Diagnostic Update

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Treatment of Methicillin-Resistant *Staphylococci pseudintermedius* in Dogs

By Sandra McConkey, Veterinary Clinical Pathologist and Pharmacologist

In the August 2013 issue of Diagnostic Update, Matthew Saab reviewed his work on methicillin-resistant staphylococci in Atlantic Canada. In this issue, I will discuss the treatment of methicillin-resistant *Staphylococcus pseudintermedius* (MRSP) in dogs.

In humans, *Staphylococcus aureus* is a common commensal organism and is carried in the nasal passages of ~30-40% of people. In contrast, *Staphylococci pseudintermedius* is a common commensal organism of the skin, nares, mouth and anus of dogs. At the Atlantic Veterinary College, *S. pseudintermedius* is routinely cultured from canine ears, bladders, infected wounds, areas of skeletal infection and surgical sites.

What should you do if you are treating a dog with MRSP?

- Infected animals should be kept in isolation in the clinic.
- Draining wounds should be covered.
- Wash or disinfect hands frequently.
- Use gowns and gloves or dedicated laboratory coats for handling the animal and when cleaning kennels, bowls or changing bandages.
- Disinfect all possible fomites regularly.

Fortunately MRSP is sensitive to most disinfectants.

Most *S. pseudintermedius* organisms are sensitive to β lactams, especially cephalosporins. If a *Staphylococcus* develops resistance to some β lactams by producing a β-lactamase, the resistance can be easily overcome by administering β-lactamase-resistant drugs such as amoxicillin with clavulanic acid (e.g. Clavamox®). However if a *S. pseudintermedius* acquires the *mecA* gene encoding a particular altered penicillin binding protein, then it becomes resistant to all β lactams, cephalosporins and carbapenems, including β-lactamase resistant drugs. Fortunately, MRSP organisms are no more virulent or difficult to disinfect for than non-MRSP organisms. The prognosis for an infected patient reflects the location and severity of the infection rather than the presence of an MRSP. Choosing an antimicrobial to treat an MRSP can be challenging. If the *S. pseudintermedius* has only the resistance gene *mecA*, then there are still many antimicrobials other than β lactams available to use. However, *S. pseudintermedius* with *mecA* genes are more likely to acquire additional resistance genes (co-resistance) that confer resistance to other antimicrobials as well. The additional resistance is most commonly to macrolides (e.g. erythromycin), lincosamides (e.g. clindamycin), tetracycline, fluoroquinolones and potentiated sulfonamides.

Owners should be warned of the potential for zoonotic transmission and the need for strict hygiene. The risk of clinical disease is relatively low for immunocompetent people but it can occur. The risk is greater for immunosuppressed people or animals. The risk of transmitting an MRSP to another animal or human is highest when there is an active clinical lesion. Investigate and treat any underlying causes of pyodermas. Use topical therapy to both restore the structure and function of the skin as well as to kill the bacteria if the MRSP infection is mild or localized.

This can be done by frequent bathing with shampoos and conditioners in addition to frequent sprays and rinses with disinfectants. Localized methicillin-resistant infections can be treated with targeted topical medications such as chlorhexidine sprays, fusidic acid or mupirocin applied BID daily until the infection has resolved. If a superficial pyoderma is more generalized, then go with a more aggressive broader topical therapy such as frequent antibacterial shampoos and appropriate antibacterial sprays BID. Chlorhexidine is a good choice as it has been reported to be more efficacious than benzoyl peroxide, ethyl lactate and chloroxylenol against MRSP.

Systemic antimicrobials are required in addition to topical therapy for severe infections. Choose the antimicrobial based on culture and sensitivity. Occasionally a minimum inhibitory concentration (MIC) result may suggest that there is still sensitivity to a particular β lactam but this is not true for any MRSP. Do NOT treat an MRSP with any β lactam drug, including β lactams that are combined with a β-lactamase-resistant product such as Clavamox®. In addition, do not use a fluoroquinolone even if the MIC indicates that the lesion is still sensitive to fluoroquinolones because this may select for a highly resistant mutant strain of bacteria that is resistant to fluoroquinolones and several other antimicrobials.

