

Common Mistakes in Everyday Parasitology

Milen Kostadinov, VD, Veterinarna Klinika Novet, Bulgaria, novet1@yahoo.com

Lecture credentials and potential benefits

- Benefits – to learn to perform better veterinary parasitology on an everyday basis and realize that parasitology can be a practice builder
- Point of view from the level of the practicing veterinarian
- From the standpoint of experience and deeper knowledge in some areas
- Point of view of the manager of two well established veterinary practices

Lesson 1: Patient Evaluation in dirofilariosis (heartworm disease). Emphasis on kidney complications.

Patient Evaluation in dirofilariosis (heartworm disease).

- To diagnose the parasite ≠ to diagnose the patient
- Find what damage is done to the dog. Find if dirofilariosis is the major cause of disease. Positive HW Ag test is not enough. Find the damage >>> Tailor the treatment.
- Perform a Knott test (fresh formalin solution) to see if you have D. repens on top of D. immitis. Observe heads and tails.
 - Knott test – confirm D. immitis. Sometimes you have a positive Ag test of one company, and negative of other. False positive results are very rare, false negative are possible if there just 1-2 worms in a patient. It is quite possible to have many MF in drop of blood or on Knott test and negative Ag test. If you observe Mf in a drop of blood and you have a negative Ag test, you MUST perform Knott test or send blood to experienced parasitologist. D. repens is quite common in all European countries and contrary to what some people think, you cannot tell the difference between Mf of D. immitis and D. repens looking at their motion in a direct smear of drop of blood. Some people get confused, because they read American recommendations and books. These recommendations are written for American vets, not for European vets and are specific for the American continent, where D. repens IS NOT prevalent. When vets in Texas or Florida see a lot of Mf in a drop of blood, it most probably is D. immitis. Sometimes American vets have to distinguish between Mf of D. immitis and Dipelatonema reconditum, another filarioid which importantly is not zoonotic. In Europe we have D. repens which is zoonotic. To give a summary in few words – if

the Ag test is negative, but you have Mf, you have to prove Mf is *D. immitis* by observing the morphology on Knott test and only then you have a parasitological diagnosis *Dirofilariosis*. If you find *D. repens*, you have to treat the dog so it is Mf free and not a risk to humans. *D. repens* is usually non-pathogenic to dogs so adulticide treatment is not justified. Usual monthly microfilaricid drugs used for prevention will clear the existing Mf and prevent further infection.

- More commonly you will have a positive Ag test and no Mf in a drop of blood and negative Knott test. This is absolutely enough to give you a parasitological diagnosis of *Dirofilariosis* and is a common occurrence in chronically infected dog in which the immune system is thought to block microfilariaemia.
- Run CBC and basic biochemistry
- Run urinalysis
- Run thoracic X-rays (Llat, DV)
- If possible run echocardiography
- If possible check blood pressure
- ! Communicate cost to the owner

Patient Evaluation in dirofilariosis

Case 1

- “Mak” – **presentation**. GS, intact male, 5y, 42kg
 - 05.05.2014 Not feeling well, not eating, losing weight, had ticks, had fever, drinks a lot of water. Treated by another vet with Doxycycline 100mg bid and aspirin ?.
 - Aggressive, sedation for examination and procedures. Dehydrated, poor body condition, elevated heart and respiratory rate. BT 39.3°C.
 - 4Dx Plus HW Positive, Knott Positive *D. immitis*
- “Mak” - **laboratory**
 - Bloodwork: UREA 10.0 mmol/L, ALT 103 U/L, Leu $16.1 \times 10^9/L$
 - Urinalysis: SG 1.015, protein SSA ++, dipstick Hb ++, pH 6.5, Sediment : spermatozoa +++, small number Er, Leu and epithelial cells, moderate number of short granular casts.
- “Mak” - **imaging**

Xrays – normal cardiac size and form, no signs of pulmonary effusion, pulmonary arterial hypertension – moderate

Ultrasound: Abdominal ultrasound – no ascites, splenomegaly. Echocardiography – parasites in the main pulmonary artery.

