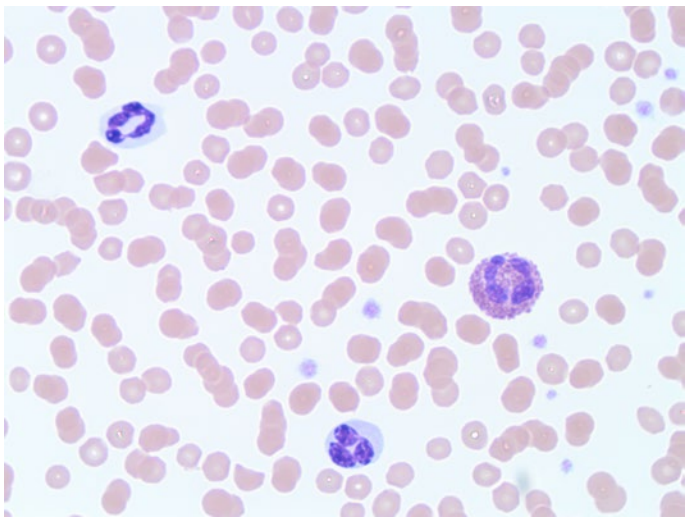
Parameter	Results	Unit		Reference range		
WBC	5.5	10^9/l		5.5	19.5	10^9/l
LYM	1.2	10^9/l	23.1 %	1.0	7.0	10^9/l
MON	0.3	10^9/l	6.1 %	0.2	1.0	10^9/l
NEU	3.4	10^9/l	62.1 %	2.8	13.0	10^9/l
EOS	0.4	10^9/l	8.7 %	0.1	1.0	10^9/l
HGB	109	g/l		80	150	g/l
MCH	15.4	pg		12.5	17.5	pg
MCHC	333	g/l		310	385	g/l
RBC	7.08	10^{12}/l		5.00	11.00	10^{12}/l
MCV	46.2	fl		39.0	50.0	fl
HCT	32.8	%		25.0	45.0	%
RDW	16.3	%	33.3 fl	14.0	18.5	%
PLT	115	10^9/l		200	500	10^9/l
MPV	9.0	fl		8.0	12.0	fl











Microscope image

Blood smear from healthy cat.

Case A02

Parameter	Result	Indication	Reference interval	Unit
ALB	33.6	H	21.0–33.0	g/L
TP	52.7	L	54.0–78.0	g/L
GLOB	19.1	L	23.0–52.0	g/L
A/G	1.76			
TB	1.7		0.0–8.6	µmol/L
GGT	<0	L	0–12	U/L
AST	21		0–80	U/L
ALT	42		0–88	U/L
ALP	112	H	0–59	U/L
AMY	628		0–900	U/L
Crea	98.2		0.0–141.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	10.35		3.30–10.70	mmol/L
BUN/CREA	105.379		27.000–182.000	
GLU	16.05	H	3.10–5.60	mmol/L
TC	5.85	H	1.80–3.90	mmol/L
TG	3.19	H	0.10–1.30	mmol/L
tCO2	15.6		13.0–25.0	mmol/L
Ca	2.46		2.00–3.00	mmol/L
PHOS	3.18	H	0.80–1.90	mmol/L
Mg	1.03		0.60–1.30	mmol/L
K^+	4.59		3.60–5.50	mmol/L
Na^+	145.8		145.0–157.0	mmol/L
Cl^-	113.8		110.0–130.0	mmol/L

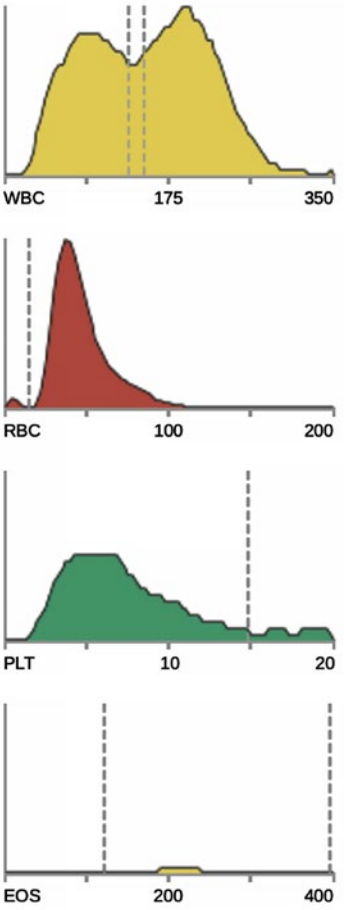
Normal

Case A03

Healthy horse

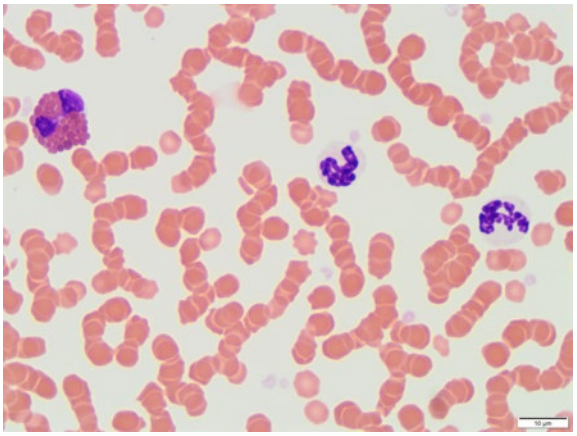
A healthy 15-year-old mare was brought for a health examination. Clinical examination was without remark. A complete blood count and biochemistry serum profile were performed.

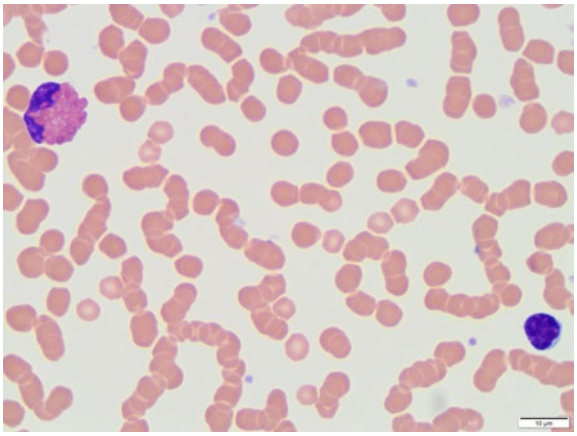
Parameter	Results	Unit		Reference range		
WBC	7.5	10^9/l		5.5	12.5	10^9/l
LYM	3.0	10^9/l	39.8 %	1.5	5.0	10^9/l
MON	0.6	10^9/l	8.5 %	0.2	1.0	10^9/l
NEU	3.7	10^9/l	49.0 %	3.0	7.0	10^9/l
EOS	0.2	10^9/l	2.7 %	0.1	1.0	10^9/l
HGB	119	g/l		110	190	g/l
MCH	15.8	pg		13.5	19.5	pg
MCHC	329	g/l	▼	340	405	g/l
RBC	7.51	10^{12}/l		6.50	12.50	10^{12}/l
MCV	48.1	fl		36.0	52.0	fl
HCT	36.1	%		32.0	52.0	%
RDW	18.1	%	38.5 fl	▲ 16.0	21.0	%
PLT	140	10^9/l		100	350	10^9/l
MPV	6.9	fl		5.5	11.0	fl



Microscope image

Blood smears from healthy horse.





Case A03

Parameter	Result	Indication	Reference interval	Unit
ALB	36.2	H	19.0–36.0	g/L
TP	72.7		56.0–79.0	g/L
GLOB	36.6		24.0–47.0	g/L
A/G	0.99			
TB	15		0.0–60.0	µmol/L
GGT	10		0–87	U/L
AST	339		100–600	U/L
ALT	<5	L	5–50	U/L
ALP	241		0–326	U/L
AMY	16		0–35	U/L
Crea	93.7		71.0–194.0	µmol/L
UA	<10.00			µmol/L
BUN	5.23		3.60–8.90	mmol/L
BUN/CREA	55.766		20.000–274.000	
GLU	6.96		3.56–8.33	mmol/L
TC	2.55		1.29–2.84	mmol/L
TG	0.55		0.30–0.76	mmol/L
tCO2	25.2			mmol/L
Ca	2.68		2.60–3.23	mmol/L
PHOS	0.81		0.58–1.81	mmol/L
Mg	1.12	H	0.71–1.01	mmol/L
K^+	4.48		3.00–5.30	mmol/L
Na^+	140.9		133.0–150.0	mmol/L
Cl^-	104.6		97.0–109.0	mmol/L

Normal

Case A04

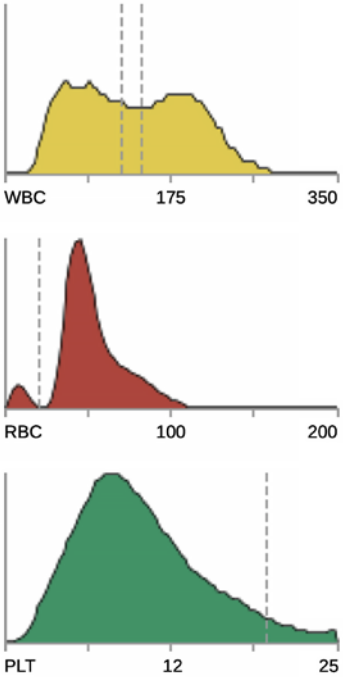
Healthy ferret with seasonal alopecia

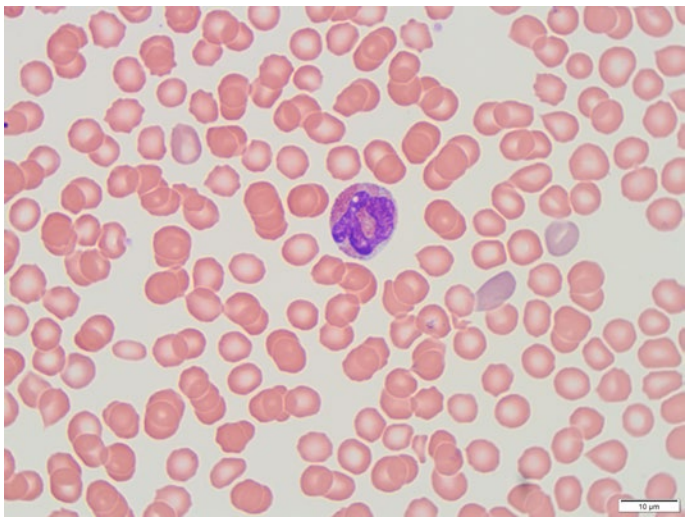
A healthy 1-year-old surgical neutered female ferret was admitted for a health examination.

The owner noticed that the hair on the tail had been falling off, and on the other parts of the body, the hair was falling only when they brush her. It seems to the owner that the hair is growing, but also falling out spontaneously. No signs of itching were present. She was fed with raw meat (turkey, chicken meat, and liver). Otherwise, she was very active, had not changed her behavior, had a good appetite with normal urination and defecation. She was regularly vaccinated against the infectious diseases.

Physical examination was without remark except for the alopecia that was present on the dorsal side of the tail, which extends from the tail base towards the caudal part. Suspected diagnosis: seasonal alopecia, active remnant ovaries, food allergy, less likely endocrinological diseases. A blood sample was collected from the vena *cephalica antebrachii* for complete blood count and biochemistry profile.

Parameter	Results	Unit		Reference range		
WBC	3.9	10^9/l	▼	5.6	10.8	10^9/l
LYM	1.7	10^9/l	45.0 %	1.7	4.7	10^9/l
MON	0.3	10^9/l	9.2 %	0.0	0.4	10^9/l
GRA	1.9	10^9/l	45.8 %	0.6	7.0	10^9/l
HGB	186	g/l	▲	152	177	g/l
MCH	17.4	pg		0.0	99.9	pg
MCHC	318	g/l	▼	320	380	g/l
RBC	10.67	10^{12}/l		6.80	11.80	10^{12}/l
MCV	54.8	fl	▲	43.0	51.0	fl
HCT	58.5	%	▲	46.0	57.0	%
RDW	15.3	%	41.1 fl	0.0	99.9	%
PLT	610	10^9/l		250	1000	10^9/l
MPV	9.7	fl		0.0	99.9	fl





Microscope image

Blood smear from healthy ferret.

Case A04

Parameter	Result	Indication	Reference interval	Unit
ALB	43.4	H	20.0–38.0	g/L
TP	78.2	H	52.0–73.0	g/L
GLOB	34.8		18.0–45.0	g/L
A/G	1.25			
TB	1.4		0.0–17.0	µmol/L
GGT	<2			U/L
AST	80		28–120	U/L
ALT	160		0–289	U/L
ALP	52		0–84	U/L
AMY	43			U/L
Crea	39		18.0–80.0	µmol/L
UA	16.66			µmol/L
BUN	11.67		3.60–16.10	mmol/L
BUN/CREA	299.437			
GLU	7.43		5.22–11.50	mmol/L
TC	6.93		1.65–7.65	mmol/L
TG	1.22			mmol/L
tCO2	17.9			mmol/L
Ca	2.56		2.00–2.95	mmol/L
PHOS	1.95		1.55–2.87	mmol/L
Mg	1.18			mmol/L
K^+	5.59		4.60–7.60	mmol/L
Na^+	151.3		139.0–169.0	mmol/L
Cl^-	113.9		106.0–125.0	mmol/L

Further diagnostics were recommended to the owner (the ultrasound of the abdomen and determination of the sex hormones in the serum). The hair had completely grown and stopped falling off, which indicated that it was a seasonal alopecia. No treatment was performed, and the outcome was good.

Like other members of the family *Mustelidae*, female ferrets are seasonally polyestrous animals. The sexual cycle (breeding season) is influenced by the length of the daylight (by extending the photoperiod) from 12 to 14 hours a day. The main role is played by melatonin, which is secreted in the dark, thus inhibits the secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus and inhibits the entry into the sexual cycle. Melatonin plays an important role in body weight and coat quality during and outside the breeding season.

In the wild, ferrets breeding season starts from early April until the end of July, or early August. Adult males come into the breeding condition late in January, and females begin to come in estrus during the late March. Under winter conditions, when hours of daylight decrease to less than 12 hours daily, the pineal gland secretes more melatonin, which induces prominent physiological changes, among which is the quality of the coat growth of the long winter hair and undercoat.

Ferrets shed seasonally, twice a year. During springtime, they shed off winter coat and have thinner summer coat, and during fall for winter coat. Usually, the duration of the shedding is around 2 to 3 weeks. If no regrowth of hair occurs during the shedding season, alopecia is then considered a non-physiological condition, especially in middle-aged surgically castrated individuals. It should be noted that alopecia appears most often in the spring and that it has the character of seasonal recurrence.

Therefore, the ferrets may go through several episodes of hair loss and regrowth before the loss becomes irreversible. Based on this difference in the dynamics of hair loss, it is possible to distinguish the seasonal process from hyperandrogenism. Permanent hair loss occurs due to the exogenization and atrophy of hair follicles and adnexal glands, follicular keratosis, epidermal thinning and orthokeratotic hyperkeratosis. In the occurrence of hyperandrogenism, the age when the gonadectomy was performed is one of the causes. Previous study has shown that the average time interval between the gonadectomy and the diagnosis is 3.5 years.

Main differential diagnosis includes hyperadrenocorticism (hyperandrogenism) and active remnant ovaries in surgical castrated ferrets, and prolonged estrus or persistent estrus (hyperestrogenism) in intact female ferrets (a life-threatening condition). The other possible causes are bacterial dermatitis, mange, dermatophytosis, and food allergy.

Pathological WBC counts

Case B01

Eosinophilic lung disease in dog

A nine-month-old, female-intact Labrador retriever, Lana, was presented to the emergency service due to progressive respiratory signs. She started coughing (with gagging and retching) 4 days ago. Owners took her to the referring veterinarian who gave her antibiotics (amoxicillin clavulanic acid). According to the owners, she had no response to therapy, her respiratory efforts increased, she was refusing food, and became lethargic. She was currently on her vaccines and ecto/endoparasite prophylaxis.

General physical examination revealed that she was depressed and in respiratory distress. Mucous membranes were red, CRT 1 second. Crepitations were heard bilaterally and diffuse over the lungs. Slight pressure on the trachea made Lana cough (with foamy sputum) very frequently. Her body temperature was 39°C, pulse 132/min and respiration 64/min. No other abnormalities were detected.

Complete blood count revealed severe eosinophilia with neutropenia and monocytopenia. Red blood cell count was lower but with normal haematocrit. Serum biochemistry was within normal limits.

Radiographs revealed a diffuse bronchointerstitial pattern.

A presumptive diagnosis of an eosinophilic lung disease was made. Suggested further workup, which included bronchoscopy with bronchoalveolar lavage sent for cytology and microbiology (to exclude infections agents) and heartworm tests and faecal examinations for pulmonary parasites (sedimentation, Baermann, and flotation concentrating techniques).

Bronchoscopy: trachea was hyperemic and, at the beginning of the second third of the trachea mucosa, was covered with mucopurulent contents. The trachea collapsed at the bifurcation. The right cranial, middle, and caudal lobes were filled with mucopurulent content that severely occludes the bronchi. The finding was the same as on the right side. Cytology from bronchoalveolar lavage suggested an eosinophilic lung disease. Culture of bronchoalveolar lavage fluid was negative. Heartworm tests and faecal examinations: negative.

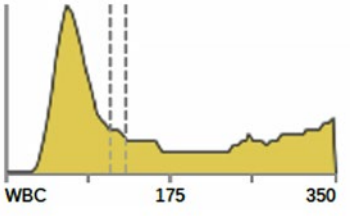
While waiting for the test results, Lana was given oxygen therapy (via nasal tubes) and crystalloid infusion. In additions, he was given inhalation and cupping.

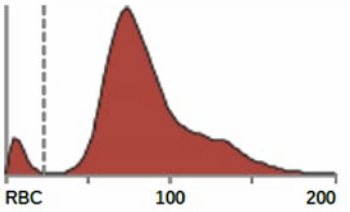
A diagnosis of eosinophilic lung disease was made, and Lana was started on immunosuppressive doses of corticosteroids.

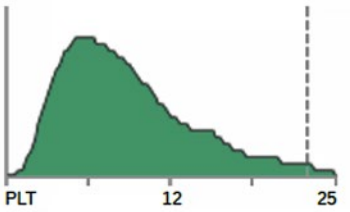
Two days after starting the therapy, Lana showed improvement, she started eating on her own, and showed interest in her surroundings. After two more days her respiratory rate became normal. She was discharged the day after and scheduled for a follow up.

