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any pathological findings, only laryngeal palpation was abnormal. Resting airway endoscopy revealed right arytenoid paresis, rostral palatopharyngeal arch displacement and feed aspiration in rostral part of trachea. The high-speed treadmill video endoscopy (trot (speed 4 m/s) and continued in gallop) of upper airways revealed loss of right sided arytenoid cartilage abduction, and axial deviation of the right vocal cord. During the examination inspiratory stridor was audible. X rays of the larynx showed hypoplasia of laryngeal cartilages (*cartilago thyroidea* and *cartilago cricoidea*) and presence of air in the rostral part of oesophagus. To enhance the efficacy ventilation laser ventriculocordectomy was chosen as a treatment. The intervention was performed in sedation (0,012 mg/kg IV detomidin/ 0,025 mg/kg IV butorphanol) and local anaesthesia of nasal, pharyngeal and laryngeal mucosa (2% lidocain). Before right laser cordectomy and ventriculotomy tracheostomy was performed. Cordectomy was made by two cuts. First cut was performed with endoscope inserted through left nostril. Before second cut nostrils were changed, the endoscope was inserted through right nostril and the grasping forceps was inserted through the left nostril into nasopharynx by the assistant. The vocal ligament was grasped under visual control and second cut was performed. Ventriculotomy was performed consecutively after grasping Morgan ventricle by forceps. We used a 600 µm bare-fibre (25 W). The post-surgical treatment consisted of dexametasona (0,1mg/kg PO) for three days, flunixin-meglumine (1.1 mg/kg IV; PO), sulfadiazin, trimetoprim (25 mg/kg IV; PO) administration for 14 days and rest for one month.

**Results and Conclusion:** The follow up examinations were performed three times during the two weeks long hospitalization after surgery. Control endoscopy revealed a gradual reduction of *rima glottides* edema. After the discharge to home care, owner stated that the mare has no problems during exercise, no stridor is audible. The horse swallowed feed without any problems.

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## P52

### Canine streptococcal mastitis with skin lesion-case report

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**Introduction:** *Streptococcus canis* is opportunistic pathogen that is zoonotic in animals and humans and cause to necrotizing fasciitis which is rapidly progressing, leading to multiple organ failure.

**Material and methods:** A six year old Kurzhaar which is suffering from physical wounds and deep skin lesions on udders is brought to Mehmet Akif Ersoy University Faculty of Veterinary Medicine- Obstetrics and Gynecology Clinic. In physical examination, necrotic wounds and fragility in the epithelial tissue from abdominal and inguinal region of the dog were observed. On the different location of body of the dog, especially at the decubitus location, ulcer like wounds and inflammation were seen. Also milk was stinking and mucoid. The body temperature was 40°C, breathing rate was 29/min and pulsation was 114/min. The swab samples taken from wounds and the milk samples of the dog were sent to Department of the Microbiology. The samples were spread onto 10 % sheep blood agar. Antibiotic susceptibilities of the bacteria isolated were determined by a disc diffusion method on a Mueller Hinton agar plate. The dog which died on the fourth day of hospitalization was sent to Pathology Laboratory of Veterinary Faculty for necropsy.

**Results:** After incubation of the samples, pure culture of *Streptococcus canis* was recovered and identified on the basis of cultural, morphological and biochemical characteristics. *S. canis* was found to be susceptible to amoxicillin, amoxicillin+ clavulanic acid, ampicillin, penicillin, cefoperazone and florfenicol; and to be resistant to trimethoprim+sulphamethoxazole, danofloxacin, enrofloxacin, gentamicin, erythromycin, lincomycin, ciprofloxacin, cefquinome, ceftiofur, and rifamycin. Severe ulceration areas, necrosis and granulation tissue proliferations were observed during the histopathological examination of udder tissue. In the histopathological examination of other tissues, lung edema, neutrophil leukocyte infiltration in heart, bile stasis in liver, hepatocyte degeneration, and mononuclear cell infiltration in kidneys were observed. Patient was diagnosed with necrotic mastitis, septicemia and toxemia considering histopathological lesions.

**Conclusion:** *S. canis* is associated with necrosis, septicemia and toxemia that cause pathologic differentiations in organs and even death.