■ “Mak” 05.05.14– **initial treatment**

- Drugs: Doxycycline 5 mg/kg bid for 14 days, Aspirin 0.5 mg/kg sid constantly, Enalapril 0.25 mg/kg bid constantly, selamectine spot on.

Referred to Central VC, Sofia for detailed echocardiography and surgical extraction of worms if possible.

- Central VC, Sofia 07.05.14: pulmonary hypertension is confirmed >>> additional drug for hypertension – sildenafil (short acting phosphodiesterase 5 (PDE5) inhibitor, trade name Viagra) 2 mg/kg bid;
- surgical extraction of 18 worms !

An article about pulmonary hypertension:

<http://veterinarymedicine.dvm360.com/canine-pulmonary-hypertension-part-2-diagnosis-and-treatment?id=&sk=&date=&pageID=5>

■ “Mak” 10.07.14 – **re-evaluation and further treatment**

- 50 days later – clinical exam with sedation medetomidine 0.01mg/kg+butorphanol 0.15mg/kg im – ok, auscultation heart and lungs - normal ;
- Blood tests Leu $21.4 \times 10^9/L$ (were 16.1), light anemia, Tr $158 \times 10^9/L$, ALB 30 g/L
- Urinalysis SG 1.033 (was 1.015), protein SSA +++ (was ++)
- X-rays VHS 11.5, pulmonary hypertension, signs of thrombosis and inflammation
- Melarsomine (Immiticide) 2.5 mg/kg im (Lumbar im injection: in the lumbar region there is very dense fascia caught tightly to the surrounding bones.)
- Metamizole 25 mg/kg sq, dexamethasone 0.1mg/kg sq

■ “Mak” 11.07.14 – **further treatment**

- 24 hours later – Clinical exam – marked swelling in the lumbar region;
- Melarsomine (Immiticide) 2.5 mg/kg im in the reverse muscle
- Metamizole 25 mg/kg sq ,metamizole 12.5 mg/kg po bid for 2 days
- Dog still on enalapril, sildenafil, aspirin.

- Increase the dose of enalapril to 0.5mg/kg po bid. Added furosemide 1 mg/kg po sid and spironolactone 1mg/kg po sid. Marbofloxacin 2mg/kg po sid 14 days
- Strict cage rest
- “Mak” 15.07.14 – **further treatment**
 - 4 days later – call back: lack of appetite, prefers to lie down.
 - Carprofen 2.5 mg/kg sid for 5 days.
 - 15.08.2014, 30 days later – call back: energetic, good appetite, back to his normal self. Should we stop the drugs? Answer – after check of urine and bloods.

Case 1 - discussion

- “Mak” illustrates a situation when the owners of the dog can afford the best care
- The dog was presented with signs of serious chronic disease. Diagnosis should always come first, which is especially important in chronic disease. In such cases we may not have time to play around giving common drugs.
- With serious illness and compliant owner we should not be afraid to offer the best options
- Even with a wealthy owner we should explain the need for everything we do.

Patient Evaluation in dirofilariosis

Case 2

- “Shirkhan”

Middle Asiatic Shepherd Dog, intact male, 2 ½ years old, 64 kg. Had fever, diagnosis ehrlichiosis from another vet clinic. The test run for ehrlichiosis was a combined test. It was positive for dirofilariosis too.

- “Killer”

Middle Asiatic Shepherd Dog, intact male, 2 ½ years old, 67 kg. Tested because of the positive tests of his brother. Turned out positive for dirofilariosis.

- A treatment with melarsomine had been recommended. The owner was worried, wanted a prognosis of treatment (the dogs are expensive breeding animals). Further evaluation had been declined by the other clinic as unnecessary, adulticide treatment had been recommended again.
- The dogs are then brought to us for a consultation and evaluation
- “Shirchan” 14.07.2014

