The cat vets review of recent feline literature

Alison Jukes BVSc (hons) FANZCVS (Feline Medicine)

Cat Specialist Services, Underwood, drjukes@catspecialists.com.au

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1. JAVMA 2025: Mark Peterson, Staphanie Carmody, Mark Rishniw Hyperthyroid cats that develop azotemia following successful radioiodine treatment have shorter survival times compared to cats that remain nonazotemic.[1]

Background: Previous dogma has been that non azotemic hyperthyroid cats treated with I131 have the same survival time whether they become azotemic post treatment or not. Chow and White[2] found increasing creatinine (CRE) after I131 was negatively associated with survival after adjusting for age.

Methods: 1047 hyperthyroid cats treated with I131 between 2013 – 2020 and were euthyroid post treatment (normal thyroxine and thyroid stimulating hormone (TSH)). These cats were part of a larger cohort of 1400 cats treated with I131. Patients that were excluded were persistent hyperthyroid (56 cats) and hypothyroid post I131 (297 cats). Azotemia or not status was identified at 6 - 12 months post I131 and then patients monitored every 6-12 months until death or end of study period (Jan 2024). Azotemia was defined as CRE >2.0mg/dl (176 umol/L) and urine specific gravity (USG) <1.035.

Results: 128/1047 cats developed azotemia 6-12 months post I131, azotemic cats had shorter median survival time (MST)than non azotemic cats (2.8 vs 4.3 years). Forty-nine percent of cats had previous treatment with methimazole. Cats that became azotemic on methimazole trial were excluded.

There was no difference between groups in regard to prior methimazole use or period of time between diagnosis of hyperthyroidism to I131 treatment. Preazotaemic cats were older, lighter, with lower body condition score and muscle condition score, higher CRE and lower USG. Additionally these cats were verging on lower TT4 (P=0.0581) and higher TSH (P=0.052). Preazotemic cats more likely to die of renal disease (70%) compared to non azotemic cats (18%). Increasing age and renal status were independently associated with survival time (multivariable cox regression), renal status after adjusted for age and sex still had impact on decreasing MST.

Strengths: Large study that has demonstrated what we all thought with regards to azotemia post I131.

Limitations: Peterson's individual dosing regime for I131 that requires scintigraphy to calculate dose and potential dosing over 2 days

Take homes: assess CRE and USG pre treatment, high end normal CRE and low USG increases odds of patient developing azotemia post I131 treatment and this can impact on MST.

2. RAAS gone wrong: Case reports

Journal of Feline Medicine and Surgery Open Reports 2024: Long- lasting hypoaldosteronism after adrenalectomy in a cat with hyperaldosteronism

Bouccara L, Dunie A, Poujol L, Blond L, Jolivet F[3]

Summary: 10yo MN DSH presented with history of poor appetite, vomiting, polyuria and polydipsia. The patient was diagnosed with hypertension, azotemia, hypokalaemia and an abdominal mass adjacent to the right kidney. Severe elevation of aldosterone was documented and a diagnosis of primary hyperaldosteronism was made. Medical stabilisation with oral potassium, spironolactone, amlodipine was performed for 1 month prior to laparotomy and adrenalectomy with nephrectomy. Histopathology of the adrenal mass revealed a cortico-adrenal carcinoma. At the time of surgery, the patients azotemia had resolved. Four days post-operatively, the patients CRE increased (180umol/L) with normal blood pressure and electrolytes. These parameters were stable at day 25 post operation. At 70 days post operation, the patient had an increase in vomiting with hyperkalaemia, low Na:K ratio and CRE 358umo/L. ACTH stimulation test was normal however serum aldosterone was <20pmol/L. The patient was treated with 1.5mg/kg SC deoxycortisone pivalate q28 days with prednisolone 0.28mg/kg PO q24h. These medicines were continued for 6 months post surgery while patient remained well with normal electrolytes however it was persistently moderately azotemic .

Take home: Transient hypoaldosteronism is reported in 16% humans post adrenalectomy but only 5% required specific treatment. Presumably this is secondary to suppression of the contralateral zona glomerulosa by excess aldosterone from the neoplastic adrenal gland. Monitoring of electrolytes and biochemistry are important in short and medium term post operative follow up of cats post adrenalectomy for hyperaldosteronism.

JVIM 2024; Pseudohypoaldosteronism and acquired renal aldosterone resistance with hyperkalemic type IV renal tubular acidosis in 2 cats.