## P53

### Chronic ehrlichiosis canis in Miniature Pinscher - clinical case

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**Introduction:** Ehrlichia canis is an obligatory intracellular pathogen causing canine ehrlichiosis, potentially fatal disease. Rapid and accurate diagnosis

leads to favorable prognosis. Three stages characterize the disease. The first, acute stage (8–20 days following transmission by infected tick) lasts 2–4 weeks manifested by fever, depression, dyspnoea, anorexia, laboratory findings: thrombocytopenia, leucopenia, mild anaemia. The second stage is subclinical, lasts 40–120 days or even years, in which the patient remains persistently infected without clinical signs except mild thrombocytopenia. The chronic stage is characterized by haemorrhages, epistaxis and edema; the results of laboratory study resemble the first phase of the disease. The course of this phase may often be complicated by superinfections by other microorganisms which can lead to bone marrow hypoplasia with negative prognosis. Dogs infected with *E. canis* remain infected even after antibiotic treatment.

**Material and methods:** Two years old female Miniature pinscher was admitted in the University veterinary hospital in Skopje for emergency cesarean section. Breathing difficulties, nausea and vomiting followed by ocular and nasal discharge, weakness, head ticks and ascites were observed few days after the surgery. Clinical examination revealed low body temperature, tachycardia and tachypnea. Laboratory findings showed severe anemia, thrombocytopenia and renal failure (urea 71.5 mmol/l and creatinine 607  $\mu$ mol/l). Antigen rapid *E. canis* test was positive and patient was treated for the renal failure with intense fluid therapy and vitamins (B12, Vit C) and doxycycline (5mg/kg BID p.o., 3 weeks). Control CBC and serum urea and creatinine were performed one and two weeks after the initial treatment. Two weeks after the treatment, the patient was improving and was discharged with doxycycline treatment for one more week with advices for special low protein diet due to the impaired kidney function.

**Results and Conclusion:** One year later, the patient was admitted again in the hospital with the similar clinical signs as previous (anorexia, nausea and vomiting, bad mouth odor). The owner did not follow diet recommendations. CBC revealed anemia (RBC  $2.821 \times 10^{12}/l$ , PCV 17.6%, Hb 7.8g/dl), serum urea and creatinine were increased (51.1mmol/l and 537.0  $\mu$ mol/l, respectively). Despite the aggressive treatment, one month later, the patient did not respond and human euthanasia was performed. Pathohistology findings showed severe organ damage: hemosiderosis hepatis, nephritis interstitialis gravis partum necroticans, pneumonia interstitialis et oedema, lymphopenia et hemosiderosis gravis lienis. Dogs in the chronic stage of the disease have guarded prognosis due to the multiple organ failure. Prompt and accurate diagnose followed by adequate therapy are crucial for long term prognosis. Recurrence can occur months to years after primary infection. The general conclusion is that relapsing renal failure, despite intensive symptomatic treatment, usually results with "infaust" prognosis.

## P54

### Presence of *Dirofilaria repens* at dogs in Belgrade area

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**Introduction:** *Dirofilaria repens* is mosquito-borne zoonotic filaria. It is present in several regions of the world, including western Balkan, southern Europe, Africa and southern Asia. Parasites live in the subcutaneous tissue of domestic and wild carnivores (dogs, cats, foxes, wolves etc.) and they are the reservoir for these parasites. *D. repens* also causes a zoonotic disease called human dirofilariosis. Humans are accidental "dead end" hosts in which the life cycle is not completed. Many European countries are considered enzootic for this type of zoonosis. In Serbia first occurrence of human ocular dirofilariosis are established 1995. and after that increasing number of human infections are reported.

*D. repens* are smaller than *D. immitis*. The parasite has a complex life cycle, and mosquitoes from the genera *Aedes* and *Culex* serving as intermediate hosts. Adults reside in the subcutaneous connective tissues where females produce larvae (microfilaria) in the natural host organism and release them into the circulation. Like those of *D. immitis*, *D. repens* microfilariae reside in the blood stream. Adult filaria in dogs is usually found during castration and surgical intervention in the abdominal area, in the connective tissue, by subcutaneous tissue and at the omentum. Infections are usually passed asymptomatic. Only in a one case are observed skin changes and lesions.

**Material and methods:** Determination of adult filaria performed by histology studying the morphology after their removal and identified by their thick laminated cuticle, broad lateral ends and large muscle cells. Microfilaria which established using modified Knott test are determined by morphological characteristic (measuring between 350 and 385  $\mu$ m in length and 7 to 8  $\mu$ m in diameter, with a curved tail and rounded cephalic extremity). Our result we confirmed by IDEXX 4DX test.

**Results and Conclusion:** In Belgrade area in period 1996-2005 *D. repens* is found at 5.3% and in period 2006-2009 at 19.2%. The latest research determined the increase of infections to 27.6%. This rapid spread of