- Clinical exam - normal
 - CBC – moderate thrombocytopenia
 - Blood biochemistry - OK
 - Knott – D. immitis
 - Urinalysis: SG 1.048, protein SSA - , dipstick pH 6.5; Sediment : small number of epithelial cells, no casts
 - X-rays declined due to cost.
 - Initial treatment: doxycycline 5 mg/kg bid for 28 days, selamectine spot on. Adulticide treatment recommended after day 14.
 - **Shirchan is in “NO RISK” group.**
- “Killer” 14.07.2014
 - Clinical exam – from a distance
 - CBC – high Leu $22.4 \times 10^9/L$
 - Blood biochemistry – high TP 84 g/L, GLOB 53 g/L
 - Knott – D. immitis **AND D. repens**
 - Urinalysis: SG 1.047, protein SSA - , dipstick pH 6.5; Sediment : high number of fine granular casts, high number of small cells, high number of big epithelial cells
 - X-rays declined due to cost
 - Initial treatment: doxycycline 5 mg/kg bid for 28 days, moxidectine spot on, enalapril 0.5 mg/kg sid, aspirin 0.5 mg/kg sid. Adulticide treatment recommended after day 14. **Killer is in “RISK” group.**

Case 2 – discussion

- “Shirchan” and “Killer” illustrate a situation when the owner CANNOT afford the best protocol for evaluation
- Still, he is worried and wants safety for his dogs
- He is provided options and we pick easier and cheaper tests
- The clinical picture can be evaluated even with limited information and the dogs treated with safe adulticide protocols.

- We find *D. repens* and motivate the owner to do serious prevention for the sake of human health
- Low cost veterinary medicine may mean to the owner low quality medicine and medicine with unsure outcome

Summary –patient evaluation in dirofilariosis

- When the patient is “not feeling well” we should pursue a diagnosis and evaluate the grade of disease. Especially in chronic disease!
- Most owners will accept the cost of veterinary services if explained and given a choice.
- If we behave like professionals we will be perceived as professionals.

IMMUNE-MEDIATED Glomerulonephritis in HW and other parasitic diseases

- **Definition.**

The two important glomerular diseases of domestic animals are glomerulonephritis (GN) and glomerular amyloidosis. GN can be immune mediated and non-immune. Immune-mediated GN is associated with immune complex deposition in the glomeruli.

IMGN in *Dirofilaria immitis* infection. Also commonly associated with proteinuria. One theory is that immune complexes form in response to shed microfilarial antigens. Another is that the mechanical presence or products of the microfilaria damage the glomerular vessels.

Nonimmune Glomerular Injury: Factors that may damage endothelial cells are:

- a. Hemodynamic factors: Intraglomerular *hypertension* secondary to hyperfiltration may physically damage endothelial cells.
- b. *Coagulation* followed by activation of the platelet release reaction may lead to endothelial damage.
- c. *Hyperlipemia* may cause endothelial damage directly.

- *Marked, persistent proteinuria is the hallmark of glomerular disease.*
- Most **common causes of IMGN in dogs**
 - Pyometra
 - Dirofilariosis
 - Ehrlichiosis , Borreliosis, Brucellosis, etc.

- Neoplasia
- Most important **clinical signs**
 - “not doing well”
 - Poor body condition, poor coat
 - anorexia, weight loss, lethargy, polyuria, polydipsia, vomiting
 - Signs may be related to underlying disease

1. Signs may be related to presence of chronic renal failure if more than 75% of the nephron population has become nonfunctional (e.g., anorexia, weight loss, lethargy, polyuria, polydipsia, vomiting). This is the most common presentation.

2. Signs may be related to an underlying infectious, inflammatory or neoplastic disease.

3. Proteinuria may be an incidental finding detected during diagnostic evaluation of another medical problem.

4. Signs may be related to classical nephrotic syndrome (e.g., ascites, subcutaneous edema).

5. Signs may be related to thromboembolism (e.g., sudden onset of dyspnea with pulmonary embolism, sudden onset of paraparesis with iliac or femoral artery embolism).

6. Sudden blindness may occur due to retinal detachment resulting from systemic hypertension. Retinal hemorrhages, vascular tortuosity, and retinal detachment may occur due to systemic hypertension.

Other physical findings may be related to the presence of underlying infectious, inflammatory, or neoplastic diseases. Affected Shar pei dogs may have a previous history of so-called Shar pei fever (episodic joint swelling usually involving the tibiotarsal joints and high fever that resolve within a few days, regardless of treatment).

