Veterinary Association of Namibia

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OFFICIAL NEWSLETTER OF THE VETERINARY ASSOCIATION OF NAMIBIA

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Presidents Desk

VAN hosted a successful and well attended CPD on "Blood-smear interpretation" at the Central Veterinary Laboratory on 1st April 2017. Suggestions for further CPD training are always welcome.

The VAN Congress date (12-14 October 2017) has been communicated to the Chief Veterinary Officer of Veterinary Service and all VAN members. Heja Lodge is booked for the event and organization is well on the way.

VAN was represented at a consultative meeting aimed at drafting amendments to the Medicines and Related Substances Control Act, Act 13 of 2003. It was organized by the Veterinary Council on 28 March 2017. The labelling issue of Schedule 0 medications was addressed as well as the problems pharmaceutical companies encounter with the registration of medications.

VAN supported the first meeting of the Para veterinarians on 30th March 2017, called the Namibian Veterinary Technicians Association (NVTA). We from VAN wish them all the best for the future.

The UNAM veterinary students will have to start with clinical experience. VAN members are asked to support UNAM veterinary students as they go into their clinical years.

Exco is communicating with UNAM about an intended "Veterinary Pharmacy degree". More information will have to be gathered about this subject, but, in general VAN is not in favour of this and has drafted a letter. It was circulated to VAN members for comments.

Kind regards

Beate Voigts

VAN CPD

Van hosted another successful CPD event on the first of April 2017. It was presented by Dr. Ulf Tubbesing, a private practitioner of Windhoek.

The topic of this CPD dealt with blood smears. How to make a proper blood smear and how to interpret it. Basic haematology was also covered. Then a few case studies were handled, including haematology and serum chemistry.

A total of 35 veterinarians attended this event on a Saturday afternoon. The response from the veterinarians was very good and the venue was packed.

VAN News

There is an App from the OIE that can be downloaded onto your cell phone. It is called WAHIS. It alerts you on any new diseases outbreaks world wide.

The Animal Health Consultative Forum held a meeting on the 13th April 2017. VAN would just like to highlight one point. As Tuberculin is very sensitive to temperature changes, veterinarians should only use Tuberculin that was delivered to them directly, not Tuberculin that has been delivered to the producer.

VAN is currently supporting the veterinary student Margareta Kangono. She has not passed her last semester and will only receive funds again once she has passed.

We are busy giving our website a 'face lift'. Thank you to all VAN members who contributed pictures of our furry friends. Keep a look out for the new and improved site.

VAN would like to welcome their new members:

- Dr. Christian B Witbooi
- Dr. Lauren Leathem

Unfortunately there are quite a few VAN members who have failed to pay their annual fees. The VAN constitution is quite clear on this matter. Invoices are sent out 1st of July each year. Payment is expected until latest 1st September. In case of non payment a notice is sent out for payment to be done by 1st October. If no payment is received then the membership will be suspended. Early bird congress fees are only available to VAN members with paid up membership.

The rabies eradication project is being supported by VAN. VAN is sponsoring multiple rabies vaccination certificates.

"It's simple. My nurse blindfolds me, I spin around a few times, and then I try to reattach your tail."

Q Fever

This article is intended to raise awareness on a disease that the veterinary profession and probably the medical profession as well have generally neglected. It has both a significant zoonotic potential and is a serious cause of reproduction losses in farm animals. According to DVS reports, the disease was last diagnosed in the Grootfontein District in 1991.This year (2017) the disease was confirmed at four localities in Zambezi, Karas and Khomas regions all from aborted goat foetuses.

A limited serological survey carried out in sheep on 9 farms in Hardap and Karas region (A. Bishi et al, in press) revealed a flock level prevalence of 78% (7 out of 9 farms) and 18% of the 273 individual animals had antibodies against *C. burnetii*. Although limited and not representative of the general population of ruminants in Namibia, the survey nevertheless revealed, that the bacterium was present in the population. Further investigations are required to establish how widespread infections by *C. burnetii* are and what impact it has on humans and animals.

Coxiellosis is the name of the disease which affects mainly domestic ruminants and is caused by an obligate intracellular gram negative bacterium called *Coxiella burnetii*. In humans, the disease is called Q fever. The Q stands for query, as before the bacterium was isolated in a human patient in 1937 by Burnet and Freeman, the causative agent had been unknown.

