

A Case of Feline Skin Fragility

The majority of diseases resulting in feline skin fragility are acquired, but the pathogenesis of this rare syndrome is unknown. It is most commonly associated with such conditions as iatrogenic or naturally occurring hypercortisolism, diabetes mellitus, or extensive use of progestational compounds. Spontaneous hypercortisolism is responsible for 50% of cases. Iatrogenic hypercortisolism can cause skin fragility but only after prolonged exposure to topical/systemic steroids. In all cases, histology shows a thin epidermis and severely atrophic dermis. Dermal collagen fibers are severely attenuated and are pale-staining with a wavy appearance. The 4-year-old, spayed domestic shorthaired cat in this study presented with severe weight loss, anorexia, and occasional diarrhea. A 5 x 5 cm tear that occurred during transport to the veterinary hospital was present in the dorsal cervical region. Complete blood count showed an inflammatory leukogram with neutrophilia, increased segmented neutrophils, and lymphopenia. The cat was anemic and mildly thrombocytopenic. A diffusely enlarged and hypoechoic liver and spleen as well as a diffusely hypoechoic pancreas with hyperechoic peripancreatic fat were apparent with ultrasonography. The cat was euthanized due to its poor prognosis and clinical condition. Histoplasma organisms were identified within the dermis, liver, spleen, pancreas, lung, and bone marrow. Proposed mechanisms for the Histoplasma-associated skin fragility included the release of inflammatory mediators by infected and activated tissue macrophages or disturbed cutaneous blood flow secondary to severe vasculitis. Although this cat did not test positive for feline immunodeficiency virus or feline leukemia virus, an underlying immunodeficiency could not be ruled out.

Commentary: This interesting case report provides an overview of cutaneous fragility syndrome in the cat and then attempts to provide a mechanism for the appearance of the syndrome in this particular case. Although the authors downplay the significance of the previously administered prednisone, I find it quite possible that this, along with cachexia secondary to systemic fungal disease, could have contributed to this cat’s skin fragility. It is worth noting that the condition was not reported prior to administration of prednisone, nor did this cat have the typical inflammatory granulomatous lesions associated with cutaneous histoplasmosis. While cause and effect cannot be proven, I feel that the prednisone treatment might be considered as a contributing factor. —Michael Schae, DVM, Diplomate ACVIM & ACVECC


Reversible Feline Pulmonary Hypertension?

Pulmonary hypertension (PH), a rare condition in cats, has been associated with heartworm infection, thromboembolic disorders, atypical inflammatory polyps, and reversed patent ductus arteriosus. The cat in this report was a 13-year-old female with a history of subcutaneous fibrosarcoma that became acutely dyspneic following concurrent administration of radiotherapy and intravenous carboplatin. Right ventricular dysfunction and congestive heart failure associated with precapillary PH were diagnosed. Pulmonary thromboembolism (PTE) with associated disseminated intravascular coagulation (DIC) was considered to be the most likely underlying disorder. The cat was aggressively treated for the next 15 days with oxygen, benazepril, furosemide, low-dose aspirin, unfractionated heparin, and intravenous fluid therapy. On day 25, the cat was found to be in good health and echocardiography was unremarkable. Possible risk factors for PTE in this cat were neoplasia, use of an indwelling catheter for administration of carboplatin, and DIC. To the authors’ knowledge, this is the first known report of reversible PH, likely secondary to PTE, in a cat.

Commentary: The diagnosis of PTE can be very frustrating as the patients often present in severe respiratory distress without diagnostic radiographic findings. This case report is a good example of how echocardiographic findings can support a diagnosis of PTE. If tricuspid regurgitation is found, the presence and severity of PH can be determined. In the absence of tricuspid regurgitation, other findings (changes in the pulmonic outflow velocity waveforms, septal flattening, right-sided heart enlargement) may be suggestive of PH, which could help support a diagnosis of PTE. As illustrated in this report, serial echocardiography can be helpful to monitor response to therapy and resolution of PH.

—Elizabeth Cole, DVM, Diplomate ACVIM