- **Most important complications**

- Hypoalbuminemia

1. The hypoalbuminemia of nephrotic syndrome is only partially explained by urinary loss of albumin.

2. Renal catabolism of albumin is increased in nephrotic syndrome due to increased reabsorption of filtered protein.

3. Although an increase in dietary protein stimulates hepatic albumin synthesis it does not correct hypoalbuminemia in patients with nephrotic syndrome and only worsens the urinary losses of protein.

- Thromboembolism and hypercoagulability

1. The nephrotic syndrome is a hypercoagulable state. Occasionally, thromboembolic phenomena are responsible for the major presenting signs and overshadow the underlying renal disease, thus complicating the clinical course and delaying the primary diagnosis.

2. Thromboembolism occurs in 15% to 25% of dogs with nephrotic syndrome. It is rare (but has been reported) in cats with glomerular disease.

3. The pulmonary artery is the most common site for thromboembolism, but emboli also may lodge in mesenteric, renal, iliac, coronary, and brachial arteries as well as in the portal vein. Dogs with pulmonary thromboembolism usually are dyspneic and hypoxic with minimal pulmonary parenchymal radiographic abnormalities.

- Hypertension

1. Systemic hypertension may occur in dogs and cats with glomerular disease due to sodium retention, activation of the RAAS, and impaired release of normal renal vasodilator substances.

2. Systemic hypertension has been associated with immune-mediated GN, glomerulosclerosis, and glomerular amyloidosis and may occur in up to 50% or more of dogs with glomerular disease.

3. Retinal hemorrhage, retinal vascular tortuosity, and retinal detachment may result from systemic hypertension, and blindness may be the presenting complaint in hypertensive dogs and cats.

4. Blood pressure measurements (by Doppler technique) should be obtained in all dogs and cats with suspected glomerular disease because control of systemic hypertension may slow progression of the glomerular disease.

- Hyperlipidemia

1. Hypercholesterolemia and hyperlipidemia are commonly associated with nephrotic syndrome.

2. In nephrotic patients, plasma albumin concentrations are inversely correlated with plasma cholesterol and triglyceride concentrations, and cholesterol and lipid concentrations tend to increase as albumin concentration decreases.

- nephrotic syndrome >>> kidney failure

- **Most important tests in GN**

- Proteins in urine (*Sulfa Salicylic Acid* test also called *Protein Precipitation Test*). It is important to view the samples against a dark field.



Marked persistent proteinuria with an inactive sediment is the hallmark of glomerular

disease. (5-Sulfosalicylic acid is used in urine tests to determine urine protein content. The chemical causes the precipitation of dissolved proteins, which is measured from the degree of turbidity. Technique: Urine mixed with 3% to 5% Sulfosalicylic Acid (SSA)

<http://www.austincc.edu/mlt/ua/UrineProteinSulfosalicylicAcidPrecipitationTest.pdf>

- Proteins in urine (UPRO:UCREA Ratio)
 - a. Avoids the confounding effect of total urine solute concentration (i.e., specific gravity) on qualitative assessment of proteinuria.
 - b. Correlated with 24-hour urinary protein loss but easier to measure (i.e., does not require a 24-hour urine sample).
 - c. Magnitude of increase in urine protein-to-creatinine ratio is roughly correlated with nature of glomerular disease.
 - d. Values are highest in dogs with glomerular amyloidosis (often >10) and lowest in those with interstitial renal disease (usually <10). Animals with GN have very variable values (normal to >30).
 - e. Presence of hematuria or pyuria can make the urine protein-to-creatinine ratio difficult to interpret (i.e., may cause a false-positive result).
 - f. Normal urine protein-to-creatinine ratio is <0.3 in dogs and cats.
 - g. As glomerular disease advances and GFR decreases, less protein is filtered and the urine protein-to-creatinine ratio can decrease. This decrease does not indicate Improvement, and is a poor prognostic sign.
- Urine SG - refractometer