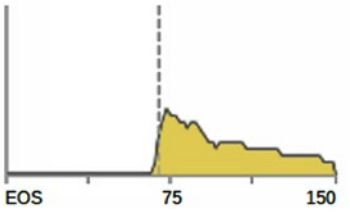
Case B01

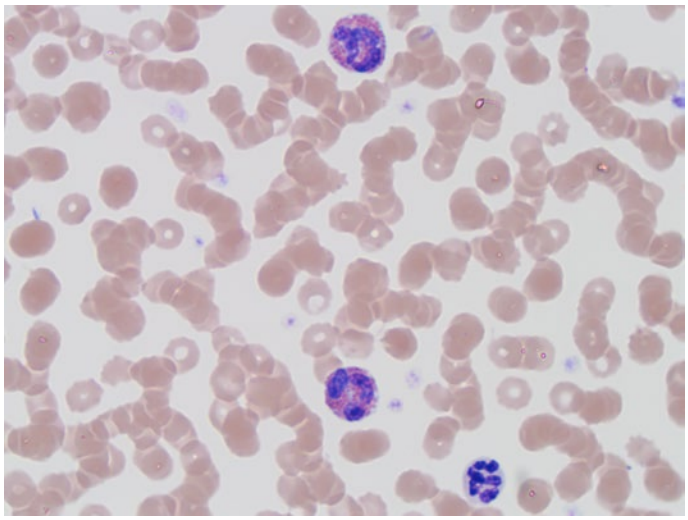
Parameter		Results	Unit		Reference range		
WBC		8.2	10^9/l		6.0	17.0	10^9/l
LYM	LM	2.4	10^9/l	29.6 %	0.9	5.0	10^9/l
MON	LM	0.2	10^9/l	▼ 3.5 %	0.3	1.5	10^9/l
NEU	LM	1.4	10^9/l	▼ 16.7 %	3.5	12.0	10^9/l
EOS		4.3	10^9/l	▲ 50.2 %	0.1	1.5	10^9/l
HGB		134	g/l		120	180	g/l
MCH		28.8	pg	▲	19.5	25.5	pg
MCHC		323	g/l		320	385	g/l
RBC		4.64	10^{12}/l	▼	5.50	8.50	10^{12}/l
MCV		89.1	fl	▲	60.0	72.0	fl
HCT		41.4	%		37.0	55.0	%
RDW		16.4	%	86.4 fl	▲ 12.0	17.5	%
PLT		237	10^9/l		200	500	10^9/l
MPV		9.2	fl		5.5	10.5	fl











Microscope image

Blood smear from dog with eosinophilic lung disease showing eosinophilia.

Case B01

Parameter	Result	Indication	Reference interval	Unit
ALB	31.8		26.0–33.0	g/L
TP	66.1		55.0–75.0	g/L
GLOB	34.2		19.0–45.0	g/L
A/G	0.93			
TB	1.4		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	27		0–82	U/L
ALT	45		0–88	U/L
ALP	41		20–156	U/L
AMY	683		0–1600	U/L
Crea	115.5		44.0–140.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	6.57		3.30–8.30	mmol/L
BUN/CREA	56.904		16.000–218.000	
GLU	7.37	H	3.60–6.50	mmol/L
TC	6.53		3.50–7.50	mmol/L
TG	0.67		0.20–1.30	mmol/L

Eosinophilic lung disease

Eosinophilic lung disease, also known as eosinophilic bronchopneumopathy, encompasses a range of inflammatory lung conditions primarily characterized by an infiltration of eosinophils. These conditions can affect either the airways or the interstitium, with various names like eosinophilic pneumonia (EP), eosinophilic bronchopneumopathy (EBP), and pulmonary infiltrates with eosinophilia.

In dogs, eosinophilic pulmonary granulomatosis is a severe form of this disease, marked by the development of nodules and often hilar lymphadenopathy. This condition must be distinguished from mycotic infection and neoplasia. While the names used to describe these conditions are descriptive, they likely encompass a variety of hypersensitivity disorders of the lung.

Identifying the underlying cause of eosinophilic inflammation is crucial. This can include heartworms, pulmonary parasites, fungal/infection agents, drugs, allergic response, and toxin exposure. In many cases, however, no underlying cause is found. Eosinophilic pulmonary granulomatosis is strongly linked with heartworm disease.

EBP is a well-reported but idiopathic condition of young to middle-aged dogs (slight female predominance). A breed predisposition has been reported in Siberian husky, Alaskan malamute, Labrador retriever, and Rottweiler breeds, but all breads can be affected.

Affected dogs typically present with progressive respiratory signs such as coughing (sometimes with gagging and retching), increased respiratory efforts, and exercise intolerance. Systemic signs like anorexia and weight loss are usually mild.

Diagnosis involves imaging studies like thoracic radiographs, which may show a diffuse interstitial or bronchointerstitial pattern. Peripheral eosinophilia on the complete blood count is not present in all animals. Bronchoscopy with bronchoalveolar lavage or lung biopsy (if needed) may be required to confirm the diagnosis. The lavage fluid is sent to cytology and microbiology. The final diagnosis is confirmed by the results of cytology, but other causes must be ruled out. Heartworm tests and fecal examinations for pulmonary parasites (sedimentation, Baermann, and flotation concentrating techniques) should be submitted.

Treatment aims to address any underlying diseases and manage inflammation. Glucocorticoids, such as prednisone, are commonly used to suppress inflammation and they are tapered to effect. Inhaled steroids may also be effective, particularly for bronchial involvement. Dogs with eosinophilic granulomatosis may require more aggressive immunosuppressive therapy, often involving a combination of glucocorticoids and cytotoxic agents like cyclophosphamide.

The prognosis for dogs with eosinophilic lung disease varies depending on the severity of the signs and the underlying causes. Generally, the prognosis is fair to good, but it can be guarded in cases of severe eosinophilic pulmonary granulomatosis. Regular monitoring and follow-up are essential to assess the response to treatment and adjust therapy as needed.

Pathological WBC counts

Case B02

Rabbit neoplasia

An 8-years-old, intact male dwarf rabbit, Mucko, was presented initially due to an enlarged right testis that the owner noticed two weeks ago. Other changes were not reported by the owner. The rabbit had a good appetite, behavior and activity, normal water intake, regular urination, and defecation. Until now, he was in good health. Suspected diagnosis: Testicular neoplasm.

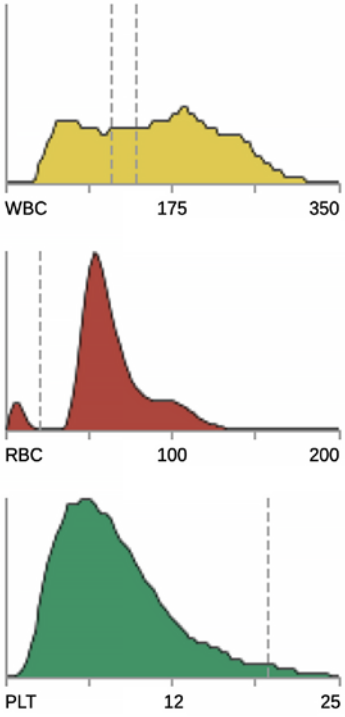
Physical examination was without remark, except for the enlarged right testicle with oval shape, approximately 3 cm in diameter, harder consistency on palpation and painless. The left testicle was in the inguinal canal, it was small, with a soft consistency on palpation.

A blood sample was collected for the complete blood count and biochemistry profile. The complete blood count revealed no clinically relevant abnormalities except mild leukopenia.

The thorax and abdomen X-ray imaging were without remark. On ultrasound examination, the right testis was enlarged, hypoechoic with generalized nodular changes, and rarely disseminated mineralization. The left testis was atrophic. The gonadectomy was performed under general anesthesia. A pathohistological examination of both testes was performed to establish a diagnosis and prognosis.

The pathohistological examination established the diagnosis of diffuse seminoma of the right testis, with atrophy and degeneration of the left testis. It appeared that the tumor had been completely excised, but considering that seminomas can metastasize, it was recommended to rule out their existence (lungs, lymph nodes) by CT.

Parameter	Results	Unit		Reference range		
WBC	3.3	10^9/l	▼	6.3	10.1	10^9/l
LYM	1.0	10^9/l	▼ 30.0 %	3.4	7.0	10^9/l
MON	0.3	10^9/l	10.1 %	0.1	0.5	10^9/l
GRA	2.0	10^9/l	59.9 %	1.5	3.2	10^9/l
HGB	143	g/l		115	151	g/l
MCH	21.2	pg		21.1	24.5	pg
MCHC	316	g/l		295	339	g/l
RBC	6.74	10^{12}/l		5.20	6.80	10^{12}/l
MCV	67.2	fl		64.6	76.2	fl
HCT	45.3	%		36.6	47.4	%
RDW	12.6	%	45.6 fl	0.0	99.9	%
PLT	359	10^9/l		250	610	10^9/l
MPV	8.0	fl		0.0	99.9	fl



Case B02

Parameter	Result	Indication	Reference interval	Unit
ALB	48.3	H	22.0–37.0	g/L
TP	70		55.0–72.0	g/L
GLOB	21.7	L	25.0–48.0	g/L
A/G	2.23			
TB	1.4		0.0–14.0	µmol/L
GGT	<2		0–14	U/L
AST	42		0–98	U/L
ALT	16		5–106	U/L
ALP	53		20–145	U/L
AMY	250		200–378	U/L
Crea	100.6		63.0–159.0	µmol/L
UA	<10.00		0.00–130.00	µmol/L
BUN	5.41		3.60–10.00	mmol/L
BUN/CREA	53.791		21.000–236.000	
GLU	8.8		4.17–9.50	mmol/L
TC	0.57	L	0.90–2.45	mmol/L
TG	2.22	H	0.30–1.76	mmol/L

Testicular neoplasms

Testicular neoplasms are not commonly reported in rabbits (the incidence is around 1.9%). It most often occurs in adult male rabbits over 2 years of age with an average age of 7.5 years, and they occur as a primary neoplasia.

The most prevalent tumor types are:

- Interstitial cell tumors (most common are Leydig cell tumors)
- Sertoli cell tumors
- Other tumors (seminomas, teratomas, Leiomyosarcomas).

Most of the mentioned tumors tend to be benign, but there are also reported cutaneous scrotal malignant melanoma. Different types of tumors can be found in testes, and they can be hormone secreting functional or non-functional tumors.

Common clinical signs and clinical manifestations are increased size of one or both testes, decreased size in the contralateral testis, behavioral changes and gynecomastia in hormone functional tumors, weight loss, and dysuria.

It is important to note that testes of physiological size can also be affected by tumor. During clinical examination, there is a palpated and enlarged, firm or nodular, non-painful testis. An ultrasound examination of the testes can detect a mass or lesion, and heterogeneous echogenicity. Definitive diagnosis is achieved by surgical procedure (gonadectomy) and histopathologic examination.

The differential diagnosis for testicular enlargement is testicular torsion, which is manifested by testicular abnormal position, swelling, pain, depression; and bacterial orchitis. The treatment is gonadectomy, which is often curative, and prognosis is good to guarded, it depends on the nature of the tumor and presence of metastasis.

Infections

Case C01

Canine babesiosis

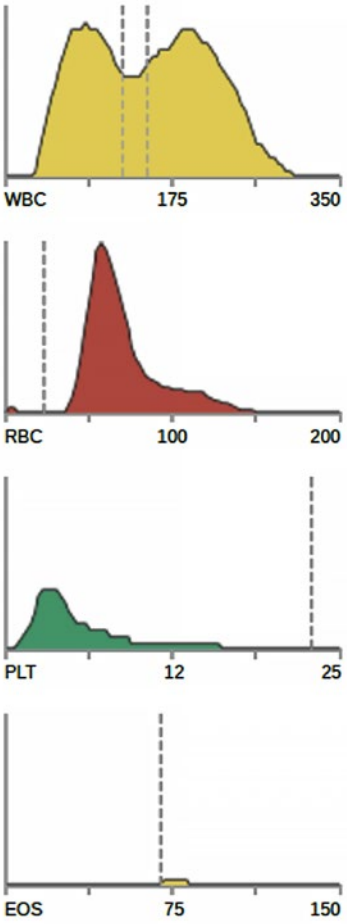
A three-year-old, male castrated golden retriever, Pico, was admitted on clinic for internal disease for diagnostics of acute lethargy and anorexia. The owner stated that Pico also had diarrhea for the last several days. Pico had no chronic illnesses, and he was not taking any therapy. He had been eating commercial dry food. He was vaccinated for rabies and infectious disease. He did not have protection for ectoparasites.

On the clinical exam, Pico walked into the clinic, interested in the environment, BCS 5/9. Visible mucous membranes pale, CRT 2 sec. Peripheral lymph nodes were generally enlarged, the size of a walnut, painless on palpation. Heart tones are loud and clear, rhythmic with no audible murmur. Auscultatory over the lungs - normal breathing sound. The abdomen was soft and compressible on palpation, no palpable organomegaly or foreign body. Vitals: temperature: 39.8°C, heart rate: 88/min, breathing: 32/min.

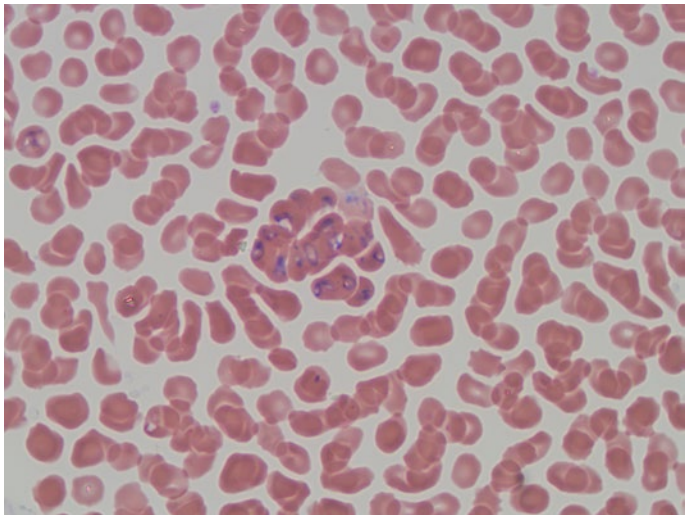
A thoracic radiographic study included two projections, left lateral (LL) image and a ventrodorsal (VD) image, showed no abnormalities. An abdominal radiographic study, including left lateral (LL) image and a ventrodorsal (VD) image, showed enlarged spleen.

Complete blood count revealed anemia and severe thrombocytopenia. Biochemistry profile showed mild hypoproteinemia and hypoalbuminemia. On blood smear cytology, erythrocyte inclusions were found, which corresponded to *Babesia canis* infection.

Parameter	Results	Unit	Reference range
WBC	6.7 10^9/l		6.0 – 17.0 10^9/l
LYM	2.6 10^9/l	38.5 %	0.9 – 5.0 10^9/l
MON	0.6 10^9/l	9.8 %	0.3 – 1.5 10^9/l
NEU	3.5 10^9/l	▼ 51.3 %	3.5 – 12.0 10^9/l
EOS	0.0 10^9/l	▼ 0.4 %	0.1 – 1.5 10^9/l
HGB	109 g/l	▼	120 – 180 g/l
MCH	24.0 pg		19.5 – 25.5 pg
MCHC	332 g/l		320 – 385 g/l
RBC	4.55 10^{12}/l	▼	5.50 – 8.50 10^{12}/l
MCV	72.2 fl	▲	60.0 – 72.0 fl
HCT	32.9 %	▼	37.0 – 55.0 %
RDW	12.5 %	49.2 fl	12.0 – 17.5 %
PLT	54 10^9/l	▼	200 – 500 10^9/l
MPV	6.6 fl		5.5 – 10.5 fl



Case C01



Microscope image

Blood smear from dog with *Babesia canis* infection showing agglutinate with babesiosis.

Parameter	Result	Indication	Reference interval	Unit
ALB	24.9	L	26.0–33.0	g/L
TP	54.3	L	55.0–75.0	g/L
GLOB	29.4		19.0–45.0	g/L
A/G	0.85			
TB	6.3		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	71		0–82	U/L
ALT	73		0–88	U/L
ALP	145		20–156	U/L
AMY	710		0–1600	U/L
Crea	88.3		44.0–140.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	8.27		3.30–8.30	mmol/L
BUN/CREA	93.687		16.000–218.000	
GLU	5.16		3.60–6.50	mmol/L
TC	6.71		3.50–7.50	mmol/L
TG	1.08		0.20–1.30	mmol/L
tCO2	12.9		12.0–27.0	mmol/L
Ca	2.13		2.10–3.10	mmol/L
PHOS	1.22		0.70–2.10	mmol/L
Mg	0.78	L	0.80–1.10	mmol/L
K^+	4.56		3.60–5.80	mmol/L
Na^+	145.5		140.0–155.0	mmol/L
Cl^-	113.8	H	90.0–110.0	mmol/L

Based on the signalment, history, clinical exam, and laboratory findings a suspicion of babesiosis without complication was made. Pico received Imidocarb dipropionate and antiemetic and was scheduled to come on recheck to check the hematocrit levels and to rule out babesiosis complication. On the next day, hematocrit levels were slightly higher with good regeneration signs of both erythrocytes and thrombocytes. Pico was discharged without any complication. Considering fast response of treatment, the prognosis was good.

Case C01

Canine Babesiosis

Canine Babesiosis is one of the most common tick-born disease worldwide discovered in the late 19th century. There has been discovered more than 100 babesia species in other animals, in dogs most common are *Babesia canis, Babesia gibsoni, Babesia rossi* and *Babesia vogeli*. Babesia prevalence in dogs varied considerably based on parasite species and geographic location, and infection occurs when a Babesia-infected tick bites a dog and releases Babesia sporozoites into the dog's bloodstream. Babesiosis is an important and life-threatening hemolytic disease where > 10% of dogs may die despite treatment. Babesiosis can induce hemolytic anemia secondary to intravascular hemolysis, immune-mediated destruction of erythrocytes (IMHA) and mechanical damage to erythrocytes during parasite migration. Thrombocytopenia may also occur, possibly from immune-mediated platelet destruction, platelet sequestration in the spleen. Dogs infected with Babesia spp. may have no signs of disease (subclinical) or may have severe or chronic to intermittent disease. Severe signs of disease are largely due to red blood cell and platelet destruction. Signs of uncomplicated babesiosis commonly include fever, pallor, weakness, pinpoint bleeding (petechia), splenomegaly and jaundice. Much of the disease process in babesiosis could also be explained by the host inflammatory response to the parasite, induction of acute phase response and activation of coagulation system. These could, in severe cases lead to systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS). The diagnosis of babesiosis is made by demonstration of the parasites within the infected erythrocytes in thin blood smears stained with May-Grünwald-Giemsa stain or using PCR (polymerase chain reaction). For *B. canis*, treatment with imidocarb dipropionate is recommended. Dogs with severe signs will require additional supportive therapy (e.g., blood transfusion, fluid support) and sometimes intensive care support). Prognosis can vary from great to poor depending on severity and number of complications.