MRSP organisms with additional multidrug resistance are usually still sensitive to chloramphenicol, rifampin and an aminoglycoside such as amikacin. Remember that chloramphenicol can cause dose dependent reversible bone marrow suppression (especially in cats) and rare idiosyncratic irreversible pancytopenia in people. It can also cause gastrointestinal upset, inappetence and weight loss sufficient to stop therapy. It inhibits hepatic P450 enzymes which can interfere with the metabolism of other drugs, especially anticonvulsants. Amikacin may be preferred over gentamicin because it is less nephrotoxic but the animal should still have a urinalysis done twice weekly and treatment should stop immediately if there is any evidence of renal toxicity. Like gentamicin, amikacin must be given parenterally but amikacin can be given subcutaneously by an owner at home. Resistance to rifampin can develop quickly when it is used as a monotherapy. Rifampin can induce hepatic enzymes (especially ALP) and can cause rare, potentially fatal hepatotoxicity in dogs. It can also cause hemolytic anemia, thrombocytopenia, orange discoloration of body fluids and is a potent inducer of P450 enzymes. Do not exceed a dose of 10 mg/kg. If the culture and sensitivity indicates that a tetracycline can be used, then use doxycycline or minocycline as both are at least as active and may be better than tetracycline.

Resolving MRSP infections may be more difficult to treat than resolving infections due to methicillin sensitive *S. pseudintermedius*. This is not because the organisms are more virulent, but because of the chronicity of the infection and the secondary skin pathologic changes. Treat superficial infections for a minimum of 3-4 weeks with 1 week of therapy past clinical resolution. Deep pyodermas should be treated for at least 4-8 weeks with two to three weeks of therapy beyond complete healing.

References:

1. Cain C. Antimicrobial Resistance in Staphylococci in Small Animals. *Vet Clin Small Anim.* 2013;43:19-40.
2. Coyner KS. Managing MRSA, MRSP, and MRSS dermatologic infections in pets. Part 1 of a two-part series on methicillin-resistant staphylococci. *Vet Med*. 2012;107:516-521.
3. Coyner KS. The emergence and prevalence of MRSA, MRSP, and MRSS in pets and people. Part 2 of a two-part series on methicillin-resistant staphylococci. *Vet Med.* 2013;108:32-38.
4. Papich MG. Selection of antibiotics for methicillin-resistant *Staphylococcus pseudintermedius*: time to revisit some old drugs? *Vet Dermatol*. 2012;23:352-360. DOI:10.1111/j.1365-3164.2011.01030.x
5. Weese JS, Faires MC, Frank LA, Reynolds LM, Battisti A. Factors associated with methicillin-resistant versus methicillin-susceptible *Staphylococcus pseudintermedius* infection in dogs. *J Am Vet Med Assoc.* 2012;40:1450-1455.
6. Young R, Buckley L, McEwan N, Nuttall T. Comparative *in vitro* efficacy of antimicrobial shampoos: a pilot study. *Vet Dermatol*. 2011;23:36-40.

To optimize the CBC results for your patients, please remember to submit an unstained air dried blood smear with the lavender topped tube of blood .

Eosinophilia in a Himalayan cat

By Dania Villarnovo, Veterinary Clinical Pathology Resident

Recently, we came across the case of Zoey, a 1 year and 4 month old female spayed Himalayan cat (Figure 1). She presented to her veterinarian for decreased appetite, vomiting, hematemesis, diarrhea, and bloody stool. EDTA-anticoagulated whole blood was submitted to the Diagnostic Laboratory Services at the Atlantic Veterinary College for a complete blood count (CBC). It revealed a marked leukocytosis characterized by a marked eosinophilia, mild lymphocytosis, and moderate monocytosis (Table 1). The marked eosinophilia was composed of large numbers of band eosinophils. An in-house ELISA FIV/FeLV test was negative. The veterinarian started Zoey on supportive therapy including prednisone, Convenia® (cefovecin sodium), Milbemax® (milbemycin oxime and praziquantel), and sucralfate. A repeat CBC 10 days later showed a marked improvement in the eosinophilia (Table 1) and she had improved clinically with resolution of the vomiting, diarrhea, and anorexia. Within an additional 2 weeks, the eosinophil count returned to within the reference interval (Table 1). Currently, Zoey continues to do well while on prednisone therapy.