- Urine sediment: hyaline and granular casts, lipid droplets.
- Albumin blood level . Hypoalbuminemia occurs in many dogs with glomerular disease (up to 75% of dogs with amyloidosis and 60% of dogs with GN).
- Urea, creatinin blood levels . Either GN or amyloidosis can lead to chronic renal failure (CRF) with the expected biochemical abnormalities (e.g., azotemia, hyperphosphatemia, metabolic acidosis).
- Sodium, phosphorus, cholesterol blood levels. Hypercholesterolemia occurs in most dogs with glomerular disease (up to 60% of dogs with GN and 90% of dogs with amyloidosis) but is a nonspecific finding in cats with renal disease. Hypercholesterolemia may be due in part to increased hepatic synthesis of cholesterol-rich lipoproteins secondary to chronic hypoalbuminemia.
- **Most important drugs in IMGN**
 - ACE inhibitors. Angiotensin converting enzyme (ACE) inhibitors (e.g., enalapril, benazepril) reduce glomerular capillary hydraulic pressure by decreasing postglomerular arteriolar resistance and, thus, reduce proteinuria. This beneficial effect must be balanced against their potential to worsen azotemia. In one study, enalapril (0.5 mg/kg PO every 12-24 hours) reduced proteinuria (as assessed by urine protein-to-creatinine ratio), reduced blood pressure, and slowed progression of renal disease in dogs with GN.
 - Aspirin In dogs, an aspirin dosage of 0.5 mg/kg PO once a day may selectively inhibit platelet cyclooxygenase without preventing the beneficial effects of prostacyclin formation (e.g., vasodilatation, inhibition of platelet aggregation).
 - Diet. Low salt diet (<0.3% on a dry matter basis).

Removal of the causative antigen may result in resolution of GN (e.g., ovariohysterectomy in dogs with pyometra, treatment of heartworm disease). Provide a renal diet, low-dose aspirin, and ACE inhibitors to patients with GN. Monitor urinary protein concentration as measured by urine protein-to-creatinine ratio. Increases in the urine protein-to-creatinine ratio or microalbuminuria indicate the possibility of

progressive renal injury and justify additional diagnostic evaluation and more aggressive treatment interventions.

- Supportive drugs
 - Diuretics – spironolacton, furosemide may be used in refractory cases but overzealous use may result in dehydration and prerenal azotemia.
 - Further management of hypertension include amlodipine, hydralazine, prazosin, and propranolol. Viagra (sildenafil).
 - Immunosuppressive drugs. Corticosteroid administration can cause proteinuria in dogs and one retrospective study suggested that corticosteroid therapy actually may be detrimental in dogs with idiopathic GN. Corticosteroids may be beneficial (or at least not detrimental) in cats with GN.
 - Omega-3 polyunsaturated fatty acids, (ω -3 PUFA) (e.g., fish oil) may suppress glomerular inflammation and coagulation by interfering with production of pro-inflammatory prostanoids.

Glomerulonephritis

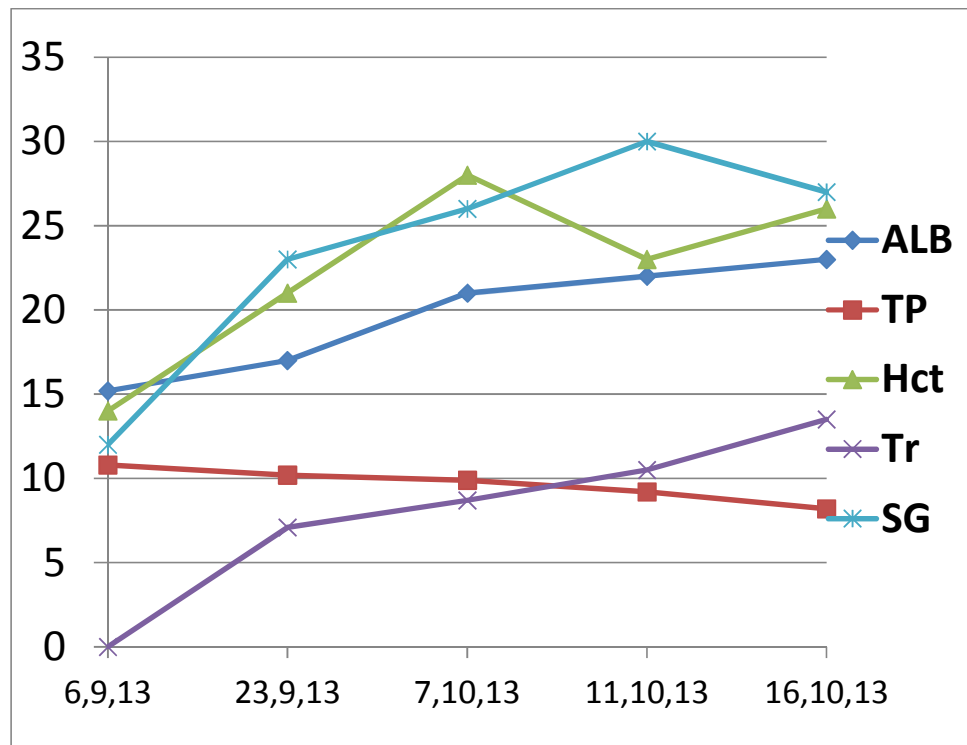
Case 1 – A surgery candidate?