Infections

Case C02

Sepsis in dog

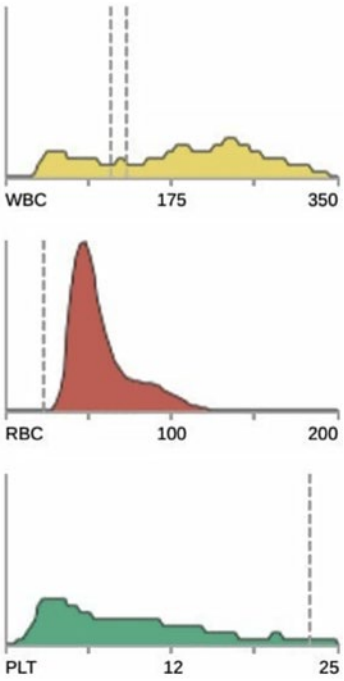
A 6-year-old male intact Akita Inu was presented initially to the clinic due to a lethargy, refusing food and vomiting. General physical examination revealed that he was very calm, body condition score 4/9. Mucous membranes were red, dry, slightly tacky, CRT was 2.5 s. Temperature, pulse, and respirations were all within normal limits. Lymph nodes were normal for the size of the animal. Cardiac auscultation: heart sounds were rhythmic, there was no heart murmur. Auscultation of the thorax was normal. Abdomen was very tense. With digito-rectal examination, enlarged prostate was palpated.

Blood samples were taken. The complete blood showed severe leukopenia and thrombocytopenia. A-FAST (abdominal fast) was done. Hypertrophy of the prostate and secondary signs of the prostatitis and peritonitis were seen. In the cysto–colic (CC) window there was free fluid, but not enough for the punction. Based on the signalment, history, clinical exam findings, and laboratory tests, a suspicion of sepsis was made.

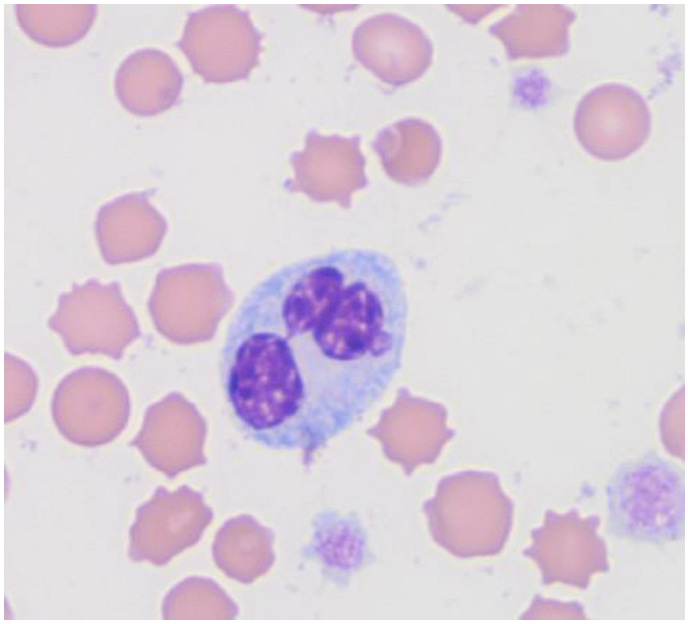
The dog received a shock dose of IV crystalloids 60 to 90 mL/kg, and then aggressive dose of fluid therapy was continued. After initial therapy temperature was 40°C and blood pressure was 80 mmHg. Glucose dropped to 1.8 mmol/L and the dog was numb. Coagulation tests (PT and aPTT) were prolonged.

The owners requested euthanasia due to high financial costs and poor prognosis.

Parameter		Results	Unit		Reference range		
WBC		1.8	10^9/l	▼	6.0	17.0	10^9/l
LYM	LW	0.4	10^9/l	▼ 23.4 %	0.9	5.0	10^9/l
MON	LW	0.0	10^9/l	▼ 4.9 %	0.3	1.5	10^9/l
NEU	LW	1.4	10^9/l	▼ 71.7 %	3.5	12.0	10^9/l
EOS		0.0	10^9/l	▼ 0.0 %	▼ 0.1	1.5	10^9/l
HGB		180	g/l		120	180	g/l
MCH		19.9	pg		19.5	25.5	pg
MCHC		335	g/l		320	385	g/l
RBC		9.04	10^{12}/l	▲	5.50	8.50	10^{12}/l
MCV		59.3	fl	▼	60.0	72.0	fl
HCT		53.6	%		37.0	55.0	%
RDW		13.8	%	41.7 fl	12.0	17.5	%
PLT		88	10^9/l	▼	200	500	10^9/l
MPV		9.2	fl		5.5	10.5	fl



Case C02



Microscope image

Blood smear from a dog with sepsis showing neutrophil with basophilic cytoplasm. Toxic neutrophil changes include retention of cytoplasmic features of immaturity and include foamy basophilic cytoplasm.

Parameter	Result	Indication	Reference interval	Unit
ALB	34.4	H	26.0–33.0	g/L
TP	65.2		55.0–75.0	g/L
GLOB	30.7		19.0–45.0	g/L
A/G	1.12			
TB	4.5		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	277	H	0–82	U/L
ALT	91	H	0–88	U/L
ALP	503	H	20–156	U/L
AMY	445		0–1600	U/L
Crea	189.4	H	44.0–140.0	µmol/L
UA	22.13		0.00–60.00	µmol/L
BUN	8.71	H	3.30–8.30	mmol/L
BUN/CREA	45.969		16.000–218.000	
GLU	7.74	H	3.60–6.50	mmol/L
TC	6.92		3.50–7.50	mmol/L
TG	4.21	H	0.20–1.30	mmol/L

Sepsis

Sepsis and septic shock are true life-threatening emergencies. Sepsis is a clinical syndrome that results from an infection that can be bacterial, fungal, protozoal, or viral in origin. For sepsis to develop, a complex pathway derived from systemic inflammatory response syndrome (SIRS) in response to an infection must occur. SIRS is the clinical manifestation of systemic inflammation in response to infectious or non-infectious causes.

The pathophysiology of sepsis is complex, but the major homeostatic changes include loss of vasomotor tone, injury to the endothelial cell layer and dysregulation of inflammation and coagulation. Vasomotor tone is lost due to the overproduction of NO_2, which causes systemic vasodilation and leads to a distributive shock state. Distributive shock occurs when there is ineffective or inappropriate circulation and distribution of blood volume. This leads to a maldistribution of blood flow, in which vessels dilate and create peripheral blood pooling. During vasodilation, the vessels expand, making the normal blood volume insufficient and causing the blood to be displaced away from the heart and central circulation. Endothelial injury occurs

from microcirculatory derangements that increase vascular permeability. This leads to vascular fluid shifts, bacterial translocation, and impaired oxygen delivery to tissues. Dysregulation of the inflammatory response occurs due to cytokine release, which upregulates tissue factor levels to initiate the coagulation cascade. The normal coagulation cascade involves activation of the platelet plug and anticoagulant pathways to maintain hemostasis; during sepsis, these processes are inhibited due to decreased levels of antithrombin. This increases the patient's risk of disseminated intravascular coagulopathy (DIC).

When the patient becomes affected by SIRS, multi-organ dysfunction syndrome (MODS) can occur. MODS is the physiologic derangements of major body systems (cardiovascular, respiratory, neurological, renal, hepatic, GI) associated with the progression of uncontrolled sepsis and SIRS.

Common causes of sepsis include abdominal disease (penetrating trauma, pancreatic abscess, hepatic abscess), GI disease (ulceration/perforation from long-term NSAID usage, foreign body, gallbladder rupture), surgical site dehiscence, cardiac disease (endocarditis), pulmonary/pleural space disease (pneumonia, lung abscess, pyothorax), renal disease (pyelonephritis, cystitis, renal abscess, uroabdomen), reproductive disease (pyometra, mastitis, prostatitis), and injured soft tissue (traumatic wounds, osteomyelitis, infectious joint effusions).

Clinical signs include lethargy, weakness, anorexia, abdominal discomfort, vomiting, diarrhea, increased respiratory rate or effort.

There are studies in both human and veterinary medicine looking to identify reliable and specific biomarkers of inflammation to assess the host inflammatory response to infection.

Management of a septic patient is centered on fluid therapy, antibiotic therapy, infectious source control, and overall supportive care. Aggressive fluid therapy is necessary to maintain circulatory status. Intravenous isotonic crystalloids are the mainstay fluid type, as they have the most similar composition to patient's extracellular fluid compartment. As septic patients suffer from a distributive shock state, shock volumes of fluids need to be initiated. The shock volume of crystalloid solutions is equal to an animal's blood volume and varies slightly depending on the reference source and species. In canines, the shock dose of IV crystalloids is 60 to 90 mL/kg. In felines, the shock dose of IV crystalloids is 45 to 60 mL/kg.

Administration of IV antibiotic therapy should be initiated as soon as possible after recognition of sepsis.

Vasopressors (norepinephrine, dopamine) may be indicated if there's no cardiovascular improvement with fluid resuscitation. They should never be used before fluid therapy.

Finding the source of infection is critical to patient outcome. Usually, those patients need surgical intervention. Patients with sepsis or septic shock are considered high-risk anesthesia candidates (ASA IV/V).

Infections

Case C03

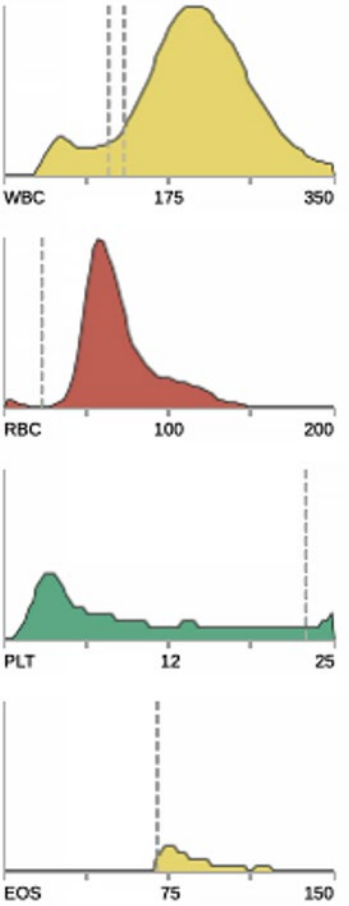
Peritonitis in dog

A 10-year-old, female-intact, mixed breed dog, Nes, was admitted because of fever that was treated by the local practice with imidocarb dipropionate 20 days ago under suspicion of piroplasmosis and was given per oral antibiotic therapy (amoxicillin and enrofloxacin). Her fever subsided but three days ago she completely stopped eating, and she had not defecated for two days. She was given a laxative, after which she had a small amount of a bit softer stool. In general, she was eating and drinking less for the last 10 days. She is vaccinated against rabies and fed with dehydrated dog food.

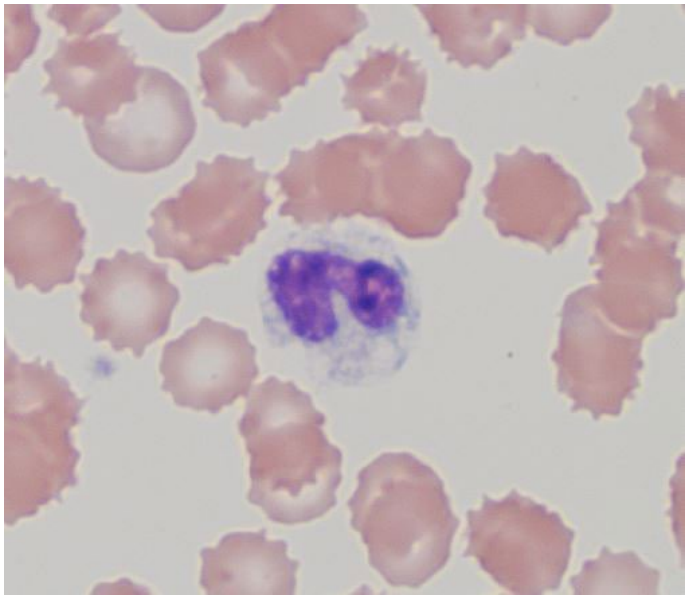
General clinical examination revealed that Nes was very calm, and her abdomen was distended and undulating. On auscultation, she was tachycardic and here lung sounds were bilaterally harsh. She was normothermic, heart rate 172/min, respiratory frequency 56/min. Based on the signalment, history, and clinical exam findings, a suspicion of ascites was made. A-FAST was performed which revealed medium amount of ascites and a complex mass of 9 × 10 cm.

Abdominocentesis was performed and 750 mL of brown fluid was extracted (total nuclear cell count > 80 000 × 10^9/L). A complete blood count was performed and reported mild anemia, leukocytosis, and thrombocytopenia. Serum biochemistry showed hypoproteinemia, hypoalbuminemia, elevated bilirubin and aspartate aminotransferase, alkaline phosphatase, blood urea nitrogen and potassium. Radiography of the thorax and abdomen suggested a diagnosis of abdominal mass and ascites. Based on the results of diagnostic procedures, suspicion of peritonitis was made and she was referred to surgery and was euthanized because tumor on small intestines was inoperable.

Parameter	Results	Unit		Reference range	
WBC	40.1	10^9/l	▲	6.0	17.0 10^9/l
LYM	3.8	10^9/l	9.5 %	0.9	5.0 10^9/l
MON	1.2	10^9/l	3.2 %	0.3	1.5 10^9/l
NEU	35.1	10^9/l	▲ 87.3 %	3.5	12.0 10^9/l
EOS	0.0	10^9/l	▼ 0.0 %	▼ 0.1	1.5 10^9/l
HGB	107	g/l	▼	120	180 g/l
MCH	23.4	pg		19.5	25.5 pg
MCHC	322	g/l		320	385 g/l
RBC	4.57	10^{12}/l	▼	5.50	8.50 10^{12}/l
MCV	72.6	fl	▲	60.0	72.0 fl
HCT	33.2	%	▼	37.0	55.0 %
RDW	13.9	%	53.1 fl	12.0	17.5 %
PLT	87	10^9/l	▼	200	500 10^9/l
MPV	9.2	fl		5.5	10.5 fl



Case C03



Microscope image

Blood smear from dog with septic peritonitis showing neutrophil with foamy vacuolation in the cytoplasm. Toxic neutrophil changes include retention of cytoplasmic features of immaturity and include foamy basophilic cytoplasm.

Parameter	Result	Indication	Reference interval	Unit
ALB	13.2	L	26.0–33.0	g/L
TP	48.7	L	55.0–75.0	g/L
GLOB	35.5		19.0–45.0	g/L
A/G	0.37			
TB	17.8	H	0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	250	H	0–82	U/L
ALT	43		0–88	U/L
ALP	419	H	20–156	U/L
AMY	897		0–1600	U/L
Crea	70.4		44.0–140.0	µmol/L
UA	28.97		0.00–60.00	µmol/L
BUN	11.97	H	3.30–8.30	mmol/L
BUN/CREA	170.031		16.000–218.000	
GLU	3.91		3.60–6.50	mmol/L
TC	6.34		3.50–7.50	mmol/L
TG	1.9	H	0.20–1.30	mmol/L
tCO2	11.8	L	12.0–27.0	mmol/L
Ca	1.64	L	2.10–3.10	mmol/L
PHOS	2.51	H	0.70–2.10	mmol/L
Mg	1.09		0.80–1.10	mmol/L
K^+	6.16	H	3.60–5.80	mmol/L
Na^+	141.4		140.0–155.0	mmol/L
Cl^-	104.4		90.0–110.0	mmol/L

Case C03

Peritonitis

Peritonitis is inflammation of the peritoneal cavity and usually associated with bacterial infection (i.e., septic), but it can also be aseptic. Septic peritonitis is most commonly bacterial, but can also be of fungal and parasitic origin. Penetrating wounds, surgical contamination, dehiscence of prior surgical sites, extension of urogenital infections, and rupture of the gastrointestinal (GI) tract are most common sources of pathogens. Aseptic or sterile peritonitis may occur with pancreatitis, biliary disease, neoplasia, feline infectious peritonitis (FIP), and pansteatitis (cats).

Presenting findings vary and may be nonspecific, such anorexia, lethargy, and abdominal pain and of vary in severity from mild clinical signs to shock. Patients can present with pale mucous membranes, dehydrated, and hypotensive. They can be hypothermic or febrile, as well as icteric. Bradycardia or tachycardia and cardiac arrhythmias may be present. In severe cases, signs of disseminated intravascular coagulation (DIC) as petechiae can be seen. Generalized lymphadenopathy may be present as well as abdominal mass or organomegaly upon abdominal palpation.

In complete blood count, marked neutrophilic leukocytosis, often with a left shift and signs of neutrophil toxicity, while leukopenia is usually a sign of a severe disease or sepsis. Anemia is also commonly present or hemoconcentration from dehydration, and thrombocytopenia secondary to consumption or DIC.

Elevation of activity of hepatic enzymes may be noted and in sepsis hyperbilirubinemia. Septic patients are also usually hypoglycemic. Dehydration may cause prerenal azotemia and hyperkalemia is commonly seen with uroperitoneum. Hypoalbuminemia is common as well as elevated C-reactive protein.

Analysis of free abdominal fluid is essential in distinguishing different types of peritonitis. Abdominocentesis can be performed under ultrasound guidance or blindly when larger amounts of effusion are present. Total nuclear cell count should be determined as well as total proteins and albumins to determine the type of effusion. In one study, nucleated cell count of peritoneal effusions that was > 13 000 cells/µL was 86% sensitive and 100% specific in dogs, and 100% sensitive and 100% specific in cats, for diagnosing septic abdomen. Determination of lactate and glucose concentration in effusion and whole blood can also be used for diagnosis of septic peritonitis. Depending on the suspected cause, concentration of creatinine, potassium, bilirubin, and lipase can also be determined. Cytology of abdominal fluid should be performed and depending on the type of effusion, culture and sensitivity may be indicated.

Blood pressure should be measured in all patients with peritonitis because systemic hypotension is frequently encountered in these patients.

Treatment is focused on identifying and treating the underline cause. Surgical exploration is indicated in all patients diagnosed with septic peritonitis as soon as the patient is stabilized. Animals presented in shock should be stabilized using balanced crystalloid solutions intravenously in shock doses (90 mL/kg), of which 25% to 50% is administered rapidly over 10 to 30 minutes, after which the patient is reassessed. If the patient is still in shock, colloids can be administered. If a patient is hypotensive regardless of fluid therapy vasopressors may be considered if adequate circulating volume has been achieved. Although culture and sensitivity testing should be used to determine best therapeutic options, broad spectrum antibiotics are initiated preoperatively to avoid delaying therapy, usually consisting of a penicillin or cephalosporin in combination with fluoroquinolone. In patients with significant hypoalbuminemia, fresh-frozen plasma should be given, especially if associated with coagulation abnormalities. Appropriate analgesia is achieved using opioids, as these patients are usually in pain. If a patient is vomiting antiemetic therapy is indicated especially because early enteral nutrition help minimize bacterial translocation from intestinal tract.