Table 1: Selected CBC results for Zoey

Parameter	Day 1	Day 10	Day 24	Day 43	Reference Interval	Units
WBC count	**74.2**	16.5	8.9	8.4	4.7 – 17.0	x 10^9/L
Segmented neutrophils	5.2	9.3	4.4	5.9	2.2 – 9.5	x 10^9/L
Eosinophils	**58.6**	**2.8**	0.1	0.3	0.0 - 1.5	x 10^9/L
Lymphocytes	**8.9**	4.1	4.0	2.2	0.5 – 7.5	x 10^9/L
Monocytes	**1.5**	0.3	0.4	0.0	0.0 - 0.6	x 10^9/L

Although a definitive diagnosis was not obtained in Zoey's case, it is interesting to consider differential diagnoses for cases of marked eosinophilia in cats. Broad categories of disease that cause eosinophilia include hypersensitivity reactions, parasitism, inflammation of mast cell-rich tissues, endocrine disease (hypoadrenocorticism), idiopathic hypereosinophilic syndrome, a paraneoplastic situation, and eosinophilic leukemia.[1] Hypersensitivity reactions with eosinophilia commonly occur with flea-bite dermatitis, eosinophilic granuloma complex, and feline bronchial asthma. Parasitism is more likely to result in eosinophilia when parasites are migrating through tissue. Ectoparasites, heartworm, and respiratory tract parasites such as *Paragonimus kellicotti* and *Aelurostrongylus abstrusus* are frequently noted to cause eosinophilia. The skin, respiratory tract, intestinal tract, and urogenital system are mast cell-rich tissues. Degranulation of mast cell contents with inflammation at these sites can be a chemoattractant to eosinophils and result in increased numbers of circulating eosinophils in blood. As cortisol suppresses inflammatory responses, a lack of cortisol can be accompanied by eosinophilia in 10-20% of canine cases of hypoadrenocorticism (incidence unknown for cats as the disease is rare in this species).[2] Paraneoplastic eosinophilia has been reported with mast cell neoplasms, lymphomas (including T-cell and gastrointestinal), carcinomas, and myeloproliferative disease in cats.[1, 3-5] Some of the cats with lymphoma and myeloproliferative disease have been FeLV positive.

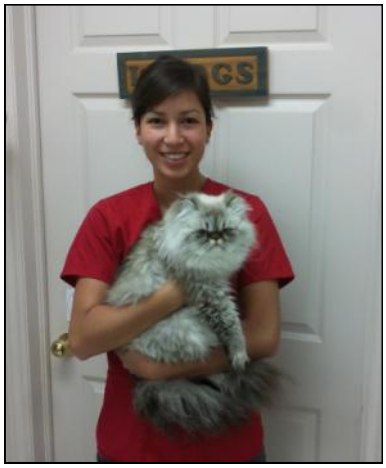

Figure 1: Zoey being held by Chantelle Fearn, a veterinary technology student, at her clinic.

Among these differentials, the most common causes of eosinophilia are flea allergy dermatitis (up to 23.5 x 10^9/L), gastrointestinal disease including endoparasites and inflammatory bowel disease (up to 12.5 x 10^9/L), focal infection or inflammation such as abscesses and trauma (up to 9.4 x 10^9/L), feline asthma (up to 20.7 x 10^9/L), and eosinophilic granuloma complex (up to 46.2 x 10^9/L).[3] Paraneoplastic eosinophilia is usually also marked but values are typically less than 5.0 x 10^9/L with the exception of rare cases of disseminated mast cell neoplasia and transitional cell carcinoma.[4-6] An eosinophilia greater than 50.0 x 10^9/L is characteristically seen with idiopathic hypereosinophilic syndrome and chronic eosinophilic leukemia.[7] These two disorders are difficult to differentiate as both can have a persistent marked eosinophilia with cellular infiltration of various tissues and organs (liver, spleen, small intestine, and lymph nodes). Idiopathic hypereosinophilic syndrome is a diagnosis of exclusion with greater prevalence in middle-aged domestic short-hair cats and possible overrepresentation in females. These patients have a hypercellular bone marrow with hyperplasia of the eosinophil lineage, a myeloid to erythroid ratio <10, orderly maturation, and a lack of immature myeloid precursors in the blood. Anemia, thrombocytopenia, and neutropenia are usually not noted.[1] In contrast, chronic myeloid leukemias may have mature and