Christina L Marino and Jonathon D Foster [4]

Pseudohypoaldosteronism is a condition where resistance to the action of aldosterone at the level of the aldosterone receptor in the renal tubule results in renal tubular acidosis and hyperkalaemia. Biochemically patients will have elevated plasma renin and aldosterone, and a mild nonanion gap metabolic acidosis. This may occur as a primary condition due to mutations of the mineralocorticoid receptor, or secondary to disease within the renal tubule including urinary tract infection, chronic kidney disease, medications that block the renal epithelial sodium channel or drugs that antagonise aldosterone at the level of the receptor including spironolactone or progestagins.[5]

This case report documents two cats in which acquired pseudohypoaldosteronism was documented. The first case occurred in a 7yo FS DSH which had a cystotomy for cystolithasis that 1 month post surgery had an acute kidney injury (AKI). The AKI episode was urine culture negative and the patient recovered to stage 2 IRIS renal disease after supportive care. Three and a half months later the patient developed progressive hyperkalaemia, hyponatraemia and metabolic acidosis with stable azotemia. The patient was asymptomatic. ACTH stimulation testing was performed revealing normal cortisol but marked elevation of aldosterone (4588 pmol/L, RI 277-721pmol/L). Therapy with

frusemide was initiated (1.2mg/kg q12h) and normalisation of electrolytes was demonstrated one month later and the patients azotemia improved (IRIS stage 1).

The second case was a 4yo MN DSH that presented for urethral obstruction and AKI (IRIS grade 5). The patient required three urinary catheters over the course of its hospitalisation and was urine culture negative. The cat was not azotemic at discharge however represented 6 days later with lethargy, pollakiuria, dehydration without urethral obstruction. Further evaluation revealed urinary traction infection, azotemia (mild), hyperkalaemia and hyponatraemia. Despite supportive care with IV fluids and antibiotics, the patient remained hyperkalemic and acidotic. Cortisol and aldosterone was measured at this time however the patient was discharged prior to results without any hormone supplementation. When electrolytes were rechecked 1 day after discharge, the acidosis and hyperkalemia had resolved. Cortisol was found to be normal however serum aldosterone was profoundly elevated at 5548pmol/L (RI 194-388 pmol/L).

These cases demonstrate evidence of acquired aldosterone resistance in the renal tubule resulting in metabolic a nonanion gap acidosis and hyperkalaemia either secondary to chronic interstitial nephritis (suspected) or urinary tract infection post complicated urethral obstruction. Limitations of these cases include the lack of follow up aldosterone measurement and blood gas analysis.

3. Veterinary Sciences 2025: Emergency Dyspnoea in 258 Cats: Insights from the French RAPID CAT study.

Abboud N, Deschamps JY, Joubert M, Roux F.[6]

Background: The original RAPID cat study[7] was published in 2018 out of the United Kingdom and investigated the underlying causes of 108 cats presenting to primary care practices with emergency dyspnoea. The goal of this study was to investigate historical and clinical examination findings that help to differentiate cardiac and non cardiac causes of acute dypsnoea. This study found 65% of cats presented with underlying cardiac causes, 16% had primary respiratory disease, 11% neoplastic disease and 8% were trauma related. Nine percent of cats did not have a definitive diagnosis. Cardiac cats were significantly more likely to have severe tachycardia (HR >200bpm), severe tachypnoea (RR >80bpm) with a gallop sound and low body temperature (<37.5 degrees). The French authors of the current study felt that the population of cats seen at their institution (a French veterinary teaching hospital in a metropolitan location) did not reflect this distribution and so retrospectively analysed cases of emergency dyspnoea presenting to their hospital.

Methods: Retrospective analysis of 312 cats presenting for severe dyspnoea (requiring O2 therapy) over a 5 year period (Jan 2018- Dec 2022) to the emergency department of the Oniris VetAgroBio, School of Veterinary Medicine, France. Initial management of the dyspnoeic cats included sedation and O2 therapy to stabilise then a combination of rapid diagnostic tests including thoracic and cardiac POCUS, radiograph, thoracocentesis. Definitive diagnosis was obtained in 258 (83%) of patients.

Results: Of the patients with a diagnosis, thirty-three percent of cats had respiratory disease, 25% had cardiac disease, 21% had underlying trauma and 21% had an underlying neoplasm. 36% of cats had pleural effusion on presentation, males were over-represented in cardiac and respiratory causes of dypsnoea. Cats with traumatic causes were significantly younger (median age 2 years), cats with cardiac and neoplastic causes were significantly older (median age 11 years), cats with respiratory disease had median age of 6 years. Hypothermia was significantly more common in the cardiac cats compared to the other groups and cats with respiratory causes were significantly more likely to be

hyperthermic (this encompassed infectious causes including pyothorax and FIP). In contrast to the UK RAPID CAT study, neither tachycardia or bradycardia was associated with any one group of dyspnoeic cats. Cats with a gallop rhythm were only identified in the cardiac group however this was only 12% of these cats. Pyothorax was identified in 10% of cats. Only 56% of cats survived to discharge (of 44% not surviving 28% died in hospital and 72% were euthanased).