Patients with peritonitis should be closely monitored (vital signs, fluid intake and urine production, blood pressure, serum lactate).

Prognosis is variable, primarily depending upon severity and underlying cause. One should have in mind that early and aggressive therapy improves the prognosis. Increased activity of hepatic enzymes, DIC, persistent hypotension, cardiovascular collapse, and multiple organ dysfunction are considered negative prognostic factors.

Infections

Case C04

Feline leukemia virus (FeLV)

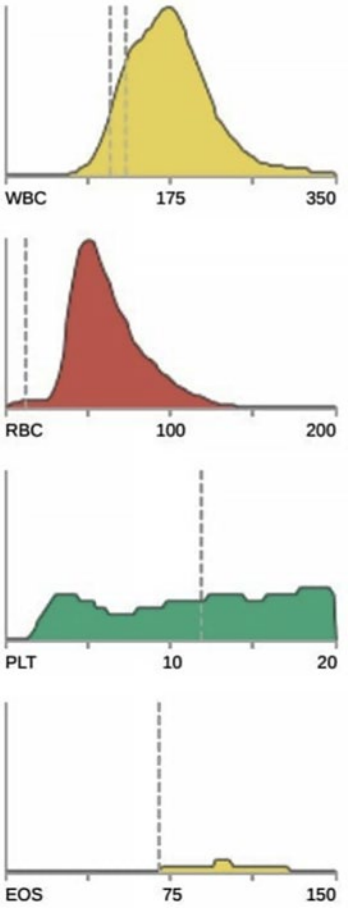
A 1-year-old male domestic breed cat was presented to the clinic because of respiratory dyspnea for two weeks. He was treated with antibiotics, corticosteroids, and diuretics. A clinical examination was done. Body condition score 3/9 (too thin). Mentation was normal. He was dyspneic, respiratory rate was 78/min, temperature 37.5°C, heart rate 180/min. Mucous membranes were pink, dry, slightly tacky, CRT was 2.5 s, eyes were sunken in sockets. Oral cavity was normal. Lymph nodes were normal for the size of the animal. Cardiac auscultation: heart sounds were muffled, there was no heart murmur. Sounds were dull all over the chest on the auscultation of the thorax. Abdomen was tender, without organomegaly.

As the first differential diagnosis was liquidotorax, thoracic fast ultrasound (T-fast) was done, and liquid was observed. After the T-fast, the cat was sedated with buprenorfine 0.02 mg/kg and midazolam 0.2 mg/kg for induction and propofol for maintenance for thoracocentesis. Thoracocentesis was done in 7th intercostal space in the ventral one-third of the thorax. It was obtained 45 mL of white, creamy effusion. Effusion was sent for cytology and total nuclear cell count (TNCC), proteins, albumins, triglyceride, and cholesterol measurement. Fluid analysis indicates chylotorax. Chylothorax is the accumulation of modified lymphatic fluid (chyle) within the pleural space. Cytology of the effusion showed exudate (chylotorax) with a lot of lymphocytes and macrophages.

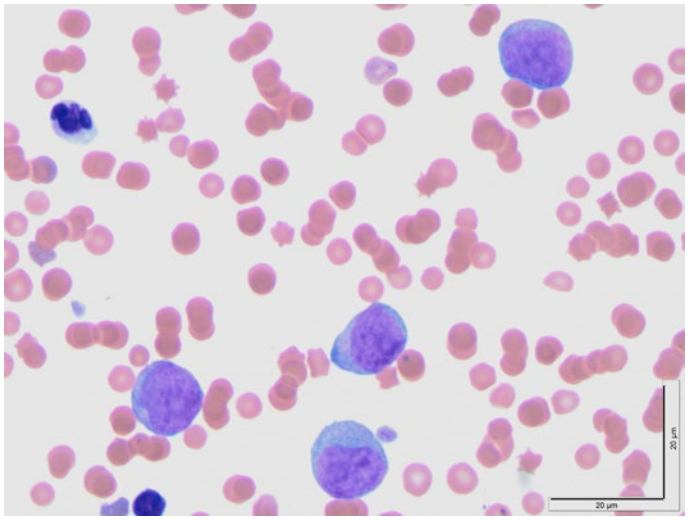
Blood samples were taken, and complete blood count and biochemistry profile were done. The complete blood count showed lymphopenia and thrombocytopenia. From the EDTA blood the feline leukemia (FeLV) and feline immunodeficiency virus (FIV) test were done to detect exposure to or infection of one of these viruses.

After the thoracocentesis, X -ray of the thorax and the abdomen was done. X-ray of the thorax showed the tumor in the mediastinum.

Parameter	Results	Unit		Reference range		
WBC	10.9	10^9/l		5.5	19.5	10^9/l
LYM	0.5	10^9/l	▼ 5.0 %	1.0	7.0	10^9/l
MON	1.1	10^9/l	▲ 11.0 %	0.2	1.0	10^9/l
NEU	8.7	10^9/l	79.9 %	2.8	13.0	10^9/l
EOS	0.4	10^9/l	4.1 %	0.1	1.0	10^9/l
HGB	146	g/l		80	150	g/l
MCH	17.6	pg	▲	12.5	17.5	pg
MCHC	278	g/l	▼	310	385	g/l
RBC	8.33	10^{12}/l		5.00	11.00	10^{12}/l
MCV	63.2	fl	▲	39.0	50.0	fl
HCT	52.7	%	▲	25.0	45.0	%
RDW	20.5	%	▲ 61.6 fl	▲ 14.0	18.5	%
PLT	83	10^9/l	▼	200	500	10^9/l
MPV	6.9	fl	▼	8.0	12.0	fl



Case C04



Microscope image

Blood smear from cat with FeLV infection showing neoplastic WBCs.

Parameter	Result	Indication	Reference interval	Unit
ALB	26.6		21.0–33.0	g/L
TP	68.8		54.0–78.0	g/L
GLOB	42.3		23.0–52.0	g/L
A/G	0.63			
TB	5		0.0–8.6	µmol/L
GGT	15	H	0–12	U/L
AST	58		0–80	U/L
ALT	<5		0–88	U/L
ALP	277	H	0–59	U/L
AMY	602		0–900	U/L
Crea	114.2		0.0–141.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	8.46		3.30–10.70	mmol/L
BUN/CREA	74.095		27.000–182.000	
GLU	12.83	H	3.10–5.60	mmol/L
TC	6.38	H	1.80–3.90	mmol/L
TG	> 10.00	H	0.10–1.30	mmol/L
tCO2	19.5		13.0–25.0	mmol/L
Ca	2.26		2.00–3.00	mmol/L
PHOS	3.7	H	0.80–1.90	mmol/L
Mg	1.81	H	0.60–1.30	mmol/L
K^+	4.32		3.60–5.50	mmol/L
Na^+	153.2		145.0–157.0	mmol/L
Cl^-	109.6	L	110.0–130.0	mmol/L

Fluid analysis	
Total nuclear cell count (TNCC)	8.0
Cholesterol	2.7
Triglycerides	18.8

Case C04

Feline leukaemia virus (FeLV)

FeLV is a gammaretrovirus belonging to the oncornavirus subfamily within the family Retroviridae. FeLV is one of the most common infectious diseases of cats worldwide. Transmission of FeLV primarily results from close contact with salivary secretions, such as through mutual grooming, licking, and shared food and water dishes. Viremic cats may also shed the infections virus in multiple body fluids, including nasal secretions, feces, milk, and urine. Evidence of horizontal transmission of FeLV by cat fleas also exist.

In the research that was performed 2018 on 324 domestic cats presented to the Department of Microbiology and Infectious Disease with Clinic at the Faculty of Veterinary Medicine, University of Zagreb, seropositive seroprevalence for FeLV was 14.50% (n = 47).

FeLV infection can lead to several types of syndromes. The most common ones are proliferative (neoplasia), degenerative (hematologic, blood cell line depletion) and immunosuppressive disease. FeLV is considered an oncogene that can cause a variety of tumors in cats. The most common neoplasms associated with FeLV are lymphoma (mediastinal, ocular, multicentric) and leukemia. FeLV infection is associated with a variety of non-neoplastic, hematologic disease from bone marrow suppression by the virus, anemia, neutropenia, pancytopenia, and thrombocytopenia.

In-house test kits using enzyme-linked immunosorbent assay (ELISA) are commonly used to screen for FeLV infections.

Clinical signs of FeLV infection vary. Signs may reflect concurrent diseases or secondary infections. Signs are often nonspecific and include anorexia, weight loss, lethargy, and fever. Cats with concurrent GI disease may have vomiting and diarrhea. Patients that have respiratory involvement (e.g., mediastinal lymphoma, secondary respiratory infections) may exhibit dyspnea, coughing, and regurgitation. Neurologic changes may include posterior paralysis, blindness, anisocoria, Horner syndrome, hyperesthesia, abnormal vocalization, paresis, and urinary incontinence. Icterus may be noted secondary to hepatopathy or hemolytic anemia.

There are many anti-FeLV therapies. The common ones are Zidovudine, human interferon alfa, feline recombinant interferon omega (IFN-ω). Zidovudin is a nucleoside analogue that blocks the viral reverse transcriptase enzyme. It can inhibit infection of new cells, but it does not decrease viral replication in currently infected cells. Interferons have some immunomodulatory and antiviral effects and is typically used.

Infections

Case C05

Feline immunodeficiency virus (FIV)

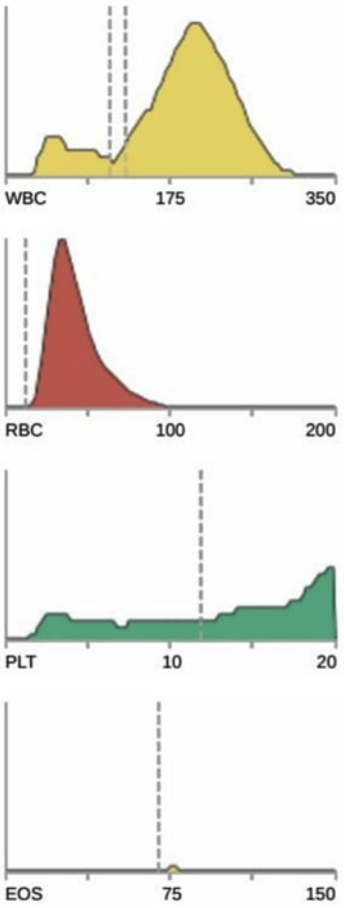
A 17-year-old male castrated domestic breed cat was presented to the clinics because of chronic vomiting. The owner said that the cat started vomiting more often lately. He is eating normally, drinking normally and his general condition is good. A clinical examination was done. General appearance was good with body condition score 3/9. Mentation was normal. Vital signs were normal (temperature 38.5°C, heart rate 180/min, respiratory rate 32/min). Mucous membranes were pink, moist, CRT was 1.5 s. Oral cavity was normal. Lymph nodes were normal for the size of the animal. Cardiac auscultation: heart sounds were rhythmic, there was no heart murmur. Respiratory rate was normal, auscultation of thorax was normal. Abdomen was tender, without organomegaly.

A complete blood count and biochemistry profile was performed. The complete blood count showed leukopenia and thrombocytopenia. Urea and creatinine were elevated in biochemistry profile. From EDTA blood, the feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV) tests were done, and the test was positive for FIV. This test is used to detect exposure to or infection of one of these viruses.

From the serum tube, T4 (thyroxine) was done to exclude hyperthyroidism, which is diagnosed in 1.5% to 11.4% of older cats worldwide.

After the blood work, X-ray of the thorax and the abdomen was made, and it was without remark. As the cat was 17 years, the owner did not want to start with therapy with interferon because the therapy is very expensive.

Parameter	Results	Unit	Reference range
WBC	4.3 10^9/l	▼	5.5 – 19.5 10^9/l
LYM	0.6 10^9/l	▼ 14.0 %	1.0 – 7.0 10^9/l
MON	0.1 10^9/l	▼ 2.7 %	0.2 – 1.0 10^9/l
NEU	3.5 10^9/l	79.8 %	2.8 – 13.0 10^9/l
EOS	0.1 10^9/l	3.5 %	0.1 – 1.0 10^9/l
HGB	109 g/l		80 – 150 g/l
MCH	13.6 pg		12.5 – 17.5 pg
MCHC	313 g/l		310 – 385 g/l
RBC	8.00 10^{12}/l		5.00 – 11.00 10^{12}/l
MCV	43.4 fl		39.0 – 50.0 fl
HCT	34.7 %		25.0 – 45.0 %
RDW	19.3 %	▲ 35.7 fl	14.0 – 18.5 %
PLT	49 10^9/l	▼	200 – 500 10^9/l
MPV	6.7 fl	▼	8.0 – 12.0 fl



Case C05

Parameter	Result	Indication	Reference interval	Unit
ALB	34.7	H	21.0–33.0	g/L
TP	75.8		54.0–78.0	g/L
GLOB	41.1		23.0–52.0	g/L
A/G	0.84			
TB	0.7		0.0–8.6	µmol/L
GGT	<0	L	0–12	U/L
AST	28		0–80	U/L
ALT	61		0–88	U/L
ALP	44		0–59	U/L
AMY	1896	H	0–900	U/L
Crea	141.6	H	0.0–141.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	14.46	H	3.30–10.70	mmol/L
BUN/CREA	102.125		27.000–182.000	
GLU	5.26		3.10–5.60	mmol/L
TC	7.18	H	1.80–3.90	mmol/L
TG	0.58		0.10–1.30	mmol/L
tCO2	11.4	L	13.0–25.0	mmol/L
Ca	2.25		2.00–3.00	mmol/L
PHOS	1.68		0.80–1.90	mmol/L
Mg	0.94		0.60–1.30	mmol/L
K^+	4.8		3.60–5.50	mmol/L
Na^+	154.5		145.0–157.0	mmol/L
Cl^-	118		110.0–130.0	mmol/L

Feline immunodeficiency virus (FIV)

FIV is a pathogenic lentivirus within the Retroviridae family. Infections in domestic cats cause immunosuppression via virus mediated CD4+ T cell depletion. FIV infection is common and widespread throughout the world but seroprevalence varies by geography and population sampled. The major mode of FIV transmission is through biting wounds and to a lesser extent transplacental, through colostrum, blood transfusion, and possibly venereal routes. Although infected cats may experience a prolonged period with no clinical abnormalities, a variety of clinical and disease conditions are associated with retroviral infections, including anaemia, haematological disorders, lymphoma, and chronic inflammatory conditions, as well as susceptibility to secondary and opportunistic infections. In the research that was performed 2018 on 324 domestic cats presented to the Department of Microbiology and Infectious Disease with Clinic at the Faculty of Veterinary Medicine, University of Zagreb, seropositive seroprevalence for FIV was 18.51% (n = 60).

Clinical signs vary widely. Possible signs include fever, lethargy, lymphadenopathy, halitosis, hypersalivation, gingivostomatitis, weight loss, subcutaneous abscesses, and anorexia. There are four stages of the disease: acute phase, latent infection, acquired immunodeficiency and terminal phase. Acute phase occurs acutely after the infection. Findings usually include depression, fever, lymphadenopathy. Latent infection is asymptomatic stage. Cats may remain in the latent stage for months to years. Acquired immunodeficiency is progression of the clinical signs. This phase has higher potential for secondary infections and immune-mediated diseases. Terminal phase can involve neurological disease, neoplasia, and severe opportunistic infection. Survival time is limited to 2 to 3 months.

In-clinic (point-of-care) test kits for FIV infection are widely available. Most are based on enzyme-linked immunosorbent assay (ELISA).

There are several anti-FIV therapy protocols. The common ones are Zidovudine, Fozivudine tidoxil, and Interferon. Zidovudin is a nucleoside analogue that blocks the viral reverse transcriptase enzyme. It can inhibit infection of new cells, but it does not decrease viral replication in currently infected cells. Fozivudine tidoxil is also a nucleoside analogue. Interferon has some immunomodulatory and antiviral effects and is typically used.

Inflammation

Case D01

Meningitis of unknown origin in dog

A 1-year-old, male, intact Pomeranian spitz presented to the emergency service due to an acute onset of vomiting, inappetence and gait abnormalities. He was reported to have vomited around 30 times in the last three days (yellow foam) and was refusing food and water. Fluid therapy, antiemetics and antibiotics were administered by the referring veterinarian. After two days of therapy, he started developing lethargy and gait abnormalities, showing ataxia and paresis with frequent falling. Teddy was current on his vaccines and data on ecto/endoparasite prophylaxis was not available.

General physical examination revealed that Teddy was non ambulatory and markedly obtunded, and that he was a unilateral cryptorchid. His pulse rate was 70/min, and the rest of the examination revealed no further abnormalities.

Neurologic examination showed marked obtundation, an ambulatory tetraparesis with vestibular ataxia of all four limbs and right sided pleurototonus, paw placement deficits of all four limbs with normal segmental spinal reflexes. Cranial nerve examination revealed no abnormalities asides from bilaterally reduced nasal sensation. Mild cervical hyperesthesia was also noted. A neuroanatomic diagnosis of multifocal brain disease (involving the forebrain and brainstem) was made. Differential diagnoses included neoplastic disease (primary neoplasia vs metastatic disease) and inflammatory disease (immune-mediated vs infectious). Complete blood count and serum biochemistry were within normal limits.

A presumptive diagnosis of an inflammatory or (less likely) neoplastic encephalopathy was made. Suggested further workup included advanced diagnostic imaging of the brain and bicavitary diagnostic imaging to exclude disseminated inflammatory or neoplastic disease.

Magnetic resonance imaging (MRI) of the brain revealed a focal, intra-axial, millimetric, elongated FLAIR-T2 hyperintensity seen along the right dorsolateral aspect of the brain stem. Differential diagnoses included an inflammatory and neoplastic brain disease or ischemic infarct.

Cerebrospinal fluid analysis (cerebellomedullary cistern) revealed a moderate pleocytosis (19 cells/µL, reference range: 0–5 cells/µL) with normal protein concentration (0.16 g/L, reference range: < 25 g/L).

