FIC, FLUTD, FUS, Pick Your Acronym Anthony Carr, DVM, DACVIM (Internal Medicine) University of Saskatchewan Saskatoon, Saskatchewan, Canada

Feline interstitial cystitis, or feline lower urinary tract disease, can be a very frustrating disease to treat. The rate of recurrence is variable and can be quite high. Few if any effective prophylactic or for that matter effective acute treatments are available. A link to other systemic disorders has been established in humans. It has been found that people with interstitial cystitis more frequently have Crohn's disease, allergies, irritable bowel syndrome, fibromyalgia, systemic lupus erythematosus, Sjogren's syndrome as well as other immune mediated diseases. The link to allergies is especially intriguing since mast cell degranulation is involved both with allergic signs and symptoms of interstitial cystitis.

Some of the abnormalities involved with FIC have been documented previously. There are indications that episodes may be precipitated in cats by stressors, much as can occur in people. This suggests that the sympathetic nervous system may be involved. Increased plasma norepinephrine concentrations have been found in these cats. This would be expected if the sympathetic nervous system were activated. The hypothalamic-pituitary-adrenal (HPA) axis was also tested in these cats by administering corticotropin releasing hormone (CRH) and was found to be normal. Further investigation of HPA axis showed however that responses were different in cats with FIC. The effects of stress on plasma catecholamine concentrations and urine cortisol excretion in 12 normal cats and 12 cats with FIC was investigated. All plasma catecholamine concentrations (dopamine, norepinephrine, epinephrine and various metabolites thereof) were elevated in FIC cats when compared to normal cats going along with an increased response to stress. Surprisingly, however, urine cortisol to urine creatinine ratio was not different in these two groups. Under normal circumstances increased cortisol excretion should occur with stress. This would suggest that the HPA axis is uncoupled in these cats from sympathetic nervous system activation.

There is evidence to support that adrenal glands and adrenal gland function in cats with FIC are different than in normal cats. The adrenal glands of 13 cats with FIC that were necropsied were found to be smaller than the adrenal glands of normal cats. The medulla was of normal size, however the cortex was smaller. This publication also included data on ACTH stimulation testing 20 cats with FIC, five of whom were later necropsied for the adrenal gland comparison. The response to ACTH was significantly lower in cats with FIC than normal cats, though this adrenal insufficiency was very mild. The response to ACTH and adrenal gland size did not correlate.

Many other studies have been carried out to look at stress and response to stress in patients with interstitial cystitis (both human and feline). There is evidence to support that the stress response is dysregulated. This dysregulation may partially be responsible for some of the clinical signs seen. Of course it cannot be discounted that chronic clinical disease can lead to dysregulation of the stress response as well, leading to the classic chicken and egg conundrum. It is hoped that by understanding these processes better, more effective therapies might be designed to help with this and similar debilitating diseases. There certainly is a need for this, since our current therapeutic armamentarium is woefully inadequate to deal with this problem.

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