- “Mecho” – **presentation**. Mix, intact male, 24 kg. Big mass over the left shoulder.



- “Mecho” - **evaluation** prior to surgery showed GN (*marked, persistent proteinuria*), anemia and thrombocytopenia caused by at least 3 reasons: Big tumor (cytology – probably benign), **Ehrlichia canis**, **Anaplasma phagocytophilum**. IMHA was ruled out with blood smears – no spherocytes or agglutinated RBCs were observed.
- “Mecho” **treatment before surgery**
 - Imidocarb dipropionate 6 mg/kg

- Doxycycline 10 mg/kg sid for 28 days
- Enalapril 0.5 mg/kg bid
- Spironolactone 0.5 mg/kg sid
- Aspirin 1mg/kg sid (¼ of 100mg tablet)
- Fish oil caps (omega 3) 1caps tid
- Nandrolone 1 mg/kg
- High quality diet
- **“Mecho” treatment at surgery**
 - Balanced anesthesia
 - Blood transfusion, iv fluids
 - Dopamine 5-10µg/kg/min iv as needed for maintaining MBP
 - Furosemide 2mg/kg iv
 - Mannitol 10g iv
 - Pain control
- **“Mecho” – progress over 2 months (06.09.2013 – 29.10.2013)**
 - Blood ALB in g/L 15.2 – 17 – 21 – 22 - 23
 - Blood TP in g/L 108 – 102 - 99 – 92 - 82
 - Hct in % 14 – 21 – 28 – 23 – 26
 - Tr x 10⁹/L 71 – 87 – 105 - 135
 - Urine SG 1.012 – 1.023 – 1.026 – 1.030 – 1.027



- **!!! Protein in urine (SSA) +++/+++ /+++ /+++ /+++**

Improvement in all parameters except protein in urine >>> persistent GN. Clinically, dog is doing excellent to this date.

▪ **Glomerulonephritis Case 1 – discussion “Mecho”**

- In a dog brought for a big tumor, there may be other reasons for disease.
- Vector borne diseases are quite common and should be suspected in any serious case.
- Anesthesia in dogs with GN is risky and may lead to irreversible kidney failure.
- GN may persist beside treatment and clinical improvement.

Glomerulonephritis

Case 2

- “Gatuzo” – Caucasus shepherd dog, intact male, 7 years old , 60 kg, 20.05.2012 **presented for:**
 - Dubious testing for dirofilariosis in another clinic. Results of HW antigen test (Vetall) had been negative, Mf in drop of blood positive.

- Not feeling well, prefers to lie down, anorexia, PU/PD
- **Evaluation.**
 - Positive results from 3 different HW Ag tests (Anigen, Heska, IDEXX), Knott D. immitis
 - Bloods - ALB 15.8 g/L, Leu $24.6 \times 10^9/L$
 - Urine - SG 1.010, protein SSA +++, rich sediment
 - Xray – only Rlat – normal
 - Ultrasound – normal abdomen, no visible worms in the heart.
- **“Gatuzo” Treatment 20.05.2012 – 29.08.2012**
 - Doxycycline 10 mg/kg sid for 28 days
 - Enalapril 0.5 mg/kg bid
 - Spironolactone 0.5 mg/kg sid
 - Aspirin 1mg/kg sid (¼ of 100mg tablet)
 - Selamectine spot on followed by ivermectin 50 µg/kg monthly
 - 29.08.2012 melarsomine 2.5 mg/kg deep im once

Did not show up for the next melarsomine treatment after 2months ...