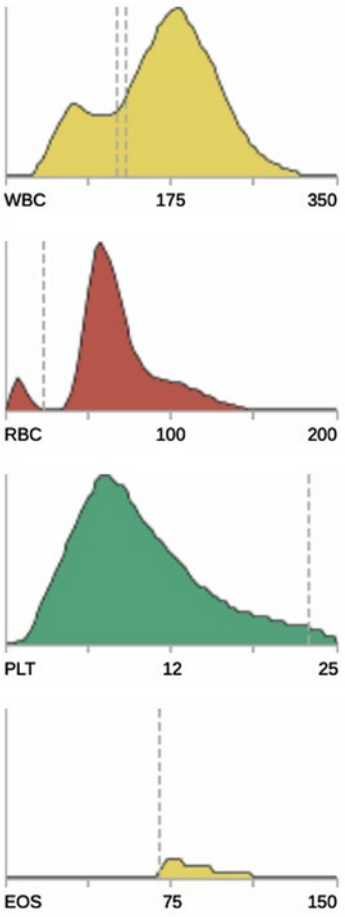
Regional infectious disease testing (polymerase chain reaction [PCR] of cerebrospinal fluid [CSF] for Toxoplasma gondii, Neospora caninum and Canine Distemper Virus) was negative.

A diagnosis of meningoencephalomyelitis of unknown origin (MUO) was made and Teddy was started on immunosuppressive doses of corticosteroids and antibiotics (prior to negative results of the infectious disease testing).

One day after starting the therapy, Teddy showed marked improvement. His level of consciousness and behavior improved, he was able to ambulate better, started eating and drinking on his own. After discharge, Teddy was lost to follow up.

Case D01

Parameter	Results	Unit		Reference range			
WBC	11.9	10⁹/l		6.0		17.0	10⁹/l
LYM	2.5	10⁹/l	21.2 %	0.9		5.0	10⁹/l
MON	0.5	10⁹/l	4.6 %	0.3		1.5	10⁹/l
NEU	8.6	10⁹/l	71.6 %	3.5		12.0	10⁹/l
EOS	0.3	10⁹/l	2.6 %	0.1		1.5	10⁹/l
HGB	146	g/l		120		180	g/l
MCH	22.5	pg		19.5		25.5	pg
MCHC	316	g/l	▼	320		385	g/l
RBC	6.46	10¹²/l		5.50		8.50	10¹²/l
MCV	71.4	fl		60.0		72.0	fl
HCT	46.1	%		37.0		55.0	%
RDW	13.3	%	50.7 fl	12.0		17.5	%
PLT	389	10⁹/l		200		500	10⁹/l
MPV	10.0	fl		5.5		10.5	fl



Parameter	Result	Indication	Reference interval	Unit
ALB	30.6		26.0–33.0	g/L
TP	58.5		55.0–75.0	g/L
GLOB	27.9		19.0–45.0	g/L
A/G	1.1			
TB	<0.0	L	0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	38		0–82	U/L
ALT	55		0–88	U/L
ALP	84		20–156	U/L
AMY	234		0–1600	U/L
Crea	52.6		44.0–140.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	5.53		3.30–8.30	mmol/L
BUN/CREA	105.158		16.000–218.000	
GLU	4.49		3.60–6.50	mmol/L
TC	4.37		3.50–7.50	mmol/L
TG	1.28		0.20–1.30	mmol/L

Case D01

Meningoencephalomyelitis of unknown origin

Meningoencephalomyelitis of unknown origin (MUO) is an umbrella term for a group of clinically very similar, but pathologically distinct, autoimmune diseases of the CNS. MUO has become increasingly recognized throughout the world. Some specific types of inflammatory brain disease have been described from tissue samples, on the basis of the distribution of inflammation and the types of inflammatory cells involved, including granulomatous meningoencephalitis (GME), necrotizing meningoencephalitis (NME), and necrotizing leukoencephalitis (NLE). Final diagnosis is only possible with histopathology. MUO is used as a synonym for autoimmune meningoencephalomyelitis in dogs, but it specifically excludes steroid-responsive meningitis (a primary disease of the meninges rather than the central nervous system [CNS] itself) and eosinophilic meningoencephalomyelitis (very uncommon).

It appears most commonly in small (terrier) breed dogs, more often females, which are young to middle aged. A complex interaction of genetics, and the animal's environment, probably contributes to developing the condition. Environmental triggers may include infectious antigens and vaccination. Genetic causes have long been suspected in dogs with MUO because of the high prevalence in specific breeds, like Pugs, Yorkshire terrier, Papillon, Pekingese, Chihuahua, Shih tzu, Maltese and others. In GME, large breed dogs comprise only 25% of cases.

Clinical signs depend on lesion location within the CNS. They may include seizures, blindness, cranial nerve deficits, altered mentation, compulsive behavior, ataxia, paresis, proprioceptive deficits, vestibular and cerebellar signs, head, and/or cervical pain. Forebrain, brainstem, or multifocal lesions occur most often. GME is located in the spinal cord of 8% of affected dogs. For ocular GME, acute blindness is often the only clinical sign. Systemic signs (extra-neural) like fever are rare.

MRI and CSF analysis are commonly used to make a diagnosis of MUO. MRI is superior to computed tomography (CT) for diagnosing this disease. Definitive diagnosis requires histopathology of affected brain or spinal cord tissue. Histopathology is most often performed postmortem.

Immunosuppressive therapy is the main treatment, with glucocorticoids being the mainstay of therapy but multi-agent therapy is often recommended to increase efficacy and minimize glucocorticoid-related side-effects. Immunosuppressive drugs like cyclosporine, cytarabine, lomustine, cyclophosphamide, and mycophenolate mofetil (amongst others) are commonly used in combination with glucocorticoids.

The prognosis for MUO is generally considered fair with adequate treatment but can be highly variable and often very difficult to predict at the time of diagnosis. Typically, a good initial response to treatment indicates a good prognosis. It is possible for animals to enter a partial or (rarely) full remission from the disease and maintain a normal or very good quality of life for many months or years. Rarely, treatment can be withdrawn with time but more commonly, it will need to be continued at low doses lifelong. Regular re-checks are required to follow the patient's response to treatment. Repeated MRI and/or CSF analysis are also recommended to monitor the disease and tailor the treatment to the animal's need.

Inflammation

Case D02

Steroid-responsive meningitis and arteritis in dog

A 3-year-old, male, intact, beagle, Rex, was presented initially due to an acute onset of lethargy and inappetence. The dog started to be calmer on the previous day, the owner noticed that he is refusing food and water, and she also had a feeling Rex was in pain. Nine months prior, Rex was reported to have had an episode of meningitis, which was treated with corticosteroids for a short period of time. In the meantime, he had had seven to eight such episodes, all of which were treated with a short course of corticosteroids. He was current on his vaccines and was currently not receiving any ecto/endoparasite prophylaxis.

General physical examination revealed that Rex was very calm, had a mild kyphosis and a low head-neck carriage. Temperature, pulse, and respirations were all within normal limits. Based on the signalment, history, and clinical exam findings, a suspicion of steroid-responsive meningitis-arteritis (SRMA) was made. Neurological examination showed no abnormalities besides the low head and neck carriage due to cervical hyperesthesia.

A complete blood count was performed and revealed no clinically relevant abnormalities. Serum biochemistry was without remark, but CRP levels showed a marked increase (181.5 mg/L, reference range 0–10.7 mg/L).

Radiography of the cervical spine, thorax, and abdomen was performed to exclude changes compatible with discospondylitis and to exclude disseminated inflammatory or neoplastic disease and showed no relevant abnormalities.

The cerebrospinal fluid (CSF) was sampled (cerebellomedullary cistern) and analysis revealed a normal total nucleated cell count (TNCC) (4 cells/mm^3, reference range 0–5 cells/mm^3) and protein concentration (0.16 g/L, reference range: < 25 g/L).

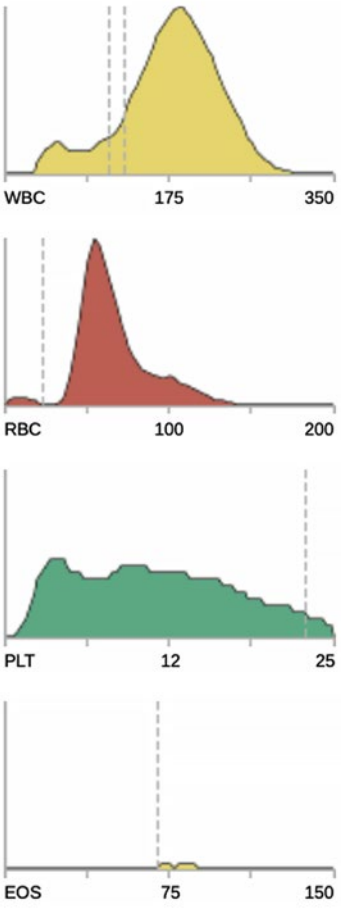
Due to normal CSF, TNCC, and protein concentration, a suspicion of chronic SRMA was made. Additional paired serum and CSF IgA concentration analysis was made (Laboklin, Bad Kissingen, Germany) and revealed a simultaneous increase of both serum (690 mg/dL, reference range: 20–120 mg/dL) and CSF IgA (0.37 mg/dL, reference range: < 0.1 mg/dL) levels.

As CSF IgA levels are increased in many dogs with SRMA and remain elevated even after resolution of clinical signs, this finding supported the diagnosis of SRMA.

Therapy with immunosuppressive doses of prednisone were started at 4 mg/kg PO once daily for 2 days, then reduced to 2 mg/kg PO once daily for 14 days, at which point a re-check was made. Rex's owners reported that he improved and was showing no pain, the clinical and neurologic examination, complete blood count, and serum CRP analysis revealed no abnormalities, due to which fact, the dose of prednisolone was further tapered to 1 mg/kg PO once daily for 8 weeks.

Case D02

Parameter	Results	Unit	Reference range
WBC	12.9 10⁹/l		6.0 – 17.0 10⁹/l
LYM	1.4 10⁹/l	11.4 %	0.9 – 5.0 10⁹/l
MON	0.6 10⁹/l	5.0 %	0.3 – 1.5 10⁹/l
NEU	10.8 10⁹/l	83.5 %	3.5 – 12.0 10⁹/l
EOS	0.0 10⁹/l	▼ 0.1 %	0.1 – 1.5 10⁹/l
HGB	166 g/l		120 – 180 g/l
MCH	22.5 pg		19.5 – 25.5 pg
MCHC	328 g/l		320 – 385 g/l
RBC	7.37 10¹²/l		5.50 – 8.50 10¹²/l
MCV	68.6 fl		60.0 – 72.0 fl
HCT	50.6 %		37.0 – 55.0 %
RDW	13.6 %	49.2 fl	12.0 – 17.5 %
PLT	213 10⁹/l		200 – 500 10⁹/l
MPV	11.1 fl	▲	5.5 – 10.5 fl



Parameter	Result	Indication	Reference interval	Unit
ALB	30		26.0–33.0	g/L
TP	64.1		55.0–75.0	g/L
GLOB	34.1		19.0–45.0	g/L
A/G	0.88			
TB	0.9		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	24		0–82	U/L
ALT	32		0–88	U/L
ALP	141		20–156	U/L
AMY	949		0–1600	U/L
Crea	63.5		44.0–140.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	5.16		3.30–8.30	mmol/L
BUN/CREA	81.263		16.000–218.000	
GLU	6.92	H	3.60–6.50	mmol/L
TC	5.51		3.50–7.50	mmol/L
TG	0.38		0.20–1.30	mmol/L
tCO2	16.9		12.0–27.0	mmol/L
Ca	2.32		2.10–3.10	mmol/L
PHOS	1.25		0.70–2.10	mmol/L
Mg	0.79	L	0.80–1.10	mmol/L
K^+	4.88		3.60–5.80	mmol/L
Na^+	144.9		140.0–155.0	mmol/L
Cl^-	106.9		90.0–110.0	mmol/L

Case D02

Steroid-Responsive Meningitis-Arteritis

Steroid responsive meningitis-arteritis (SRMA) is an immune-mediated inflammatory disorder targeting the leptomeninges and associated vessels of dogs. The disorder not been reported in cats. This disease is known under many names, such as polyarteritis, beagle pain syndrome, aseptic suppurative meningitis, and more. SRMA is the established and most widely used nomenclature today.

SRMA is most commonly seen in dogs between the ages of 6 and 18 months, although the reported age range is anywhere from 12 weeks to 7 years. While any breed can be affected, including mixed breed dogs, the most commonly affected breeds include Boxers, Beagles, Bernese Mountain Dogs, Nova Scotia duck tolling retrievers, Border collies, and Weimaraners among others.

Two forms of the disease have been described:

1. An acute fulminating form which is characterized by neutrophilic pleocytosis in cerebrospinal fluid (CSF).
2. A chronic form commonly associated with ataxia, paresis/paraplegia, and neurological deficits is characterized by mild, mononuclear, or mixed cell pleocytosis in CSF.

Complete etiopathogenesis is still unknown; but an immune-mediated response directed against the central nervous system (CNS) is considered most likely.

During the acute stage, clinical signs may be restricted to intermittent fever, lethargy, and cervical (neck) pain. As the disease becomes chronic, leptomeninges become fibrotic and thickened, and affected arterial walls may also show similar changes. Thrombosis and vascular occlusion may lead to neural ischemia that causes neurological signs. Although SRMA primarily results in meningitis, it can occur concurrently with immune-mediated polyarthritis resulting in clinical findings of joint pain and effusion.

Clinicopathologic findings may reveal an inflammatory leukogram characterized by a potentially marked neutrophilia with or without left shift, hypoalbuminemia, hyperglobulinemia and elevated serum CRP levels. Diagnosis is typically confirmed with CSF analysis, which usually shows a typical marked non-degenerate neutrophilic pleocytosis. As SMRA becomes chronic (> 7 days), diminished protein elevation and fewer white blood cells may be found. In addition, the cell population often becomes more mixed, with a predominance of mononuclear cells. CSF culture is negative in dogs with SRMA.

Differential diagnoses for SRMA include discospondylitis, other causes of meningitis such as infections, necrotizing, granulomatous, eosinophilic, other causes of polyarthritis and intervertebral disk disease.

Treatment for SRMA typically involves the use of immunosuppressive doses of corticosteroids, most commonly prednisone or prednisolone, at a starting dose of 4 mg/kg/day. Most dogs will show dramatic improvement in clinical signs within a few days. Corticosteroid treatment is then continued for a minimum of 6 months with a gradual tapering of the dosage every 6 to 8 weeks. Regular recheck examinations every 6 to 8 weeks is recommended to ensure that patients do not develop long-term steroid-adverse effects as well as to allow for identification of signs of relapse.

With early, aggressive immunosuppressive therapy, 60% to 80% of affected dogs can be cured. Relapses occur in 20% to 40% of cases, and these dogs require a longer treatment. Relapses can occur during treatment tapering as well as after cessation of therapy.

To conclude, SRMA is a relatively common immune-mediated disease of young dogs primarily presenting with marked cervical pain, lethargy, and fever. Patient signalment and clinical and clinicopathologic abnormalities are often typical, especially in the acute form of SRMA, allowing for a relatively straightforward diagnosis. Treatment with long-term immunosuppressive corticosteroid therapy shows an overall good prognosis. Early identification of clinical signs consistent with SRMA is paramount for optimal treatment efficacy and prognosis. Failure of adequate, early treatment can result in the development of the chronic form of SRMA which is more difficult to diagnose and to treat.

Erythrocyte abnormalities, anemia Case E01

Immune-Mediated Hemolytic Anemia (IMHA) in dog

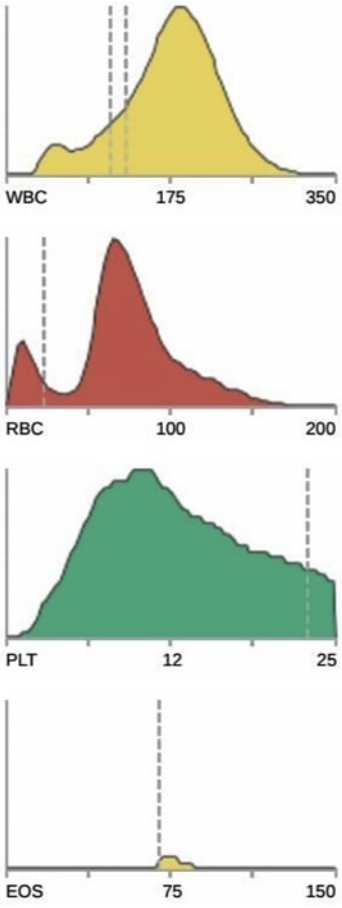
A 9-year-old mixed breed castrated bitch was presented to the clinic because of acute vomiting, hematemesis. She was lethargic and she refused food. A year ago, she had splenectomy because of the hypoechoic lesions on the spleen. Pathohystology showed nodular lymphoid hyperplasia of the spleen.

On general clinical examination, she was lethargic. Body condition score 3/9 (too thin). Mentation was normal. Vital signs were normal (temperature 38.5°C, heart rate 120/min, respiratory rate 32/min). Mucous membranes were pale, dry, slightly tacky, CRT was 2 s. Oral cavity was normal. Lymph nodes were normal for the size of the animal. Cardiac auscultation: heart sounds were rhythmic, there was no heart murmur. Auscultation of the thorax was normal. Abdomen was tender, without organomegaly. There were no petechiae on the body.

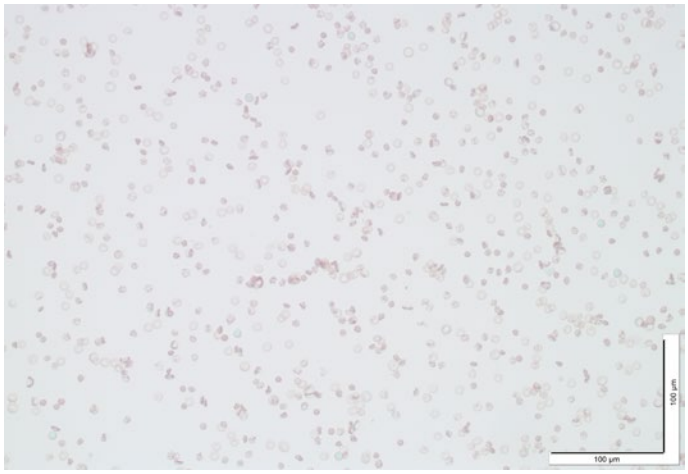
A blood samples were taken. Complete blood count showed regenerative anemia and leukocytosis. Biochemical test showed elevated urea and elevated ALT, GGT, and AP. Slide agglutination test was positive. Snap test for vector-borne diseases was negative. PCR was done for vector-borne anemia: hemotropic mycoplasma, Babesia, *Ehrilichia canis, Anaplasma phagocytophilum* was negative. Coombs test was positive. X-ray of the thorax and the abdomen was made and was without remark. Ultrasound of the abdomen was made and enlarged liver was spotted with coarse echostructure. As all the tests came back negative, biopsy of the liver and bone marrow was suggested but owners refused it.