immature eosinophils in the bone marrow and blood with dysplastic changes, a high bone marrow myeloid to erythroid ratio, and a concurrent anemia, thrombocytopenia, and/or neutropenia. This type of leukemia has been reported in FeLV positive cats.

Given the degree of eosinophilia in Zoey's case, our top differentials were idiopathic hypereosinophilic syndrome and chronic eosinophilic leukemia. Without further diagnostic testing such as an assessment of visceral organs and bone marrow, a definitive diagnosis cannot be made. We wish Zoey the very best!

References:

1. Stockham SL, Scott MA. Leukocytes. In: *Fundamentals of Veterinary Clinical Pathology*. 2nd ed. Iowa: Blackwell; 2008:53-106.
2. Klein SC, Peterson ME. Canine hypoadrenocorticism: Part I. *Can Vet J.* 2010;51:63-69.
3. Center SA, Randolph JF, Erb HN, Reiter S. Eosinophilia in the cat: A retrospective study of 312 cases (1975 to 1986). *J Am Anim Hosp Assoc*. 1990;26:349-358.
4. Peaston AE, Griffey SM. Visceral mast cell tumour with eosinophilia and eosinophilic peritoneal and pleural effusions in a cat. *Aust Vet J.* 1994;71:215-217.
5. Sellon RK, Rottman JB, Jordan HL, et al. Hypereosinophilia associated with transitional cell carcinoma in a cat. *J Am Vet Med Assoc.* 1992;201:591-593.
6. Bortnowski HB, Rosenthal RC. Gastrointestinal mast cell tumors and eosinophilia in two cats. *J Am Anim Hosp Assoc*. 1992;28:271-275.
7. Gelain ME, Antoniazzi E, Bertazzolo W, Zaccolo M, Comazzi S. Chronic eosinophilic leukemia in a cat: Cytochemical and immunophenotypical features. *Vet Clin Pathol.* 2006;35:454-459.

Good luck Shannon!

By Andrea Bourque, Veterinary Anatomic Pathologist

It is with heavy hearts that we say a very fond goodbye to Dr. Shannon Martinson as she leaves Diagnostic Services for a new exciting position at the University of New Hampshire and the New Hampshire Veterinary Diagnostic Laboratory.

Shannon has been an important part of our team of anatomic pathologists since finishing her MVSc and residency in 2007 here at the AVC. Shannon's genuine passion for and expertise in her specialty makes her an incredible asset to any institution lucky enough to have her. An excellent diagnostician, she has been an important contributor to our surgical biopsy and post-mortem services, providing timely, clinically relevant results to our clients. Over the years, Shannon has also become an excellent teacher to both veterinary students and residents alike. Her use of interesting case material, her engaging teaching style and her practical and clinically relevant approach to teaching make her lectures a favorite among students (whether pizza is served or not!). Her knowledge and experience in reptile pathology, her sense of humor, her collaborative approach to work, and love of pathology will be sorely missed.

As friends and colleagues, we wish Shannon, her family, her mutant weiner dogs and crazy Beagle all the best in New Hampshire!

This is a reminder that **Diagnostic Services is open Saturday mornings** from 8 am until 12 pm to perform routine diagnostic testing. Please contact Midland Courier for specimen pick up on Friday for Saturday delivery. Terry Beek is the Midland Courier contact if you experience any difficulties and he can be reached at 506-858-7667. If you have any questions or comments regarding the Saturday service please contact Liz Dobbin, Director of Diagnostic Services, by phone at 902-566-0831 or by e-mail at edobbin@upei.ca. Liz would be appreciative of your feedback on the service.

Don't Forget the Platelets!!