...Instead, he showed up 1 year later 28.05.2013

- **“Gatuzo”** is feeling fine, owner wants recheck
 - HW Ag test (Heska) POSITIVE
 - Knott NEGATIVE
 - TP 75 g/L, ALB 30 g/L
 - Urinalysis : SG 1.032, SSA ++, sediment: a lot of fine granulated and hyaline casts.
 - Still on Enalapril 0.5 mg/kg bid and Spironolactone 0.5 mg/kg sid

GN is still there...and 4 months later 04.10.2013

- **“Gatuzo”,** again recheck
 - HW Ag test (IDEXX) NEGATIVE.
 - Knott NEGATIVE

- TP 70 g/L, ALB 28 g/L
- Urinalysis : SG 1.019, SSA ++, sediment: a lot of finely granulated casts

GN is still there... BUT...

A year later (July 2014) owner comes by and says Gatuso is fine, still on enalapril and spironolactone.

Glomerulonephritis

Case 2 – discussion “Gatuso”

- A 7 year old, giant breed dog like this Caucasus shepherd dog, will not live 2 more years with albumin 15 g/L, urine SG 1.010 and inability to walk.
- Even if owners are not fully compliant because of the cost, you can still have nice clinical results with cheap drugs that control GN.
- One dose of melarsomine and monthly prevention with ivermectine can obviously kill all parasites and negate the Ag test. This “slow kill” method is not suitable in cases like Gatuzo. Removal of the causative antigen as soon as possible may result in quicker resolution of GN.

Summary – glomerulonephritis in parasitic disease

- Definite treatment is not always possible, but you can control disease and provide quality of life to many patients. First line drugs – cheap and simple.
- If you don’t pay attention to kidney damage you will lose animals of kidney failure, despite treating dirofilariosis, ehrlichiosis, etc.
- Diagnosing IMGN can be done with simple tools (SG, SSA)
- Think about thromboembolism in a dog with dirofilariosis, versus thromboembolism in a dog with dirofilariosis AND immune mediated glomerulonephritis. Which will be worse? Which dog needs more attention?

Lesson 2: Routine testing for parasitic diseases with zoonotic potential.

- We as small animal veterinarians tend to underestimate our role as guardians of the public health
- In parasitology we often follow a routine of blind prescription of drugs. Not knowing the prevalence of parasites diminishes our motivation to insist on prevention. If we are not motivated, it is unlikely that we will motivate pet owners for thorough prevention.

- It is better to create a routine of testing, followed by adequate prescription.

Of course the aim of this lesson is not to cover all the parasitic diseases in small pet animals but to show some examples of parasites which are common in our everyday practice and a common health problem to people.

Fecal testing – How?

- Direct fecal test
- Fecal flotation
- Fecal flotation with concentration
- Baerman

Main mistake - we don't combine tests (direct + flotation is easiest)

Fecal testing – When?

- Puppies/kittens
- Animals with diarrhea, a serial testing may be necessary
- New clients
- Expected baby in the family
- Breeding animals
- Exotic animals
- Annually with vaccination, more frequently if owners afraid of drugs (q 3 months)

Our major mistake is that we don't do fecal testing as often as we should.

Fecal testing – common findings

- *Giardia intestinalis*. Can cause chronic debilitating diarrhea in humans. Strains found in dogs and cats rarely infect people (zoonotic potential with types A1 and B), but still there is risk. Other pets – exotic animals like rats, guinea pigs, chinchillas and ferrets can pose a risk. **Mistake – diagnosis missed if direct fecal test/Ag test not done.**
<http://www.capcvet.org/capc-recommendations/giardia>
- *Toxoplasma gondii*. Nasty disease in humans. Can cause abortions in pregnant women, but also brain and ocular damage to babies sometimes quite late in their life. **Mistake – diagnosis missed if flotation fecal test/Ab test not done.**
- *Isospora* spp