The literature reports that the disease causes significant economic losses in domestic ruminants as a result of abortions and stillbirths. In humans, the disease is characterised by fever, severe pneumonia or hepatitis, these may progress to debilitating chronic forms that may be fatal when endocarditis develops. Infected animals excrete the highest load of the organisms at parturition in birth products, faeces, urine, milk, sputum, semen, and vaginal fluids.

Transmission to humans (commonly veterinarians, veterinary paraprofessionals, farmers and farm workers who work closely with animals) and animals occur when they come into contact with infected animals particularly their birth products following abortion or normal birth. Infection can also occur by inhalation of pathogen-contaminated dust or aerosols and may also occur via tick bite or ingestion of contaminated milk. It is reported in the literature that the disease in humans is usually misdiagnosed as non-malarial fever or fever of unknown origin.

Literature shows that the disease is probably ubiquitous with high sero-prevalence in humans and ruminants ranging from as low as 3 to over 50% with veterinarians having a higher sero-prevalence compared to the general population. In Australia Q fever is rated as the most diagnosed zoonotic disease affecting veterinarians. It is thus not clear why the disease is underreported or not reported at all in some parts of the world. Perhaps it is because veterinarians and farmers alike have neglected this disease, or its impact is poorly understood, while they put emphasis on diseases like brucellosis and campylobacteriosis which have an impact on trade.

According to Dr Umberto Molini at the Central Veterinary Laboratory the test in use is the real-time PCR specific for the DNA gyrase subunit A. Suitable samples for PCR are the aborted fetus (all the internal organs can be infected by *C. burnetii)*, placenta from aborted fetus and vaginal discharge soon after abortion or parturition (not more than 8 days after). PCR also works well in milk samples suspected to be contaminated by the bacteria. Samples need to be sent to the laboratory in a cool box, because high temperatures can support the growth of other bacteria usually present in the samples that may result in false negative results.

Article by Alec Bishi

Top 5 Leukogram Patterns

Sarah Schmidt, DVM, DACVP (Clinical Pathology)*
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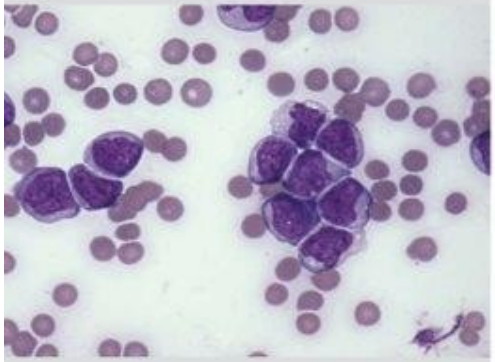


Top 5 Leukogram Patterns

1. Stress Leukogram
2. Inflammatory Leukogram
3. Persistent Lymphocytosis (of Mature Lymphocytes)
4. Neutropenia
5. Eosinophilia

A CBC is often completed as part of the minimum database. When the CBC is performed with an in-house hematology analyzer, a blood smear evaluation should be pursued as well to verify automated results, including confirmation of white blood cell (WBC) differential and evaluation for atypical cells, such as mast cells, blasts, and nucleated red blood cells. Following confirmation of automated results, evaluation of the leukogram, which includes total WBC count, absolute values of individual leukocytes, and leukocyte morphology, should be performed to identify any pattern. When evaluating WBC differentials, absolute cell numbers (WBC count × leukocyte %) should be interpreted as opposed to leukocyte percentage only.

1 Stress Leukogram

A stress leukogram is characterized by neutrophilia, lymphopenia, eosinopenia, and potentially monocytosis.[1] It occurs primarily in dogs. The term *stress* denotes the presence of increased cortisol released from the adrenal gland secondary to severe disease (eg, diabetic ketoacidosis, renal failure), high body temperature, pain, dehydration, or hyperadrenocorticism.[1]

- Neutrophilia occurs because of increased release of mature neutrophils from the bone marrow storage pool to circulating blood and decreased movement of neutrophils from circulating blood to tissue.
- Lymphopenia results from retention of lymphocytes in lymphoid organs and lymphocyte lysis.
- Eosinopenia is caused by decreased release of eosinophils from the bone marrow and increased lysis.
- Lymphopenia is the hallmark of a stress leukogram. While the degree of neutrophilia may decrease over time, lymphopenia and often eosinopenia will persist as long as plasma concentrations of steroid hormones are increased.
- Monocytosis is often found in dogs because of movement of monocytes from the marginating to circulating pool. Monocytosis can be variably found in feline stress leukograms.