By Linda Ruschkowski, Veterinary Laboratory Technologist

Platelets are often overlooked in favor of their more colorful neighbors, the red and white blood cells. However, evaluation of platelet numbers and characteristics is extremely important.

Most veterinary laboratory instruments will provide an electronically generated platelet count. Nevertheless, we highly encourage all our clients to validate this instrument generated number by evaluating a blood smear. When looking at the blood smear, ask yourself a few questions:

1. Are there any platelet clumps? Before you hit the panic button and think your patient is going to bleed out imminently from thrombocytopenia, check the feathered edge of the blood smear for platelet clumps (Figure 1). Also check the K_3-EDTA (purple topped) blood tube for any small clots. If either is seen, the instrument generated platelet count is artifactually lowered and not reliable. Obtaining a new sample is always best if possible.
2. Does the electronic number reflect the number of platelets seen on the blood smear? To evaluate this, do a rough estimate of the platelet numbers. You should see at least 8-10 platelets per high powered field (/hpf) using the 100x objective in the monolayer of the smear for most veterinary species for the platelets to be considered adequate in number. For horses, ~5 platelets/hpf is adequate. Here are 2 ways to estimate platelet numbers in the monolayer of blood smears with evenly distributed platelets (no platelet clumping):

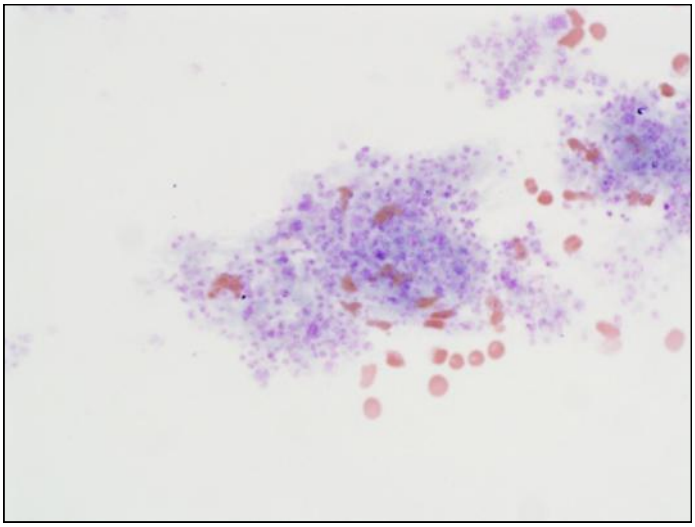

Figure 1: Platelet clumps at the feathered edge of a canine blood smear. Wright-Giemsa, x 60 objective.

   a. Count the total number of platelets in 10 random fields (100x objective) and divide this number by 10 to get the average number. Multiply this average number by 15 – 20 to obtain a range of the estimated platelet count in units of x 10^9/L. *Example:* If 120 platelets are counted in 10 random fields, the average number is 12 (120 ÷ 10 = 12). This number (12) is multiplied by 15 and then also by 20 to obtain an estimated platelet count of between ~ 180 - 240 x 10^9/L.
   b. If you have a reliable red blood cell count (RBC) available, you can also use another method. Count the platelets in 5 fields (100x objective) of ~200 red blood cells each (essentially you are counting the # of platelets seen while 1000 red blood cells are seen). Multiply this number by the RBC count in units of 10^{12}/L. This will give you an estimation of platelet numbers in units of x 10^9/L. *Example:* If 60 platelets are present over 5 fields of ~ 200 RBCs each and the patient RBC count is 6.5 x 10^{12}/L, the estimated platelet count is 60 x 6.5 =390 x 10^9/L.
3. Do the platelets appear normal sized or is there a significant population of larger platelets? Giant platelets may indicate active thrombopoiesis in response to various conditions.

Some disease states potentially causing thrombocytopenia require careful platelet monitoring. Some of these include immune-mediated thrombocytopenia, disseminated intravascular coagulation, certain infectious diseases such as *Anaplasma phagocytophilum* or effects due to chemotherapy or neoplasia.