Immunosuppressive therapy was started with prednisone 2 mg/kg PO twice a day. She responded very good to therapy. After the five days, the dose was reduced to 1 mg/kg PO twice a day. After 2 weeks, the dose was reduced to 0.5 mg/kg twice a day. The animal was doing great, she responded very well to glucocorticoid therapy. Antithrombotic therapy (clopidogrel 1.1 mg/kg PO q.24 h) was also started because of common complication thromboembolic disease.

Parameter	Results	Unit		Reference range		
WBC	33.5	10^9/l	▲	6.0	17.0	10^9/l
LYM	4.0	10^9/l	12.2 %	0.9	5.0	10^9/l
MON	2.1	10^9/l	▲ 6.3 %	0.3	1.5	10^9/l
NEU	27.3	10^9/l	▲ 81.5 %	3.5	12.0	10^9/l
EOS	0.0	10^9/l	▼ 0.0 %	▼ 0.1	1.5	10^9/l
HGB	88	g/l	▼	120	180	g/l
MCH	25.0	pg		19.5	25.5	pg
MCHC	307	g/l	▼	320	385	g/l
RBC	3.51	10^{12}/l	▼	5.50	8.50	10^{12}/l
MCV	81.3	fl	▲	60.0	72.0	fl
HCT	28.6	%	▼	37.0	55.0	%
RDW	18.4	%	▲ 71.3 fl	▲ 12.0	17.5	%
PLT	419	10^9/l		200	500	10^9/l
MPV	12.3	fl	▲	5.5	10.5	fl

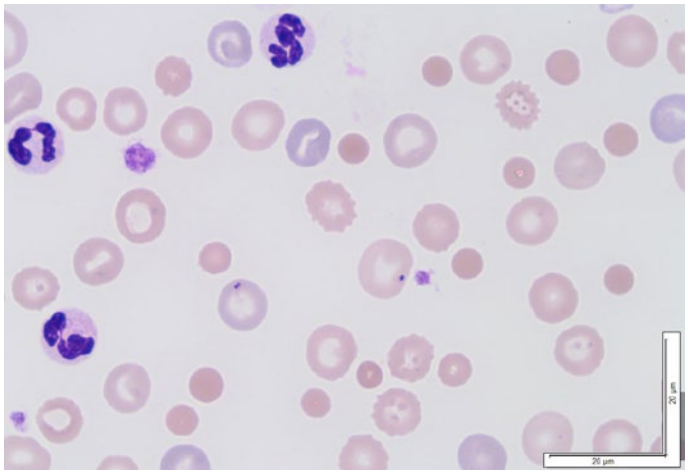


Case E01



Microscope image

Positive agglutination test.
Microagglutination observed on a standard stained blood smear from a dog with IMHA. RBCs are adhered to one another by a high level of anti-RBC antibodies.



Microscope image

Blood smear from dog showing spherocytes, that is, small RBCs with a loss of central pallor produced by incomplete destruction of RBCs by macrophages. Spherocytosis, anisocytosis and polychromasia is very suggestive of IMHA.

Parameter	Result	Indication	Reference interval	Unit
ALB	31.8		26.0–33.0	g/L
TP	57.7		55.0–75.0	g/L
GLOB	25.9		19.0–45.0	g/L
A/G	1.23			
TB	3.3		0.0–8.6	µmol/L
GGT	101	H	0–5	U/L
AST	73		0–82	U/L
ALT	> 650	H	0–88	U/L
ALP	1256	H	20–156	U/L
AMY	750		0–1600	U/L
Crea	87.1		44.0–140.0	µmol/L
UA	10.13		0.00–60.00	µmol/L
BUN	14.19	H	3.30–8.30	mmol/L
BUN/CREA	162.909		16.000–218.000	
GLU	8.02	H	3.60–6.50	mmol/L
TC	5.34		3.50–7.50	mmol/L
TG	1.44	H	0.20–1.30	mmol/L
tCO2	20		12.0–27.0	mmol/L
Ca	2.38		2.10–3.10	mmol/L
PHOS	1.68		0.70–2.10	mmol/L
Mg	0.94		0.80–1.10	mmol/L
K^+	5.55		3.60–5.80	mmol/L
Na^+	147		140.0–155.0	mmol/L
Cl^-	104.2		90.0–110.0	mmol/L

Case E01

Immune-mediated hemolytic anemia

Immune-mediated hemolytic anemia (IMHA) is characterized by an immune system dysregulation that leads to antibody and complement-mediated destruction of erythrocytes. IMHA is either primary or secondary to another disorder. Primary IMHA is idiopathic. Secondary IMHA occurs from a variety of disorders. Primary IMHA accounts for approximately 60% to 75% of cases of IMHA in dogs. Potentially, any infection can trigger immune dysregulation and development of an immune-mediated disease. Etiological primary infectious agents reported in the dog include Ehrlichia canis, hemotropic mycoplasmas, Leptospira spp., Babesia spp., *Dirofilaria immitis, Leishmania infantum, Anaplasma phagocytophilum, Ancylostoma caninum,* and *Trichuris vulpis*. Various neoplasms are reported as causes of secondary IMHA, including lymphoma, leukemia, mast cell tumor, soft tissue sarcoma, multiple myeloma, bronchoalveolar carcinoma, and splenic hemangioma. Administration of certain drugs can lead to secondary IMHA (carprofen, phenylbutazone, chlorpromazine, cephalosporins, griseofulvin, methimazole, penicillins, trimethoprim/sulphonamide, etc.).

Physical examination findings include patients with lethargy, depression, weakness. Vomiting, diarrhea, melena, hematemesis, and hematochezia may also be present. Mucous membranes are usually pallor, abdominal pain can be present, tachypnoea, dyspnea, epistaxis, petechiae, hepatomegaly, splenomegaly.

Complete blood count includes regenerative or nonregenerative anemia, leukocytosis, thrombocytopenia secondary to immune-mediated platelet destruction (Evan syndrome). In biochemistry profile, hyperbilirubinemia is the most common biochemistry abnormality. Other biochemistry abnormalities can include elevated AST, ALT, AP, hemoglobinemia, hyperglobulinemia, azotemia. Saline agglutination test evaluates autoagglutination of erythrocytes caused by antibodies. Positive test (autoagglutination) is indicative of an immune process. Direct Coombs test is used to detect antibodies and complement on the surface of erythrocytes. Cytology of blood smears can include anisocytosis, poikilocytosis, polychromasia, spherocytosis.

Treatment of IMHA include immunosuppression. Glucocorticoids are the first to use with IMHA. Usually, prednisone or prednisolone are used at a dose of 2–4 mg/kg/day. If the animals do not respond well to glucocorticoids, other immunosuppression agents can be used. Mycophenolate mofetil at the dose of 10 mg/kg PO q.12 h, azathioprine in dose 2 mg/kg PO q.24 h and cyclosporine 5-10 mg/kg PO q.12–24 h. As thromboembolic disease is the common complication of IMHA, antithrombotic therapy is also recommended (clopidogrel, aspirin, heparin). Prognosis is variable and depends on the primary cause of the IMHA.

Erythrocyte abnormalities, anemia Case E02

Erythrocytosis (Polycythemia) in dog

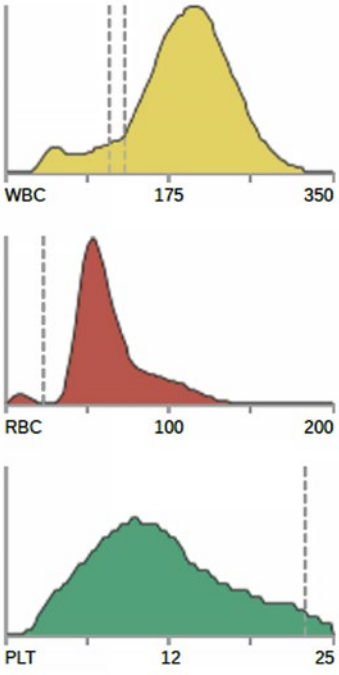
A 10-year-old, male, intact pug, Milo, was presented to the clinic due to a chronic diarrhea, vomiting, inappetence, and weight loss. Fluid therapy, antiemetics, antibiotics, and corticosteroids were administered by the referring veterinarian. Milo was currently on his vaccines and ecto/endoparasite prophylaxis. General physical examination revealed that Milo was having insufficient body score condition (2/9). The rest of general examination was without remark, except very loud inspiratory breathing sounds, due to brachycephalic syndrome. His temperature, pulse rate and breathing rate were in reference range.

Complete blood count showed erythrocytosis and serum biochemistry revealed mildly decreased concentration of total protein and albumin and mildly elevated activity of liver enzymes. Further diagnostic procedures were done (urin analysis with UPCR, abdominal ultrasound, fecal analysis). A presumptive diagnosis of a gastrointestinal protein loss– inflammatory or (less likely) neoplastic (e.g., Intestinal lymphoma)—was made. Suggested further workup included gastrointestinal endoscopy with biopsy, which confirmed chronic enteropathy. Elevated activity of liver enzymes was assumed to be a consequence of corticosteroid application and was corrected spontaneously after corticosteroids were discontinued.

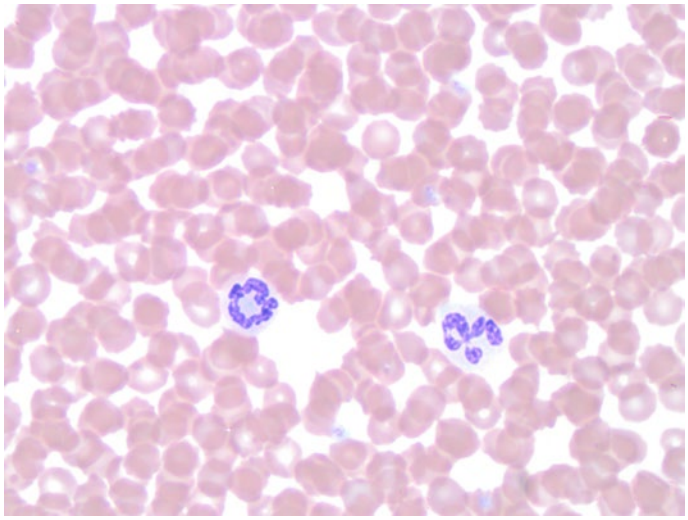
A thoracic radiographic study included two projections, left lateral (LL) image and a ventrodorsal (VD) image, and showed no abnormalities. An abdominal radiographic study included left lateral (LL) image and a ventrodorsal (VD) image, which showed no abnormalities.

Erythrocytosis was assumed to be a consequence of chronic respiratory disease: brachycephalic syndrome. Milo was treated for gastrointestinal disease and responded good to diet change.

Parameter	Results	Unit		Reference range		
WBC	11.1	10^9/l		6.0	17.0	10^9/l
LYM	1.0	10^9/l	9.3 %	0.9	5.0	10^9/l
MON	0.3	10^9/l	3.5 %	0.3	1.5	10^9/l
GRA	9.8	10^9/l	87.2 %	3.5	12.0	10^9/l
HGB	219	g/l	▲	120	180	g/l
MCH	23.2	pg		19.5	25.5	pg
MCHC	350	g/l		320	385	g/l
RBC	9.43	10^{12}/l	▲	5.50	8.50	10^{12}/l
MCV	66.2	fl		60.0	72.0	fl
HCT	62.5	%	▲	37.0	55.0	%
RDW	14.1	%	52.9 fl	12.0	17.5	%
PLT	253	10^9/l		200	500	10^9/l
MPV	11.5	fl	▲	5.5	10.5	fl



Case E02



Microscope image

Blood smear from dog showing erythrocytosis.

Parameter	Result	Indication	Reference interval	Unit
ALB	23	L	26.0–33.0	g/L
TP	54.5	L	55.0–75.0	g/L
GLOB	31.5		19.0–45.0	g/L
A/G	0.73			
TB	3.2		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	265	H	0–82	U/L
ALT	98	H	0–88	U/L
ALP	657	H	20–156	U/L
AMY	945		0–1600	U/L
Crea	84.2		44.0–140.0	µmol/L
UA	12.82		0.00–60.00	µmol/L
BUN	4.54		3.30–8.30	mmol/L
BUN/CREA	53.869		16.000–218.000	
GLU	7.16	H	3.60–6.50	mmol/L
TC	7.49		3.50–7.50	mmol/L
TG	1.09		0.20–1.30	mmol/L
tCO2	16.8		12.0–27.0	mmol/L
Ca	2.21		2.10–3.10	mmol/L
PHOS	1.26		0.70–2.10	mmol/L
Mg	1.1		0.80–1.10	mmol/L
K^+	4.74		3.60–5.80	mmol/L
Na^+	147.9		140.0–155.0	mmol/L
Cl^-	108.7		90.0–110.0	mmol/L

Erythrocytosis

Erythrocytosis, also sometimes called polycythemia, is defined as an increase in RBC mass and an increased HCT and hemoglobin concentration. An increased HCT or RBC count can be relative or absolute.

Relative erythrocytosis is proportional change of RBC count in relation to plasma. Absolute erythrocytosis is a true increase in RBC count due to excessive erythropoiesis. These different types of erythrocytosis can be distinguished by several clinical and laboratory tests. Usually, clinical signs and signalment like dehydration, response to fluid therapy, and high total protein concentration suggest relative erythrocytosis and should correct with appropriate fluid treatment.

Polycythemia vera is a primary absolute erythrocytosis (chronic myeloproliferative disorder) and is a neoplastic condition in which RBC production is independent of erythropoietin concentrations.

Case E02

Secondary absolute erythrocytosis is appropriate if there is a physiologic stimulus for erythropoietin production (hypoxia), or inappropriate (independent of hypoxia, e.g., erythropoietin-producing neoplasms).

The clinical signs of relative erythrocytosis are typically those of dehydration (and the underlying disease that caused the dehydration. Clinical signs of absolute erythrocytosis are generally not observed until a HCT exceeds 60% and can include red mucous membranes, bleeding tendencies and neurologic signs, like ataxia, weakness, seizures, and blindness.

Besides beforementioned, other diagnostic procedures can be done to find a possible cause of erythrocytosis. Arterial blood gas analysis can suggest hypoxia; cardiovascular and pulmonary evaluation can be useful in detection of increased RBC production, which would result in a true increase in RBC mass or erythrocytosis. Liver and renal disease, including neoplasia also can result in a true increase in RBC mass. Measurement of erythropoietin concentrations can be performed. Bone marrow aspiration is usually not worthwhile as the marrow shows an erythroid hyperplasia in all causes of erythrocytosis.

Treatment for relative erythrocytosis due to dehydration includes rehydration with IV fluids and treating the underlying cause.

For primary erythrocytosis (polycythemia vera), initial treatment consists of periodic phlebotomy with simultaneous fluid replacement with or without extra-label administration of hydroxyurea. For appropriate secondary erythrocytosis, the underlying problem should be treated. For inappropriate secondary erythrocytosis, erythropoietin secreting tumors should be managed with surgery, chemotherapy, or radiation therapy.

Erythrocyte abnormalities, anemia Case E03

Nonregenerative anemia in cat

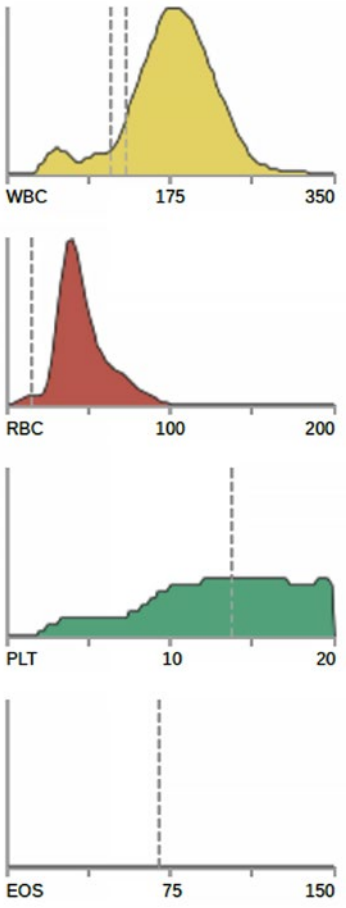
A 13-year-old, spayed female, domestic shorthair cat, Mucica, was initially presented due to a chronic onset of polyuria and polydipsia, lethargy, and inappetence. The cat started to be calmer, the owner noticed that she was vomiting once a day.

General physical examination revealed that Mucica was very calm, body condition score 2/9, and she had pale mucous membranes. Temperature was low (37.3°C) and pulse, and respirations were all within normal limits.

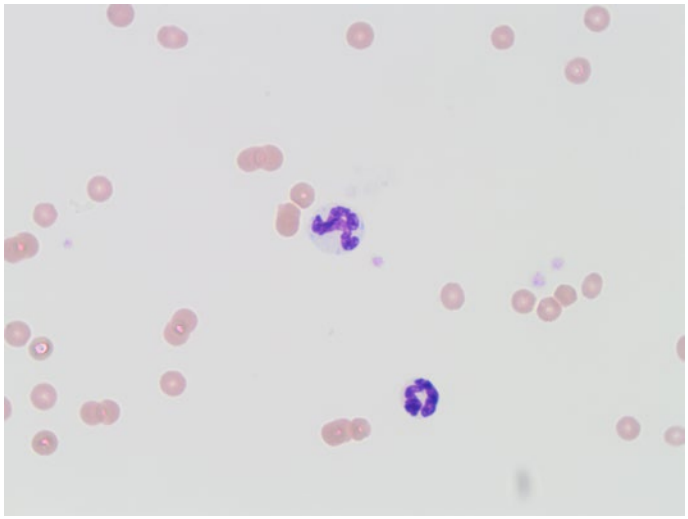
A complete blood count was performed and revealed anemia. Reticulocytes were counted manually on blood smears and showed that anemia was nonregenerative. Serum biochemistry revealed azotemia. Urine analysis showed isosthenuria and proteinuria with a urine protein creatinine ratio (UPCR) of 0.8. Systemic blood pressure was 180 mmHg.

Based on the signalment, history, and clinical exam findings, a suspicion of chronic renal disease (CKD) was made. Based on recommendations of International Renal Interest Society (IRIS), CKD was staged as IRIS 4, proteinuric and hypertensive. Radiography of the thorax and abdomen was performed to exclude other changes and showed no relevant abnormalities. The cat was treated by IRIS Treatment Recommendations.

Parameter	Results	Unit		Reference range		
WBC	8.2	10^9/l		5.5	19.5	10^9/l
LYM	0.7	10^9/l	▼ 9.3 %	1.0	7.0	10^9/l
MON	0.3	10^9/l	4.4 %	0.2	1.0	10^9/l
NEU	7.0	10^9/l	84.7 %	2.8	13.0	10^9/l
EOS	0.1	10^9/l	1.6 %	0.1	1.0	10^9/l
HGB	75	g/l	▼	80	150	g/l
MCH	15.4	pg		12.5	17.5	pg
MCHC	324	g/l		310	385	g/l
RBC	4.91	10^{12}/l	▼	5.00	11.00	10^{12}/l
MCV	47.6	fl		39.0	50.0	fl
HCT	23.4	%	▼	25.0	45.0	%
RDW	17.4	%	35.7 fl	14.0	18.5	%
PLT	88	10^9/l	▼	200	500	10^9/l
MPV	9.6	fl		8.0	12.0	fl



Case E03



Microscope image

Blood smear from cat with nongenerative anemia, showing few RBCs.