Strengths: this study is an analysis of the largest group of dyspnoeic cats with an effort to find history or clinical examination findings to assist in the diagnosis of an underlying cause of dyspnoea.

Limitations: Retrospective, lacking control group to be able to effectively analyse breed or lifestyle variables that may contribute to dyspnoea in pet cats. Cats not admitted to the hospital were excluded from analysis. Still not a very large study.

Take home: Hypothermia and gallop rhythm are more likely to be associated with cardiac causes of dyspnoea. Cardiac and neoplastic causes of dyspnoea are much more common in older cats than trauma or primary respiratory disease. Local disease prevalence matters – Australia is likely not the UK or France.

4. JFMS 2025: Thoracic radiographic findings in cats with feline infectious peritonitis
Repyak K, Atiee G, Cook A, Bryan L, Gremillion C[8]

Background: Feline infectious peritonitis (FIP) is a common disease in young cats (<2yo) however it can be diagnosed at any age. Cats will typically present with a history of waxing and waning symptoms with multiple body systems involved. Definitive diagnosis of FIP using immunohistochemistry can be invasive and costly so often a presumptive diagnosis will be made based on clinical signs, classical changes on laboratory testing including haematology, serum biochemistry, cytology and fluid analysis +/- coronavirus PCR in conjunction with the lack of an alternative disease diagnosis which explains the clinical presentation. Some cats with wet form may present with pleural effusion and respiratory signs however other thoracic variants of FIP have been described including pleuropneumonia and pyogranulomatous pneumonia. The aim of this study was to investigate thoracic radiographic changes in general in cats with a diagnosis of FIP.

Methods: Retrospective study of 35 cats presenting between 2007-2022 to Texas A&M Veterinary Medical Teaching Hospital with a definitive or presumptive diagnosis of FIP and orthogonal thoracic radiographs performed within 7 days of presentation.

Findings: 91% of cats had abnormal thoracic radiographs of which 27% had pleural effusion. Pulmonary parenchymal changes were abnormal in 25 (71%) cats. 21/25 cats had an unstructured interstitial pattern, 11/25 bronchial pattern and 10/25 had alveolar pattern. Three cats had pulmonary nodules and 18 cats had a mixed pulmonary parenchymal patterns. Most pulmonary patterns had a diffuse distribution. Sternal lymphadenopathy was present in 45% of cats, one cat had a mediastinal lymphadenopathy, no tracheobronchial lymphadenopathy was noted. 17% of cats had cardiomegaly (one of these cats had tamponade and an underlying cardiomyopathy and another high output state due to anaemia). Pulmonary oedema was the most common histologic finding in 16/17 cats with histopathology followed by vasculitis and pleuritis. Cats with mild histopathologic findings of perivascular oedema had normal pulmonary parenchyma on radiography while other cats with normal histopathology had mild radiographic changes. Moderate to severe histopathologic change correlated with moderate to severe pulmonary changes on radiography. Five patients were diagnosed with myocarditis on necropsy – three of these had radiographic evidence of cardiomegaly.

Limitations; Cats with diagnosis of FIP but no thoracic radiographs were excluded (bias population to cats with respiratory signs or more severe disease) thereby potentially over-estimating the amount of thoracic disease present in cats with FIP. Discussion about why this cohort of cats had radiography performed was not discussed. Confirmed diagnosis of FIP was not achieved in all cats.

Take home: thoracic radiographic changes are common in cats with FIP though changes are non specific, lymphadenopathy (sternal) additionally is common (likely due to sternal lymph nodes draining abdominal structures). Myocarditis may be cause for cardiomegaly in a cat presenting with FIP.

5. **Veterinary Microbiology 2025; Feline coronavirus-associated uveitis: The eye as a gateway to systemic spread and feline infectious peritonitis?**
   Martinez A, Lavergne E, Brauge C et al[9]

Background: Uveitis is a common clinical sign of FIP. FIP virus is more likely than enteric coronavirus to have mutations in the spike gene (M1058L S1060A) which are thought to be involved in the change of tropism from enterocytes to monocytes/macrophages.

Methods: Retrospective analysis of aqueous humour collected from 193 cats that was RT-positive for FCoV between 2013 and 2023. Cats were divided into two groups based on clinical signs – suspected FIP or uveitis alone. A subset of cats which had viral loads high enough (36 uveitis alone, 41 suspected Fip) had Sanger sequencing performed to assess if mutations M1058L or S1060A were present.
Results: There was no difference in age between uveitis alone or suspected FIP cats. Cats with both uveitis alone and uveitis in conjunction with systemic clinical signs compatible with FIP exhibited M1058L or S1060A mutations though cats with suspect FIP had a higher odds ratio (OR) of these changes than cats with uveitis alone. Cats with suspect FIP had borderline increased viral load (P=0.06) than cats with uveitis alone.