Finally, an interesting aspect about platelets is that some Cavalier King Charles Spaniels can have platelet numbers below the normal canine reference interval, but these platelets are often larger than normal. This is due to an inherited β-tubulin defect and the condition is sometimes termed macrothrombocytopenia.[1] These dogs have an overall normal body platelet mass and are asymptomatic. It is important to recognize this inherited trait as it is completely benign, lifelong and does not require therapy. Without this knowledge, completely healthy dogs of this breed can be mistakenly treated with immunosuppressive drugs for a presumed situation of immune-mediated thrombocytopenia. Not all Cavalier King Charles Spaniels have this condition but it is important to keep it in mind when evaluating platelet parameters in individuals of this breed.

Reference:

1. Davis B, Toivio-Kinnucan M, Schuller S, Boudreaux MK. Mutation in β1-Tubulin correlates with macrothrombocytopenia in Cavalier King Charles Spaniels. *J Vet Intern Med.* 2008;22:540-545.

What is Pelger-Huët Anomaly?

By Cornelia Gilroy, Veterinary Clinical Pathologist

A 2 year old intact female mixed breed dog named Annie had a CBC and serum biochemical panel performed prior to a routine ovariohysterectomy. Annie's initial CBC results are listed in Table 1.

Table 1: Initial CBC data

Parameter	Annie's Value	Reference Interval	Units
WBC	10.2	5.4 – 14.3	x 10^9/L
Hct	0.42	0.40 – 0.56	L/L
RBC	6.2	5.7 – 8.4	x 10^{12}/L
Hgb	145	135 – 198	g/L
MCV	67	64 – 75	fL
MCHC	349	334 – 357	g/L
Platelets	**487**	218 – 470	x 10^9/L

The mild thrombocytosis was likely due to redistribution of platelets secondary to excitement. Upon microscopic evaluation of the blood smear, it was noted that the neutrophils and eosinophils had hyposegmented nuclei, a mature condensed chromatin pattern and no toxic change (Figure 1). Annie was diagnosed with Pelger-Huët anomaly.

Pelger-Huët anomaly is an uncommon inherited disorder that occurs in many veterinary species, including dogs, cats, horses and rabbits. A variety of dog breeds can be affected with a higher incidence reported in Australian Shepherd dogs. With this anomaly, the mature granulocyte nuclei do not become lobulated. As such, the nuclei of neutrophils (and often eosinophils and/or basophils) appear round, oval, peanut shaped, spectacle shaped or resemble a band. The key to differentiating these cells from immature neutrophils associated with inflammation is the chromatin pattern and whether toxic change is observed or not. The chromatin pattern of cells in Pelger-Huët affected animals is mature and condensed, while immature neutrophil bands and metamyelocytes have an open nuclear chromatin pattern and often cytoplasmic toxic change (Figure 2). It is important to make this distinction so that a patient with Pelger-Huët anomaly is not misdiagnosed as having an inflammatory condition and receives unnecessary therapy. If a patient affected with Pelger-Huët anomaly has an inflammatory response at the time a CBC is performed, it can be very difficult to diagnose the anomaly until the inflammation has resolved.

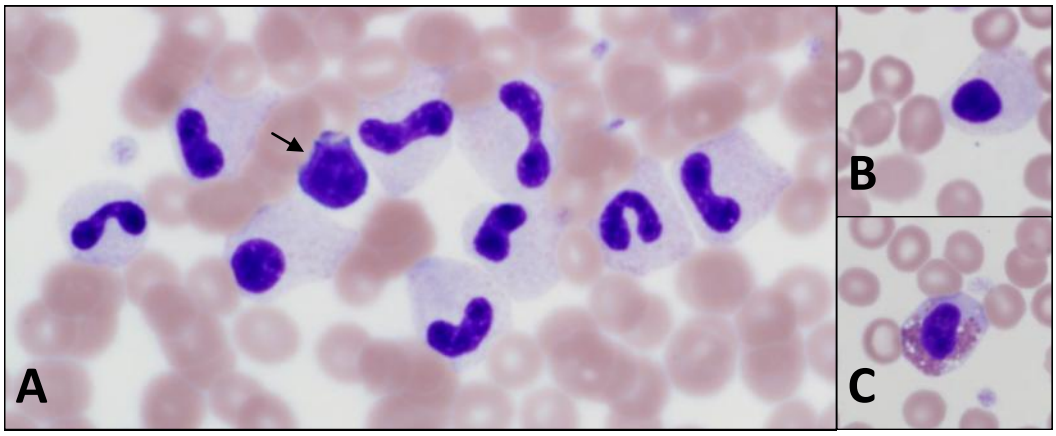

Figure 1: Hyposegmented neutrophils (A & B) and an eosinophil (C) in a dog with Pelger-Huët anomaly. A single lymphocyte (arrow) is present (A). Wright-Giemsa, x 100 objective.

