DRIED SOLIDIFIED BLOOD CALCULI:

Dried solidified blood calculi (DSBC) are a relatively rare stone that can be found in the urinary tract of cats. They have been documented as increasing in frequency over the previous decade. DSBC are composed of 100% dried, solidified blood and contain no crystalline or mineral material. They can be distinguished from gelatinous blood clots, as they are solid, stone-like concretions that can be cracked with Rongeur forceps into multiple smaller pieces.

A 2006 retrospective study from UC Davis Stone Analysis Laboratory reported 49 samples from cats with DSBC between 1986 and 2003. Interestingly, almost half of the cases were submitted after 2001, consistent with the observance that these calculi are on the rise. Further studies out of the UC Davis lab found the numbers continued to increase into 2004. The distribution of stones was approximately 50% from the upper and 50% from the lower urinary tract. The stones were more commonly submitted from male cats, but this is likely due to their greater propensity to present for urinary obstructions.

In the 12/49 cases that were submitted from within the UC Davis teaching hospital, 50% presented for ureteral obstruction. All cats had marked hematuria with > 100 RBC/hpf on urinalysis. A unique feature of DSBC is that they are not easily imaged and can be missed on plain radiographs, ultrasound, computed tomography, and or with contrast studies. Often signs consistent with a ureteral or renal pelvic obstruction can be identified such as pyelectasia and ureteral dilation, but no clear cause for these changes are seen complicating the diagnosis. Even contrast studies might suggest obstruction but rarely identify calculi. The etiology of these stones is unclear, but marked hematuria is a common feature suggesting that these might develop secondary to renal hematuria. Anecdotally, these stones have been noted to form more commonly in cats receiving fluoroquinolone antibiotics.

DSBC are inherently difficult to diagnose, but we will likely continue to see more cases of these stones as more veterinarians become familiar with their intricacies.

NEUROGENIC KCS AND XEROMYCTERIA:
Keratoconjunctivitis sicca (KCS) most commonly presents in dogs with bilateral mucoid ocular discharge and conjunctival hyperemia. The etiology is thought to be immune-mediated in origin, though certain breeds predispositions exist. In contrast, neurogenic KCS is generally unilateral and seen in conjunction with xeromycteria (dry nose). This can present as unilateral ocular disease, unilateral nasal disease, or both. Efferent innervation to the lacrimal gland is via the parasympathetic fibers of CN VII (facial nerve). The same nerve fibers also innervate the lateral nasal gland which is responsible for humification of the nasal cavity. Most cases are idiopathic in origin, however given the path of CN VII, otitis media/interna and trauma to petrous temporal bone have also been implicated.

The majority of dogs with this disease are middled aged, but no clear breed or gender predilections exist. Schirmer tear testing in the affected eye is markedly reduced or absent. The quintessential nasal associated clinical sign is an ipsilateral dry, crusty, and frequently occluded nares. Generally, no other neurologic or ophthalmologic abnormalities are found.

Therapy for classic KCS (Cyclosporine, artificial tears) is often attempted with limited s. Pilocarpine 1-2% ophthalmic drops given orally on food can be utilized as a parasympathomimetic agent. Initially, one drop per 20# of body weight is given twice daily. The dose is gradually increased by one drop weekly until clinical signs resolve or signs of systemic toxicity occur. These signs include hypersalivation, vomiting, or diarrhea. Signs can take weeks to months to resolve if ever. In addition to artificial tears, vaseline can be applied to the affected nares for moisture and lubrication.

Dogs are frequently referred to an internal medicine specialist for a nasal work-up based on the dry and crusty nasal discharge. Recognition of this disorder can spare clients and patient from expensive and sometimes invasive work ups for more traditional nasal disorders.

**SYSTEMIC EFFECTS OF CUTEREBRIASIS:**

*Cuterebra spp* are non-biting, oviparous, bumblebee-like flies. Over 34 species have been identified in North America. Natural hosts are rabbits and rodents. Female *Cuterebra* flies lay their eggs around entrances to burrows or nest of small animals. Eggs hatch in response to sudden increases in temperature. Hatched first-instar larvae moved onto the fur of passing hosts. Larva on hosts travel to a preferred site of penetration such as the mouth, nares, eyes, wounds, or anal mucosa. In a natural host, the first-instar larva penetrates the nasopharyngeal or tracheal mucosa and migrates through pleural and peritoneal spaces to the subcutaneous tissues. The larva forms a “warble”, a local reaction to the parasite that appears as a firm swelling in the skin with an orifice at its apex. After full migration, the third-instar larva emerges from the skin and drops to the
ground to pupate in the soil. Adult flies emerge several months to years later. In atypical hosts (dogs, cats, humans) the *Cuterebra* larvae undergo aberrant migration. This manifests as either cutaneous myiasis or signs referable to migration though the respiratory tract or nervous system. Diagnosis is typically made by visualization of the larva or warble, endoscopy of respiratory airways, or advance imaging of the CNS via MRI or CT scan. Treatment involves removal of the larva if possible. If the larva is not reachable as in CNS migration, treatment with ivermectin, glucocorticoids, and diphenhydramine is often successful.

Recently, reports of systemic illness associated with *Cuterebra* have been described. Marked proteinuria is a consistent finding and is thought to be associated with immune complex disease secondary to *Cuterebra* antigen. Liver enzyme elevation has also been seen and may be associated with parasitic migration, systemic inflammation, a *Cuterebra* toxin, or ischemic disease. A shared feature of these cases is that they often begin with mild respiratory signs (sneezing or coughing). This is not surprising given that the respiratory tract is a common location for *Cuterebra* to enter the body. Suspicion of and treatment via removal of the larva or systemic therapy has led to rapid resolution of the systemic effects of *Cuterebra* in the few cases that have been described. Specifically, a combination of respiratory signs and marked proteinuria should raise consideration of systemic cuterebriasis.

**SELECT REFERENCES:**