A stress leukogram may also be seen following administration of exogenous glucocorticoids. A single dose of glucocorticoids may result in a stress leukogram lasting for approximately 24 hours; glucocorticoids administered for longer than 10 days may result in a stress leukogram that persists for 2–3 days after the drug is discontinued.[2] The magnitude of the neutrophilia is usually up to twice the upper limit of the normal reference interval.

2 Inflammatory Leukogram

An inflammatory leukogram may be characterized by neutrophilia, or in some instances neutropenia, with or without a left shift or toxic change. Inflammatory mediators result in the release of neutrophils from the bone marrow storage pool to the circulation, and an increased rate of bone marrow neutrophil production, with or

*At the time of publication, Dr. Schmidt works with IDEXX. At the time of article creation, Dr. Schmidt was an independent contractor.

without the presence of a left shift and toxic change. Following stimulation of the bone marrow by inflammatory mediators, it takes approximately 2 to 5 days for maturation and release of neutrophils.

A left shift indicates that immature (nonsegmented) neutrophils have been released from the bone marrow into circulation prior to maturation to segmented cells.

Toxic change includes Dohle bodies (pale blue irregularly shaped cytoplasmic inclusions), increased cytoplasmic basophilia, and foamy/vacuolated cytoplasm. Toxic change indicates accelerated production of neutrophils by the bone marrow (**Figure 1**).

In general, as the left shift or degree of toxic change increases, so does the severity of the underlying inflammatory process.

While neutrophils are emerging from the bone marrow, circulating neutrophils are migrating to the source of the inflammation. The presence of a neutrophilia vs neutropenia depends on the balance between neutrophil production and increased tissue demand.

Inflammatory diseases include infectious causes (bacterial, fungal, viral, protozoal), immune-mediated diseases (immune-mediated hemolytic anemia, polyarthritis), and tissue necrosis. Many inflammatory diseases result in a neutrophilia that is 2 to 10 times the upper limit of the normal reference interval.

Other diseases in which the infection is isolated, such as a walled-off abscess, may result in a marked neutrophilia because production and release from bone marrow is high but tissue consumption of circulating neutrophils is low as the cells cannot easily infiltrate the lesion.[1]

After the source of the inflammation is removed (eg, infected uterus), neutrophilia may persist for 2–5 days because earlier stimulated neutrophil precursors will continue with maturation and the source of tissue consumption no longer exists.

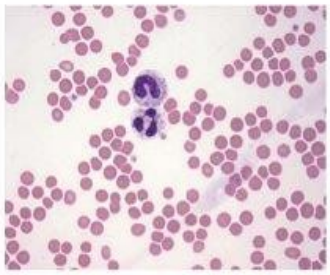
3 Persistent Lymphocytosis (of Mature Lymphocytes)

As lymphocytes may transiently increase following excitement or exercise secondary to epinephrine, a lymphocytosis should be verified by a repeat CBC. A review of the blood smear is indicated if the lymphocytosis is persistent, as differentials will be dependent on lymphocyte morphology (small, mature lymphocytes vs lymphoblasts) (**Figures 2** and **3**).

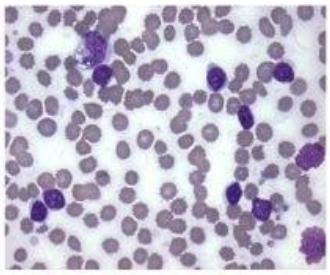
Persistently increased numbers of small to intermediate lymphocytes with condensed (mature) chromatin can be the result of a neoplastic lymphocytosis or a nonneoplastic lymphocytosis.