Parameter	Result	Indication	Reference interval	Unit
ALB	34.9	H	21.0–33.0	g/L
TP	81.9	H	54.0–78.0	g/L
GLOB	47		23.0–52.0	g/L
A/G	0.74			
TB	<0.1		0.0–8.6	µmol/L
GGT	<0	L	0–12	U/L
AST	18		0–80	U/L
ALT	51		0–88	U/L
ALP	25		0–59	U/L
AMY	2135	H	0–900	U/L
Crea	783.8	H	0.0–141.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	40.13	H	3.30–10.70	mmol/L
BUN/CREA	51.198		27.000–182.000	
GLU	7.51	H	3.10–5.60	mmol/L
TC	6.71	H	1.80–3.90	mmol/L
TG	0.73		0.10–1.30	mmol/L
tCO2	<5.0	L	13.0–25.0	mmol/L
Ca	2.34		2.00–3.00	mmol/L
PHOS	5.17	H	0.80–1.90	mmol/L
Mg	1.15		0.60–1.30	mmol/L
K^+	5.65	H	3.60–5.50	mmol/L
Na^+	150.6		145.0–157.0	mmol/L
Cl^-	116.9		110.0–130.0	mmol/L

Nonregenerative anemia

Anemia is defined as a decreased HCT or HGB level. HCT is a calculated value from the MCV and RBC count and HGB concentration and can be used as a surrogate for HCT. Young animals frequently have a physiologic anemia because of rapid growth rate with hemodilution from plasma volume expansion, dilutional from ingested colostrum, destruction of fetal RBC and a low erythropoietin concentration in the first months of life.

Anemia is characterized by severity, RBC indices, and regenerative response. Severity is determined by the degree of decrease in the HCT and will depend on the lower limit of the reference interval. RBC indices include MCV and MCHC. Anemia can be characterized as macrocytic, normocytic, or microcytic, and hyperchromic, normochromic or hypochromic. Anemia can be characterized as regenerative or nonregenerative.

Case E03

The diagnosis and treatment of nonregenerative anemia are usually more challenging than the regenerative ones. The most reliable non-invasive method to access bone marrow erythroid production is reticulocyte count. Reticulocytes lower than 60 000/µL or 1% for dogs and lower than 15 000/µL or 0.4% of aggregate reticulocytes for cats are expected in nonregenerative anemias. However, interpreting reticulocyte counts is more complex than just assessing the absolute number of reticulocytes. Erythrocytes have a long lifespan, so sometimes it may take months to diagnosis of anemia. Some diseases do not stop erythrocyte production completely but can cause a shorter erythrocyte lifespan, which can lead to a development of anemia. A normal bone marrow needs up to five days to respond and produce reticulocytes. This period can be seen in acute blood loss or in acute hemolytic anemia so regeneration should be assessed with caution because acute hemorrhagic or hemolytic anemia may initially appear nonregenerative. The major causes of a true nonregenerative anemia are decreased erythrocyte production or, less commonly, a defective erythropoiesis, which can be caused by many diseases and conditions.

Decreased erythrocyte production can be caused by inflammatory disease, chronic renal disease, bone marrow hypoplasia or aplasia caused by infections, drugs, toxins, irradiation, myelophthisis, myelofibrosis or osteopetrosis; selective erythroid hypoplasia or aplasia (PRCA, FeLV, endocrine, liver disease). Defective erythropoiesis can commonly be caused by nutritional anemia (iron, copper, vitamin B12, folate, or cobalamin), FeLV, immune-mediated destruction of erythroid progenitors, or pure red cell aplasia (PRCA).

Anemia in patients with chronic renal disease may be mild to severe and is mainly caused by decreased erythropoietin production. Erythropoiesis can be decreased due to an absolute or a relative lack of erythropoietin, or secondary to a decreased bone marrow response to erythropoietin. Erythropoietin is a glycoprotein hormone produced by the kidney in the peritubular interstitial cells of the inner renal cortex and outer medulla. Renal hypoxia is the main stimulating factor for erythropoietin synthesis. Erythropoietin production is decreased by both acute and chronic causes of kidney disease, and chronic kidney disease is known to be a common cause of nonregenerative anemia in the cat. Also, several other factors considered to have a role in anemia are found in kidney diseases, inflammatory cytokines can impair bone marrow responsiveness to erythropoietin, uremic toxins can reduce RBC lifespan, animals with uremia frequently suffer from oral and gastrointestinal ulcers, which can cause chronic hemorrhage and lack of energy, and mineral deficiencies will also inhibit erythropoiesis.

Cats have two types of reticulocytes: punctate and aggregate. Aggregate reticulocytes are released by the bone marrow and then mature into punctate reticulocytes. Aggregate reticulocytes are those considered to represent the regenerative response. Feline reticulocytes can be counted using manual techniques on blood smear slides or automated techniques. Reticulocyte counts should be performed as soon as possible after blood sample collection to prevent reticulocytes maturing *ex vivo*.

In cats, erythropoietin stimulating agents are commonly used to treat nonregenerative anemia secondary to chronic kidney disease with good response.

Erythrocyte abnormalities, anemia Case E04

Erythrocytosis (polycythemia) in ferret

A 6-year-old, surgical neutered female ferret was initially presented due to an onset of respiratory distress. The ferret was adopted 5 years ago. For the last 3 months, the owner has noticed that she started to be disproportionately obese with distended abdomen. A week ago, a difficulty with the breathing started and she was now no longer active, with difficulty in moving. Her appetite had increased a lot, she drank water normally, urinated normally, the stool was soft to the point of diarrhea, brown in color. She was fed with raw meat (poultry). The owner periodically measures blood glucose, which was normal. She was regularly vaccinated against infectious diseases. Suspected diagnosis: obesity, neoplastic, endocrine, or cardiorespiratory disease.

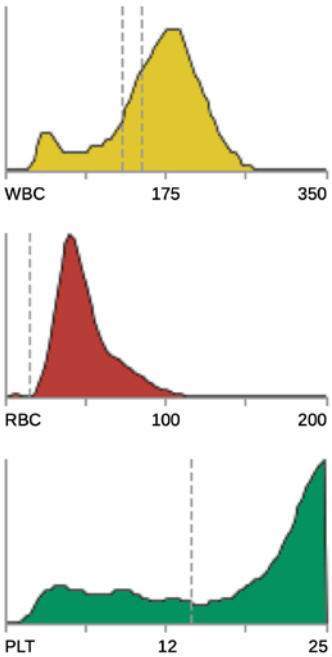
Observed changes at the clinical examination included pink to cyanotic mucous membranes, body condition score 9/9, thorax auscultation was sharper in inspiration. During the inspection, abdomen was distended. A large amount of subcutaneous fat tissue was found. The skin around the anogenital region was inflamed. A blood sample was collected from the vena *cephalica antebrachii* for complete blood count.

The blood tests showed the presence of erythrocytosis (polycythemia). The X-ray findings showed pneumonia, an enlarged liver and spleen, a large amount of fatty tissue in the abdomen and thorax with a minimal amount of gas in the right hemithorax. The ultrasound of the thorax and heart showed a physiological dimension of heart with a suspicious thoracic mass. The ferret was rehydrated with balanced crystalloids fluid therapy, phlebotomy was performed. CT imaging under general anesthesia was done but the ferret died during anesthesia.

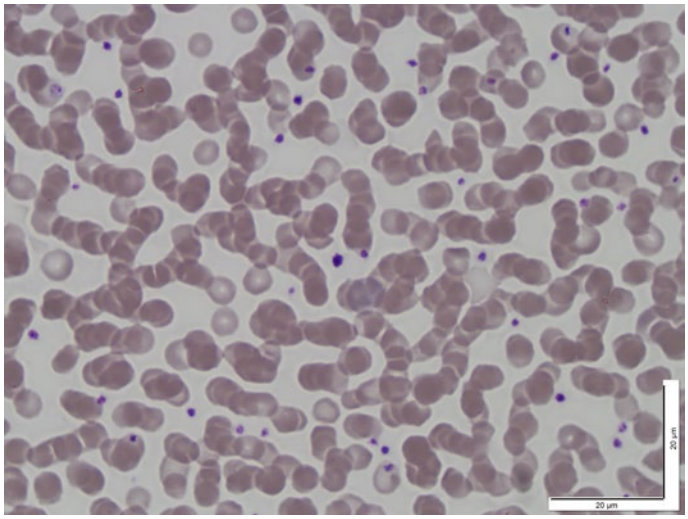
The finding of CT diagnostics showed moderate to severe adipose tissue within the thoracic and abdominal cavity. Left mid-cranial abdominal steatitic, hepatic and splenic nodules, right renal cortical cystic like lesion right hepatic cystic like lesion, cranial mediastinal, left gastric and splenic lymphadenomegaly (reactive vs neoplastic).

Diagnosis: secondary polycythaemia caused by respiratory hypoxia, due to fatty tissue accumulation and suspected neoplastic process.

Parameter	Results	Unit		Reference range		
WBC	3.8	10^9/l	▼	5.6	10.8	10^9/l
LYM	0.8	10^9/l	▼ 21.2 %	1.7	4.7	10^9/l
MON	0.4	10^9/l	12.2 %	0.0	0.4	10^9/l
GRA	2.6	10^9/l	66.6 %	0.6	7.0	10^9/l
HGB	238	g/l	▲	152	177	g/l
MCH	16.4	pg		0.0	99.9	pg
MCHC	329	g/l		320	380	g/l
RBC	14.47	10^{12}/l	▲	6.80	11.80	10^{12}/l
MCV	49.9	fl		43.0	51.0	fl
HCT	72.3	%	▲	46.0	57.0	%
RDW	19.1	%	42.7 fl	0.0	99.9	%
PLT	130	10^9/l	▼	250	1000	10^9/l
MPV	7.7	fl		0.0	99.9	fl



Case E04



Microscope image

Blood smear from ferret showing abundant RBCs and PLTs.

Erythrocytosis in ferrets

In ferrets, erythrocytosis (often referred to as polycythemia) has not been sufficiently investigated and therefore species-specific data are lacking. For this purpose, previous findings were taken from the dogs and cats (due to morphological similarity).

In physiological conditions, erythropoiesis is regulated by erythropoietin (EPO) and renal oxygenation. The factors that induce production of EPO include renal hypoxia, anemia, impaired renal blood flow, hypoxemia, and cardiopulmonary dysfunction.

Erythrocytosis is the term that indicates an increase in RBC count, hemoglobin concentration, and hematocrit. Erythrocytosis is caused by relative, absolute or transient elevation in the number of circulating erythrocytes.

The consequence of polycythemia is a hyperviscosity that leads to decreased capillary blood flow and tissue hypoxia. Hyperviscosity can also lead to the activation of the coagulation cascade, increasing the risk of thrombosis.

Relative erythrocytosis occurs when plasma water is lost without concurrent loss of RBCs. The temporary reduction in plasma volume, for example, hemoconcentration, is the most common in practice or a temporary increase in the percentage of total body RBCs in circulation.

Absolute erythrocytosis refers to the true elevated RBC volume mass and is classified like primary or secondary erythrocytosis.

Primary erythrocytosis (polycythaemia vera) is a myeloproliferative disease of the bone marrow that results in expansion of erythroid cells, independent from EPO.

Secondary erythrocytosis occurs from excessive EPO-dependent stimulation of erythrocyte production. Secondary erythrocytosis is further divided into secondary appropriate erythrocytosis (response to tissue hypoxia) and secondary inappropriate erythrocytosis (abnormal production of EPO).

Relative erythrocytosis, typically, increases HCT only mildly, and clinical signs are usually associated with the underlying disease, rather than erythrocytosis. Relative erythrocytosis may occur with a marked dehydration from water deprivation, vomiting, diarrhea, heat stroke, burns, hyperventilation, diuresis, or shifts of vascular fluid into interstitial space.

In secondary erythrocytosis, there is an appropriate physiologic response in conditions associated with systemic hypoxia. The most common causes of appropriate secondary erythrocytosis are congenital heart defects (e.g., ventricular septal defect, tetralogy of Fallot, reverse patent ductus arteriosus), and other less common causes, such as chronic pulmonary disease, upper airway obstruction, brachycephalic syndrome, severe obesity, methemoglobinemia.

Case E04

Secondary inappropriate erythrocytosis occurs when EPO levels rise in the absence of systemic hypoxia. Common causes are renal hypoxia or EPO-producing tumors. Associated diseases include renal tumors, polycystic kidney disease, renal amyloidosis, hydronephrosis, necrotizing pyelonephritis, glomerulonephritis, hyperadrenocorticism, hyperthyroidism, pheochromocytoma, leiomyosarcoma, hyperandrogenism, hepatic neoplasia, overuse of exogenous EPO, and, in one case report, splenic hemangiosarcoma.

Physical abnormalities, clinical signs, and clinical manifestation may include ataxia, seizures, mentation changes, depression, weakness, exercise intolerance, circling, paresis, blindness, uveitis, vomiting, diarrhea, hyperemic or cyanotic mucous membranes, lethargy, anorexia, splenomegaly, hepatomegaly, epistaxis, hyphema, gastrointestinal bleeding, cutaneous erythema, cardiac murmur, tachycardia, tachypnoea, hyperthermia, signs of thrombosis, polydipsia, polyuria, and cardiopulmonary signs.

The treatment depends on the underlying cause of the disease, but immediate therapy is fluid therapy and phlebotomy.

Leukemia and lymphoma

Case F01

Leukemia/lymphoma in dog

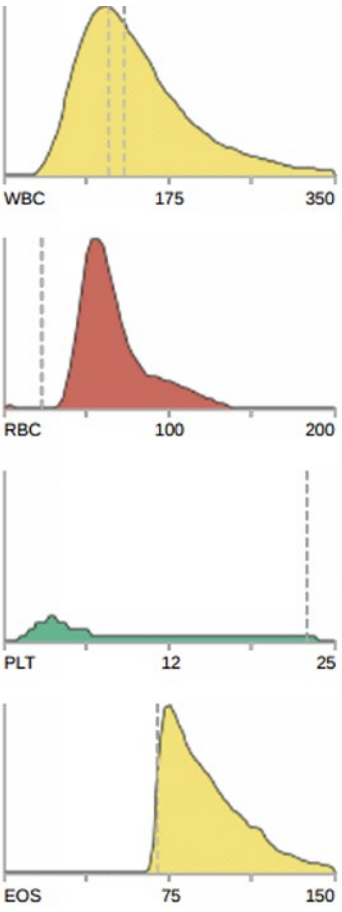
A ten-year-old, male, castrated flat-coated retriever, Matt, was initially presented due to a chronic inappetence and vomiting. He was admitted to a local clinic for a general check-up, where generalized lymphadenopathy was found and was referred to clinic for internal medicine. The owner stated that Matt did not have any other comorbidities and was generally healthy before last week, when symptoms of vomiting and inappetence started. He was vaccinated for rabies and infectious diseases and had protection against ectoparasites.

General physical examination revealed that Matt was very calm, and submandibular, prescapular, and popliteal lymph nodes were enlarged. Temperature, pulse, and respirations were all within normal limits.

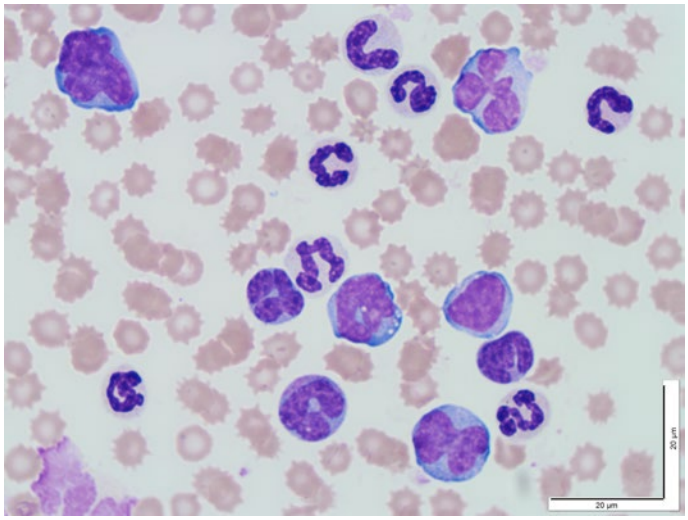
Complete blood count revealed severe leukocytosis with severe lymphocytosis, severe monocytosis, and mild neutrophilia. Blood smear cytology revealed multiple atypical lymphocytes. In biochemistry profile, a marked increase in ALP activity and mild hypoalbuminemia were found. Fine-needle aspiration (FNA) of enlarged lymph nodes was done and cytology showed suspected lymphoproliferative disease. Chest and abdominal X-ray were without remark. Differential diagnosis: end stage lymphoma vs leukemia. Although lymphoma cannot be ruled out at this point, leukemia was a more probable diagnosis considering bloodwork cytology.

To obtain definitive diagnosis, further diagnostics was required and it involved blood flow cytometry and bone marrow aspiration. The owners opted for euthanasia considering the high costs of diagnostics and treatment with poor prognosis.

Parameter		Results	Unit		Reference range
WBC		149.9 10^9/l	▲		6.0 – 17.0 10^9/l
LYM	MM	52.7 10^9/l	▲	35.1 %	0.9 – 5.0 10^9/l
MON	MM	23.1 10^9/l	▲	15.5 %	0.3 – 1.5 10^9/l
NEU	MM	62.7 10^9/l	▲	41.7 %	3.5 – 12.0 10^9/l
EOS		11.4 10^9/l	▲	7.7 %	0.1 – 1.5 10^9/l
HGB		96 g/l	▼		120 – 180 g/l
MCH		26.0 pg	▲		19.5 – 25.5 pg
MCHC		381 g/l			320 – 385 g/l
RBC		3.69 10^{12}/l	▼		5.50 – 8.50 10^{12}/l
MCV		68.3 fl			60.0 – 72.0 fl
HCT		25.2 %	▼		37.0 – 55.0 %
RDW		13.9 %		53.5 fl	12.0 – 17.5 %
PLT		36 10^9/l	▼		200 – 500 10^9/l
MPV		9.9 fl			5.5 – 10.5 fl



Case F01



Microscope image

Blood smear from dog showing cytological appearance of leukemia.