Pseudo-Pelger-Huët anomaly is also uncommon. It is an acquired condition of granulocyte hyposegmentation secondary to a disease or disorder such as some infections, myelodysplastic syndromes or treatment with certain drugs. If the underlying cause is removed, the hyposegmentation resolves.

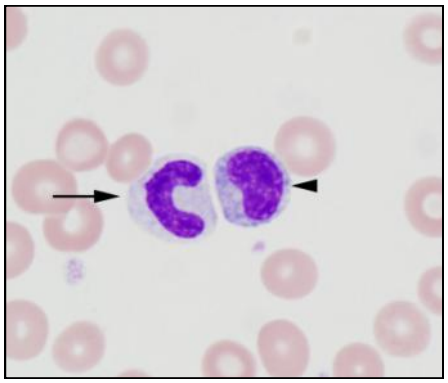

Figure 2: Band neutrophil (arrow) and metamyelocyte (arrowhead) from a dog with inflammation. Note the open chromatin pattern of the nuclei and the toxic change (mostly foamy cytoplasmic basophilia). Wright-Giemsa, x 100 objective.

The granulocytes in heterozygote patients affected with Pelger-Huët anomaly function normally. Therefore, this condition is typically detected in healthy patients as an incidental finding, as was the case with Annie.

References:

1. Stockham SL, Scott MA. Leukocytes. In: *Fundamentals of Veterinary Clinical Pathology*. 2nd ed. Iowa: Blackwell; 2008:53-106.
2. Vale AM, Tomaz KLR, Sousa RS, Soto-Blanco B. Pelger-Huët anomaly in two related mixed-breed dogs. *J Vet Diagn Invest.* 2011;23:863-865.
3. Weiss, DJ. Neutrophil function disorders. In: *Schalm's Veterinary Hematology*. 6th ed. Iowa: Wiley-Blackwell; 2010:275-280.

Porcine Epidemic Diarrhea Virus (PEDV) Testing at AVC Diagnostic Services

A Real Time polymerase chain reaction (PCR) for detection of PEDV is now available at the Regional Diagnostic Virology Services (RDVS) at the Atlantic Veterinary College. The recommended specimens are fresh fecal samples or fresh intestinal contents from acutely infected pigs.

Porcine Epidemic Diarrhea is an acute highly contagious disease characterized by severe enteritis and diarrhea with very high morbidity affecting all age groups of pigs and very high mortality in suckling pigs. The disease has been reported in several states in the United States since the spring of 2013. The PEDV has not been reported in Canada*.

Please contact the RDVS Laboratory by phone at 902-566-0877, if you have any questions or if you need more information with regards to this new test.

***Please Note:** Since the production of this volume of the Diagnostic Update Newsletter **cases of Porcine Epidemic Diarrhea Virus have been confirmed in Ontario.**

Laboratory News

By Cornelia Gilroy, Veterinary Clinical Pathologist

Here are some recent happenings in Diagnostic Services:

- We bid farewell to one of our hematology technologists, Jennifer Boutilier, who left to begin a new job as a technologist in her hometown of Bridgewater, Nova Scotia. We wish Jennifer all the best!
- We welcomed our new hematology technologist, Alfred Mitchell, who began working with us in August 2013.
- We bid Dr. Shannon Martinson farewell as she has left Diagnostic Services and has started a new job at the University of New Hampshire and the New Hampshire Veterinary Diagnostic Laboratory (please see full article on page 4).
- We wish all the best to Dr. Lisa Miller, veterinary anatomic pathologist, who recently retired after 24 years of service at the University of Prince Edward Island.
- Dr. Heather Fenton, who recently completed a wildlife anatomic pathology residency, has left the Atlantic Veterinary College to start her new position as a wildlife veterinarian with the Southeastern Cooperative Wildlife Disease Study located at the University of Georgia. This role will allow Dr. Fenton to collaborate with the Canadian Cooperative Wildlife Health Centre. This will serve to strengthen ties between the two centers as they have similar mandates for the detection and management of wildlife, human and domestic animal health internationally.
- Dr. Dania Villarnovo, clinical pathology resident, presented a research abstract at the annual meeting of the American College of Veterinary Pathology and American Society for Veterinary Clinical Pathology in Montreal in November. The abstract focused on Dr. Villarnovo's evaluation of a commercially available major cross-matching kit (RapidVet®-H) for use in dogs.
- Dr. Cornelia Gilroy presented a mystery case at the European Society of Veterinary Clinical Pathology Congress in Berlin, Germany in November.
- Dr. Sandra McConkey, veterinary clinical pathologist and pharmacologist, was a speaker at the 2013 Canadian Veterinary Medical Association convention and at the 2013 Animal Welfare in Practice Conference: Companion Animal Behaviour, held at the Atlantic Veterinary College.

Staff Focus

Dr. Sandra McConkey

By Shelley Burton, Veterinary Clinical Pathologist

Dr. Sandra McConkey is a familiar name to veterinarians in Atlantic Canada, who rely on her timely therapeutic drug monitoring interpretations. Dr. McConkey has the impressive distinction of being both a clinical pharmacologist and a clinical pathologist. We have been pleased to welcome her back to performing clinical pathology duty in our laboratory in recent months!

Originally from Unionville, Ontario, Dr. McConkey graduated from the Ontario Veterinary College in 1985. For nearly 9 years, she worked in a busy small animal practice on PEI; the communication skills and practical knowledge she developed then have made it easy for her to relate to practitioners when discussing cases now as a specialist. In 1994, Sandra entered into a 3 year clinical pathology residency at the Atlantic Veterinary College (AVC). In 1997, she passed (on the first try!) the intensive Certifying Examination of the American College of Veterinary Pathologists (ACVP) to become a boarded Diplomate in clinical pathology. This accomplishment was particularly impressive as it had come on the heels of a busy residency program while raising a young family.

For the next several years, Sandra strengthened her diagnostic and teaching skills by working in the AVC Diagnostic Services Laboratory (including serving as Director in 2001) and teaching 4th year veterinary students and residents. She then pursued graduate training in clinical pharmacology, culminating with her PhD degree in 2008. Dr. McConkey is currently a tenured Associate Professor of Clinical Pharmacology. Her busy professional life includes teaching veterinary and graduate students, performing research and diagnostic work, and providing continuing education, most notably at the Atlantic Provinces Veterinary Conference. Although the majority of her work is in clinical pharmacology, Sandra loves clinical pathology and so enthusiastically and graciously agreed to undertake diagnostic duty in this area again. Her quick mind, wonderful sense of humor and flexible collegial attitude are strong assets to our group.

Sandra has many interests outside of work. Always athletic, she has participated in many marathons, including those in Boston and New York. Travelling is another passion and her sojourns have taken her to many locales, including Japan and Africa. Her interest in reading stimulated her formation of a very enjoyable book club with a group of colleagues. She has a passion for wildlife and is a board member of PEI's Island Nature Trust. Sandra enjoys family time with her husband, Dr. John Drake, and their two grown daughters, Carrie and Katherine. Tracker, their sweet black Labrador Retriever, is a much loved family member as are Hummer, their impressively large Birman cat, and Lou, their much smaller tortoiseshell cat. When on slow walks with the quite elderly Tracker, Sandra has to keep an eye on Hummer, an opinionated cat with a strong personality who has an ill-considered tendency to try to chase off foxes!

Reader Feedback: The ***Diagnostic Update*** group invites comments or suggestions for future topics in the newsletter. Please submit your comments to *Dr. Cora Gilroy* (cgilroy@upei.ca), Diagnostic Services, Atlantic Veterinary College, UPEI, Charlottetown, PE, C1A 4P3 and they will be forwarded appropriately.