Strengths: Large study which is attempting to answer questions around the relationship between uveitis, feline coronavirus and FIP.

Limitations: Retrospective. Definitive diagnosis of cats suspected with FIP was not proven. Follow up of cats with FCoV positive aqueous humor was not performed (did some or none of these cats go onto develop FIP? (Authors report five uveitis only cats that they had follow up for went on to develop serious systemic disease resulting in euthanasia). What is the total proportion of cats with present only with uveitis that are positive for FCoV on RT-PCR?

Take home: FCoV can hide out in eyes. Positive M1058L or S1060A gene mutation in a patient with uveitis does not mean that cat has FIP – correlation with clinical signs is required.

Future directions: Maybe cats with uveitis that are positive for FCoV on RT-PCR in aqueous in the absence of systemic disease may benefit from antivirals to prevent progression to FIP.

6. **JSAP 2025: Prevalence of behavioural signs commonly associated with feline hyperaesthesia syndrome among healthy cats.**
   Avril M, Maoureux A, Velentin S, Jeandel A[10]

Background: Feline hyperaesthesia syndrome (FHS) is thought to be a maladaptive pain condition by which affected individuals are hypersensitive to touch.[11] It is characterised by behavioural signs of lumbar and tail hyperaesthesia and may include self mutilation.[11] This condition is a diagnosis of exclusion of other causes of pain or discomfort (such as dermatologic or neuromuscular conditions) as well as correcting poor environment and stress triggers.[11] Common behaviours associated with FHS include licking or biting the tail, flanks, anal region or lumbar region, rolling skin, dilated pupils and vocalisation.[11,12] Typically owners will describe affected cats as highly aroused during episodes and behaviour may be provoked by touch and be more common if stressed.[11] There may breed predisposition in Siamese, Burmese, Himalayan and Abyssinian cats as well and clinical signs tend to appear for the first time in young cats (1 - 4 years old).[11,12]

Methods: Prospective study of healthy cats presenting to first opinion practice for vaccination were treated against ectoparasites, had a normal physical examination and were considered well by their owners. The owners of cats completed a survey at the start of the consultation about their cats behaviour at home (9 behaviours associated with FHS were chosen based on previous published literature) and behaviour when petted along their spine as well as their cats environment (indoors vs outdoors). The owners were unaware of the study aims. The attending veterinarian then completed a survey about the cats behaviour in consultation as well as the cats response to lumbar palpation. Six veterinarians were involved in data collection. The owners were then contacted one month after this visit to assess if any new medical condition had developed and if so these cats were excluded.

Results: 208 cats fulfilled the study criteria between October 2020 and May 2022. One hundred and fifty-two (73%) of cats consistently exhibited one or more of the 9 behavioural signs of FHS at home ranging from 27.4% only displaying one sign to one cat that displayed 8 of the 9 signs. Cats that lived exclusively indoors had an increased odds ratio (2.08) of showing one or more behavioural sign of FHS than cats that had some access to outdoors. Cats that reacted to owners petting along the spine had an OR 5.14 of having signs of FHS. Triggering of signs of FHS after spinal palpation in consultation did not seem to be associated with signs of FHS at home. The most common behavioural signs observed at home were jumping and wild uncontrolled running in 59% of cats. Other common behaviours observed were licking or biting of tail, flanks, anal region or lumbar region (20%), lying down and raising the lumbar region (19%), rolling skin (19%), sudden behavioural changes (16%).

Limitations: Overall small study size, interpretation of pet cats behaviour based on owners observations at home. The behaviour symptoms chosen for the owner survey were based out of the literature however 5/6 sources were text book chapters or review articles (I couldn't get the 1980 JAVMA paper by Tuttle). Different behavioural signs analysed were weighted equally in the analysis and different behavioural signs haven't formally been quantified in confirmed cases of FHS in the literature. Indoors cats having a higher odds of behavioural signs may be due to poor environment vs being seen by owners more hours of the day.

Take home: Behavioural signs of FHS are common in pet cats that are not affected by FHS. There is a suggestion that these behaviours are more common in pet cats that may be frustrated (indoors only environment) as well as cats that are reactive to spinal petting at home. More information on frequency of these clinical signs in confirmed cases will help to develop a scoring system for assessment of FHS. As with most aspects of cat medicine, there is always room for discussion of optimising environments and reducing stress.

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