Parameter	Result	Indication	Reference interval	Unit
ALB	25.8	L	26.0–33.0	g/L
TP	63.7		55.0–75.0	g/L
GLOB	38		19.0–45.0	g/L
A/G	0.68			
TB	3.5		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	101	H	0–82	U/L
ALT	23		0–88	U/L
ALP	718	H	20–156	U/L
AMY	> 4000	H	0–1600	U/L
Crea	90.7		44.0–140.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	5.19		3.30–8.30	mmol/L
BUN/CREA	57.205		16.000–218.000	
GLU	5.63		3.60–6.50	mmol/L
TC	4.53		3.50–7.50	mmol/L
TG	0.79		0.20–1.30	mmol/L
tCO2	13.3		12.0–27.0	mmol/L
Ca	2.16		2.10–3.10	mmol/L
PHOS	1.44		0.70–2.10	mmol/L
Mg	0.78	L	0.80–1.10	mmol/L
K^+	5.02		3.60–5.80	mmol/L
Na^+	145.4		140.0–155.0	mmol/L
Cl^-	111.9	H	90.0–110.0	mmol/L

Leukemia

Leukemia is a broad term for cancers of the blood cells and lymphatic system. The type of leukemia depends on the type of blood cell that becomes cancer and whether it grows quickly or slowly. Leukemia represents less than 10% of hematopoietic neoplasia in dogs and around 15% to 35% in cats. Cell lineage identification is an important step in the classification of leukemia, as therapeutic protocols will depend on it. Initially, leukemia is classified in lymphoproliferative or myeloproliferative disorders, and then in their acute or chronic forms and cell subtypes. Lymphocytic leukemia develops from cells that give rise to T lymphocytes, B lymphocytes, or natural killer cells. Myeloid leukemia develops from cells that give rise to the WBC subgroups granulocytes and monocytes.

Case F01

Acute leukemia is usually associated with a more aggressive onset of disease, a short survival time in untreated cases and the involvement of blast cells. Chronic leukemia has a slower progression, and the neoplastic cells are more differentiated and usually have a normal appearance. Symptoms are usually absent in the early stage of disease, in the advanced stage of disease symptoms are very unspecific and can be lethargy, inappetence, vomiting, weight loss and fever.

A diagnosis of leukemia is usually made by analyzing a patient's blood sample through a complete blood count, or microscopic evaluation of the blood, or by using flow cytometry, in some cases bone marrow biopsy is required. Special stains (cytochemistry), monoclonal antibodies techniques (immunocytochemistry and flow cytometry), and molecular genetic tests may be necessary to reach a specific diagnosis. Additional tests are used in the differentiation of chronic well-differentiated leukemia from inflammatory or benign reactive conditions. Other causes of a lymphocytosis should be ruled out if hematologic abnormalities or the clinical presentation/history does not make leukemia an obvious conclusion. Many of these lymphocytosis is transient and should not last more than a several weeks. Samples for the determination of leukemia are typically taken from bone marrow using a bone marrow aspirate.

Treatment involves chemotherapy and supportive care. The side effects of chemotherapy and its relative effectiveness make the pursuit of a specific diagnosis the first goal in the management of leukemic patients. In general, the prognosis for chronic leukemia is better than for the acute one. Supportive therapy and the following up of clinical signs and the complete blood count of the patient is important for therapeutic and prognostic purposes. Every effort to maintain the patient's quality of life should be done.

Leukemia and lymphoma

Case F02

Lymphoma in dog

A 9-year-old, female-spayed labrador, Kira, was initially presented due to an acute onset of lethargy and inappetence. Kira started to be calmer five days ago, the owner noticed that she was refusing food and water, and the owner also had a feeling that she had some problems swallowing. Two months prior, Kira had a similar episode of inappetence, which was treated with corticosteroids for a short period of time, and she was better for that period. She was currently not receiving any therapy but ecto/endoparasite prophylaxis.

General physical examination revealed that Kira was calm, had a body score condition of 3/9, and her temperature, pulse, and respirations were all within normal limits. All peripheral lymph nodes were enlarged, and the palpation of abdomen showed suspected multiple masses in mesogastrium and enlarged liver and spleen. Lung respiration sounds auscultation and heart auscultation showed no abnormalities.

A complete blood count and biochemistry profile was performed and revealed severe leukocytosis with severe lymphocytosis, moderate monocytosis, and mild thrombocytopenia. Biochemistry profile shows only mild dehydration. Based on the signalment, history, and clinical exam findings, a suspicion of lymphomegaly (reactive or neoplastic) was made.

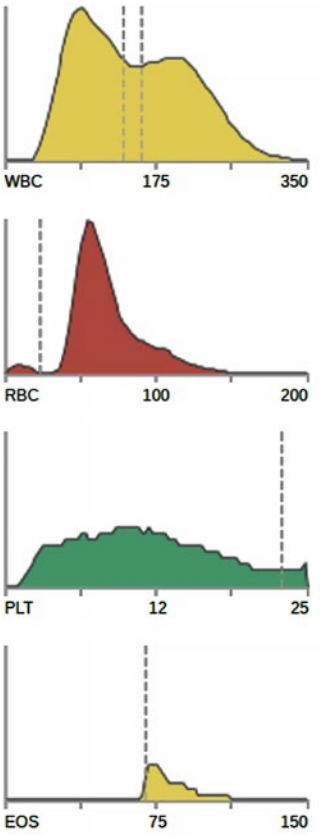
A thoracic radiographic study included two projections, left lateral (LL) image and a ventrodorsal (VD) image, and showed no abnormalities. An abdominal radiographic study included left lateral (LL) image and a ventrodorsal (VD) image, which showed enlarged liver and spleen, and severely enlarged sublumbal lymph nodes.

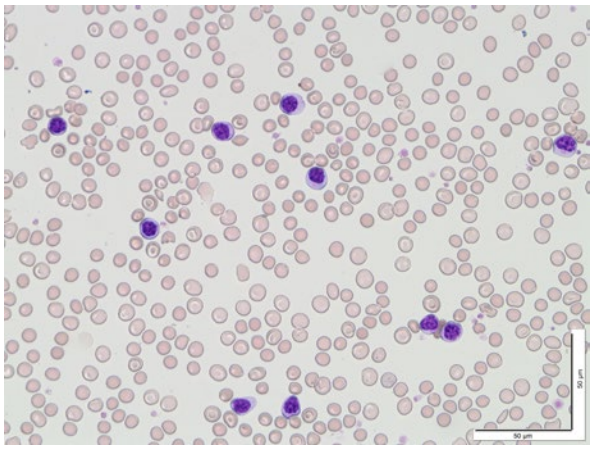
Also, a fine-needle aspiration (FNA) of peripheral lymph nodes was done and cytological examination and immunocytochemistry was performed. Cytological examination showed suspicion on lymphoma, which was confirmed by immunocytochemistry, so the diagnosis of malignant B lymphoma was established.

The chemotherapeutic protocol was started (Madison-Wisconsin weekly protocol). In the first week, vincristine (0.5 mg/m^2) was given with supportive care. After several days, Kira was feeling better and peripheral lymph nodes were 50% smaller in size. After about ten days Kira was brought to emergency because of acute onset of weakness and lethargy. Kira was admitted in ICU for intensive care, but owners decided to do a euthanasia considering poor prognosis.

Case F02

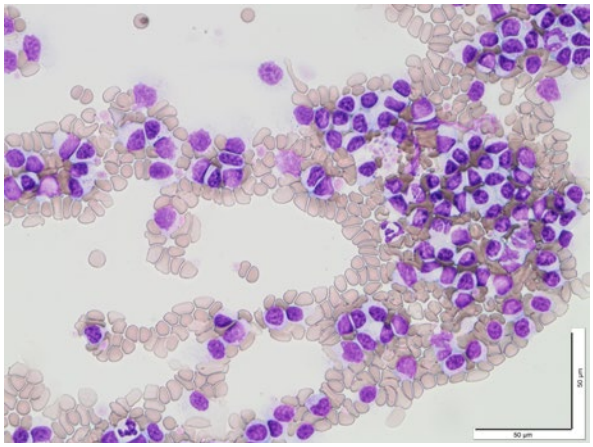
Parameter	Results	Unit		Reference range		
WBC	29.8	10^9/l	▲	6.0	17.0	10^9/l
LYM	14.3	10^9/l	▲ 47.8 %	0.9	5.0	10^9/l
MON	2.5	10^9/l	▲ 8.5 %	0.3	1.5	10^9/l
NEU	12.9	10^9/l	▲ 43.2 %	3.5	12.0	10^9/l
EOS	0.1	10^9/l	0.5 %	0.1	1.5	10^9/l
HGB	132	g/l		120	180	g/l
MCH	23.6	pg		19.5	25.5	pg
MCHC	335	g/l		320	385	g/l
RBC	5.59	10^{12}/l		5.50	8.50	10^{12}/l
MCV	70.4	fl		60.0	72.0	fl
HCT	39.4	%		37.0	55.0	%
RDW	14.3	%	53.1 fl	12.0	17.5	%
PLT	163	10^9/l	▼	200	500	10^9/l
MPV	11.1	fl	▲	5.5	10.5	fl





Microscope image

Blood smear from dog showing cytological appearance of a lymphoma.



Microscope image

Feathered edge of blood smear from dog with lymphoma.

Case F02

Parameter	Result	Indication	Reference interval	Unit
ALB	33.9	H	26.0–33.0	g/L
TP	57.9		55.0–75.0	g/L
GLOB	24		19.0–45.0	g/L
A/G	1.41			
TB	4.2		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	34		0–82	U/L
ALT	73		0–88	U/L
ALP	269	H	20–156	U/L
AMY	578		0–1600	U/L
Crea	116.1		44.0–140.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	7.35		3.30–8.30	mmol/L
BUN/CREA	63.299		16.000–218.000	
GLU	7.89	H	3.60–6.50	mmol/L
TC	3.62		3.50–7.50	mmol/L
TG	1.62	H	0.20–1.30	mmol/L
tCO2	20.4		12.0–27.0	mmol/L
Ca	2.45		2.10–3.10	mmol/L
PHOS	1.62		0.70–2.10	mmol/L
Mg	0.99		0.80–1.10	mmol/L
K^+	4.39		3.60–5.80	mmol/L
Na^+	146.5		140.0–155.0	mmol/L
Cl^-	103.6		90.0–110.0	mmol/L

Lymphoma

Canine lymphoma is a one of the most common types of neoplasia in dogs with an estimated incidence rate of 20 to 100 cases per 100 000 dogs and is comparable to non-Hodgkin lymphoma in humans. Although the exact cause is unknown, environmental factors and genetic susceptibility are thought to play an important role. Lymphoma can affect any dog breed, but middle-sized to larger dog breeds are overrepresented. Lymphoma can be diagnosed at any age, but predominantly affects middle-aged to older dogs with the incidence rate increasing with age from 1.5 cases per 100 000 dogs for dogs < 1 year of age to 84 per 100 000 for dogs > 10 years. The most common clinical presentation of canine lymphoma is the multicentric form that affects the peripheral lymph nodes, but other forms exist and include mediastinal, gastrointestinal, hepatic, splenic, renal, cutaneous, ocular, central nervous system, and pulmonary lymphoma. For diagnosis and determination of type, it is mandatory to do a complete bloodwork with fine needle biopsy (FNB) of the lymph nodes. Sometimes, cytology is not enough for a complete diagnosis and tissue biopsy is required to perform histological study. Given the systemic nature of lymphoma, chemotherapy is considered the therapy of choice. The goal is to obtain a maximum effect with a minimum of consultations, drug administrations, and toxicity. There are several comprehensive chemotherapeutic protocols currently described for treatment of lymphoma. Many prognostic factors have been evaluated in the dog and include clinical data, pretreatment clinical pathology results, histology, immunophenotype, grade, proliferation markers, molecular prognosticators, and biomarkers.

Platelet abnormalities

Case G01

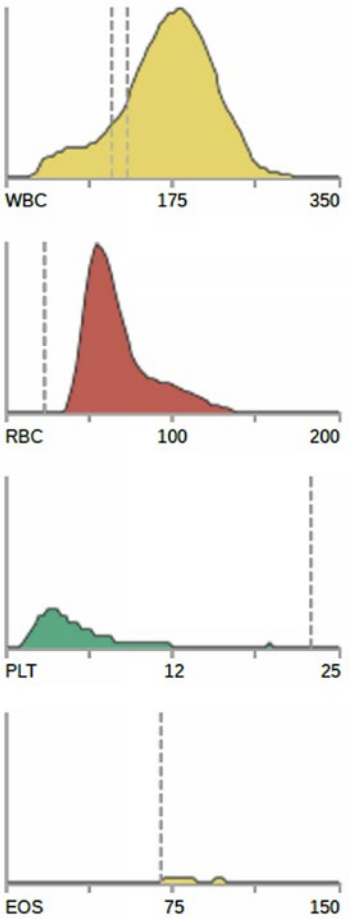
Immune-mediated thrombocytopenia (IMT) in dog

A 10-year-old, male, intact, Maltese, Barney, was initially presented due to an acute onset of lethargy, inappetence, and hematuria. He was currently on his vaccines and is currently not receiving any ecto/endoparasite prophylaxis. General physical examination revealed that Barney had ecchymosis on skin of abdomen and petechiae on mucous membranes. A complete blood count was performed and revealed thrombocytopenia. Serum biochemistry was without remark with mild azotemia, but CRP levels showed a marked increase. PCR for vector borne diseases was negative.

Radiography of thorax and abdomen was performed to exclude other conditions, such as effusions or neoplasms disease and showed no relevant abnormalities.

Based on the signalment, history, and clinical exam findings, a suspicion of immune-mediated thrombocytopenia was made. Platelet-associated antibodies were determined and tested positive. Barney was started on immuno-suppressive dose of corticosteroids and had a good response to therapy.

Parameter	Results	Unit		Reference range		
WBC	9.2	10^9/l		6.0	17.0	10^9/l
LYM	1.2	10^9/l	13.1 %	0.9	5.0	10^9/l
MON	0.6	10^9/l	7.0 %	0.3	1.5	10^9/l
NEU	7.3	10^9/l	79.3 %	3.5	12.0	10^9/l
EOS	0.0	10^9/l	▼ 0.6 %	0.1	1.5	10^9/l
HGB	182	g/l	▲	120	180	g/l
MCH	24.0	pg		19.5	25.5	pg
MCHC	347	g/l		320	385	g/l
RBC	7.60	10^{12}/l		5.50	8.50	10^{12}/l
MCV	69.0	fl		60.0	72.0	fl
HCT	52.4	%		37.0	55.0	%
RDW	13.2	%	53.4 fl	12.0	17.5	%
PLT	42	10^9/l	▼	200	500	10^9/l
MPV	7.4	fl		5.5	10.5	fl



Case G01

Parameter	Result	Indication	Reference interval	Unit
ALB	21.6	L	26.0–33.0	g/L
TP	64		55.0–75.0	g/L
GLOB	42.4		19.0–45.0	g/L
A/G	0.51			
TB	0.1		0.0–8.6	µmol/L
GGT	<2		0–5	U/L
AST	21		0–82	U/L
ALT	35		0–88	U/L
ALP	127		20–156	U/L
AMY	1772	H	0–1600	U/L
Crea	162.9	H	44.0–140.0	µmol/L
UA	<10.00		0.00–60.00	µmol/L
BUN	12.15	H	3.30–8.30	mmol/L
BUN/CREA	74.622		16.000–218.000	
GLU	6.43		3.60–6.50	mmol/L
TC	5.77		3.50–7.50	mmol/L
TG	1.07		0.20–1.30	mmol/L

Immune-mediated thrombocytopenia

The thrombogram includes all tests that evaluate platelets, including the PLT count and assessment of MPV.

PLT counts can be done manually using a hemocytometer or with an automated analyzer. Counts can also be estimated during blood smear examination. Automated analyzers can measure the MPV in femtoliters (fL). Large platelets are usually young. Thrombocytopenia, a decreased number of PLT, can be a consequence of impaired thrombopoiesis, increased platelet destruction and consumption, and sequestration of platelets (splenomegaly). More than one pathophysiological mechanism can occur in the same disorder, further complicating diagnostic and therapeutic decisions.

Severe thrombocytopenia can be a life-threating disease, as it can cause spontaneous bleeding. Spurious thrombocytopenia can be found as macrotrombocytes in Cavalier King Charles and platelet aggregates with many diseases and collection techniques. Greyhounds have a mild thrombocytopenia.

Decreased PLT production may be associated with an overall decreased hematopoiesis due to many drug reactions (chemotherapeutics, estrogens), infections (*Ehrlichia* spp.), and myelophthisis (leukemia, myeloma, myelofibrosis) but is often idiopathic or immune-mediated.

Increased PLT destruction is commonly associated with IMT, but can also be found with neoplasia, disseminated intravascular coagulation (DIC) or vasculitis. IMT can be divided into primary (idiopathic thrombocytopenia purpura [ITP]) and secondary forms triggered by infections (vector borne diseases, vaccines), drugs, and neoplasia. Primary ITP is characterized by immune-mediated destruction of either circulating PLTs or marrow megakaryocytes. It has been seen in dogs, horses, and rarely cats. Middle-aged female or spayed female dogs, especially Cocker Spaniels, are over-represented.

Most common clinical signs of severe thrombocytopenia are petechiae, ecchymosis, scleral and retinal bleeding epistaxis, gastrointestinal bleeding, and hematuria. Anemia can be due to blood loss or due to immune-mediated hemolytic anemia (Evans' syndrome).

Diagnostic procedures usually include a careful history, clinical examination, a search for an underlying cause (blood smear, serology, PCR). Also, diagnostic procedures for excluding neoplasia are commonly done (diagnostic imaging, fine needle aspiration, biopsy, etc.). Bone marrow aspiration is relatively invasive but may sometimes reveal a specific cause. A diagnosis of ITP is mostly based upon excluding other causes of thrombocytopenia, and platelet-associated antibodies can be determined to confirm IMT. Therapy of thrombocytopenia depends on the underlying cause and severity of clinical signs. Therapy usually includes withdrawal of any potential triggering agents, supportive care, and correction of severe anemia. Platelet transfusions are very rarely indicated. Affected animals should be kept at rest.

Case G01

In cases of IMT, immunosuppressive therapy is used to inhibit platelet phagocytosis by macrophages and platelet-antibody production, and glucocorticosteroids are the main immunosuppressive agents. Most clinicians start off with prednisone at immune-suppressive dose, and therapy tapering and is usually lasting for months. Also, vincristine is considered highly effective and relatively safe in the initial management of severe acute cases. Vincristine has been shown to accelerate the PLT count increase, when used in combination with prednisone. The PLT count should be monitored as steroids are decreased because relapses may occur. In uncomplicated cases of IMT, the prognosis is good, and a response is generally expected within days with PLT counts rising above the level of 40 000/µL,

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