Small object – Toxoplasma oocyst, big one - sporulated Isospora oocyst

- *Campylobacter* spp Can cause severe diarrhea in humans. Accountable for 5-15% of diarrhea cases in humans. Watch video about health hazards: https://www.youtube.com/watch?v=V3QS_EgEYIk **Mistake – diagnosis missed if direct fecal test not done.**
- *Toxocara* spp. Main parasite causing Larva migrans syndrome in people. Responsible for several forms of disease heaviest of which are the ocular toxocariasis (OT), visceral larva migrans (VLM) and cerebral or neurological toxocariasis. Examples: “In a study carried out in Italy, on more than 700 patients who resulted negative for allergy tests but suffering of chronic recurrent respiratory, eye, skin or gastrointestinal symptoms, about 31% resulted positive for *T. canis* IgG antibodies. Among them 25% suffered of asthma. Two thirds of patients underwent subsequent antihelminthic therapy with a complete remission of symptoms. (Qualizza et al., 2011. Seroprevalence of IgG anti-Toxocara species antibodies in a population of patients with suspected allergy. *Int. J. Gen. Med.* 4:783-787.).”; “Beaver et al (1952) recognized the causative role of *Toxocara canis* larvae in cases of sustained eosinophilia (above 50%), pneumonitis, and hepatomegaly in children younger than 3 years old and dubbed the condition visceral larva migrans. As a horrible sequel occurring at 3 to 13 years, the larvae may produce granulomatous retinitis. Misdiagnosis of *Toxocara canis*-induced granulomatous retinitis as retinoblastoma has prompted the unnecessary enucleation of childrens’ eyes in at least 36 reported cases (Bowman, Dwight D., 2009. *Georgis’ parasitology for veterinarians.* p. 207).”

It is important to note toxocariasis in puppies. During the last trimester of pregnancy, arrested larvae are reactivated and migrate from the tissues of the bitch to the pups in utero (Fülleborn, 1921). This happens even in bitches with negative fecal flotation tests. Owing to transplacental transmission all pups may be assumed to be infected. Medication in puppies should start routinely as early as the second week of life and be repeated every 2 weeks until the pup is 3 months old (Bowman, Dwight D., 2009. *Georgis’ parasitology for veterinarians.* p. 203.).

Mistake – diagnosis missed if flotation fecal test is not done.

Mistake – puppies are not treated early enough and often enough against toxocariasis.

- Taenia spp. Eggs cannot be differentiated. False negative results more often than with other helminths. Very serious health hazard with echinococcosis!
- Dipylidium caninum. Proglottids may be visible in feces 2 – 3 weeks after ingestion of an infected flea. The fact sheets of products containing the combination of febantel, pyrantel and praziquantel state that they are safe to use in puppies over 2 weeks old. Deworming against tapeworms should start as early as 2-3 weeks, especially if flea control is not adequate. **Mistakes – we forget the connection between fleas and tapeworms. We deworm puppies/kittens only against roundworms.**
- Ancylostoma and Uncinaria spp. Risk for cutaneous larva migrans in people. **Common mistake - we forget to advice that environment is contaminated. The same is valid for Toxocara spp eggs.**

Prevention mistakes

- **What should we recommend for prevention?**
 - Early start in life for roundworms, tapeworms, heartworm, fleas
 - Adequate frequency . HW – 30 days. If more than 45 days – infection. July 1-st - August 20-th IS NOT SAFE. Intestinal worms – based on fecal testing. Bulgaria – general deworming in adult dogs and cats - once a month.
 - Proper products – for example products with repellent action CANNOT guarantee protection against HW infection. Oral suspensions for puppies DO NOT kill tapeworms.

Routine testing – the point of view of the practice manager

- successful business ≠ best possible care, but the best possible care cannot happen without successful business
 - Fecal testing – inexpensive method, very good medical results, high profit margin
 - Ethical point – routine fecal testing is fully justified because it is an important tool against parasitic zoonoses
 - The same is true with serological testing for HW, ehrlichiosis, anaplasmosis, brucellosis, giardiasis and others
 - ***Adequate frequency of testing and treating with the proper products is not only good prevention and good veterinary standard, it is good business, which helps a practice and its members grow professionally and serve society in the best possible way.***

References, resources:

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