Chronic lymphocytic leukemia (CLL) is a type of neoplastic lymphocytosis in which lymphocytes appear small to intermediate in size and have condensed chromatin. The absolute lymphocyte count for CLL can be vary between 6000/µL and 8000/µL in dogs and cats, respectively, to greater than 150,000/µL.[3]

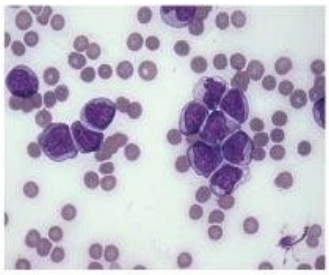
Lymphocyte counts greater than 20,000–30,000/µL are generally more



1 A toxic band neutrophil is found at the top of the image above a toxic segmented neutrophil. Both cells have increased cytoplasmic basophilia and foamy cytoplasm. (Modified Wright's stain, 50× original magnification)



2 The majority of the leukocytes are small lymphocytes with condensed chromatin. A vacuolated monocyte is present at the top left of the image. Two broken cells are present at the right side of the image. (Modified Wright's stain, 50× original magnification)



3 The leukocytes are blast cells, most suggestive of lymphoblasts. The cells are approximately 12–15 µm in diameter with immature chromatin and variably distinct nucleoli. (Modified Wright's stain, 50× original magnification)

CLL = chronic lymphocytic leukemia, WBC = white blood cell

continues

worrisome for CLL than for nonneoplastic conditions, although some overlap may occur.

Causes for nonneoplastic lymphocytosis include diseases that result in immune stimulation, including *Ehrlichia canis* infections in dogs, feline leukemia/feline immunodeficiency virus infection in cats, and *Mycoplasma felis* infection in cats.[2,3]

Thymoma and hypoadrenocorticism are differentials for dogs and cats. Hyperthyroidism and immune-mediated hemolytic anemia are additional differentials for cats. Flow cytometry of a fresh blood sample (ideally within 2 days) can greatly aid in determining a neoplastic lymphocytosis from a nonneoplastic lymphocytosis. The presence of blast cells should be reviewed by a clinical pathologist.

4 Neutropenia

Neutropenia can be caused by increased consumption of recently released and circulating mature segmented cells, decreased production, and immune-mediated destruction. Increased consumption can result from severe inflammatory disease, bacterial infections, or endotoxemia. Decreased production can result from insult to myeloid precursor cells from drugs or toxins or replacement of myeloid precursors by neoplastic cells. Immune-mediated destruction is suspected when other causes of neutropenia have been ruled out and the patient responds to immunosuppressive drugs.

The cause for neutropenia may be multifactorial as seen in parvovirus infections, in which the virus attacks rapidly dividing myeloid precursor cells and compromises gastrointestinal integrity, resulting in endotoxemia, as well as feline leukemia virus infections that can damage hematopoietic precursor cells and lead to secondary infections that consume circulating neutrophils.[4,5]

5 Eosinophilia

Increased bone marrow production of eosinophils is most commonly the result of parasite antigens or allergens. Parasites commonly associated with eosinophilia are those found within tissues including *Dirofilaria immitis, Aelurostrongylus abstrusus, Toxocara* spp, *Toxascaris leonina, Ancylostoma caninum, A braziliense, A tubaeforme,* and *Uncinaria stenocephala.* Blood cell parasites, including *Mycoplasma* spp, *Babesia* spp, and *Cytauxzoon* spp, do not typically result in an eosinophilia. Hypersensitivity reactions secondary to cutaneous allergens such as flea saliva are commonly associated with eosinophilia, whereas hypersensitivity reactions to inhaled allergens (atopic dermatitis) are generally not.[6]

Eosinophilia has also been reported with generalized inflammation (including bacterial infections) of tissues rich in mast cells, including the lungs, intestinal tract, skin, and genitourinary tract. Reported eosinophil counts that have been associated with various diseases affecting these tissues range from 2000–21,000/µL with a median value of generally less than 7000/µL.[6]

Hypoadrenocorticism (Addison's disease) is another consideration for eosinophilia and has been associated with eosinophil counts of 2000–4000 µL.

Idiopathic hypereosinophilic syndrome, which is characterized by eosinophil tissue infiltration, circulating granulocytes with mature morphologic features, and lack of anemia or thrombocytopenia is strongly considered with eosinophil counts greater than 20,000 µL.

Conclusion

Evaluation of the leukogram pattern can aid in formulating differential diagnoses. ■ cb

CLL = chronic lymphocytic leukemia

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Suggested Reading

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