Received: 16 November 2023 | Accepted: 18 April 2024

DOI: 10.1111/jvim.17098

Journal of Veterinary Internal Medicine Open Access

ACVIM American College of Veterinary Internal Medicine

CASE REPORT

Pseudohypoaldosteronism and acquired renal aldosterone resistance with hyperkalemic type IV renal tubular acidosis in 2 cats

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Abstract

This report describes the diagnosis and treatment of aldosterone resistance (AR) and acquired hyperkalemic type IV renal tubular acidosis (RTA) in 2 cats comparable to acquired pseudohypoaldosteronism in people. One cat developed AR from chronic kidney disease after an acute kidney injury and was treated with furosemide per os, which resolved the hyperkalemic RTA. The second cat developed transient AR secondary to a bacterial urinary tract infection associated with urethral catheterization, and treatment with antibiotics resolved the hyperkalemic RTA.

KEYWORDS

chronic kidney disease, endocrinopathy, hyponatremia, mineralocorticoid

1 | INTRODUCTION

Hyperkalemic type IV renal tubular acidosis (RTA) secondary to aldosterone resistance (AR) is called pseudohypoaldosteronism (PHA) in humans.[1-4] Pseudohypoaldosteronism can be caused by genetic receptor mutations and medications that affect the mineralocorticoid receptor (MR), and acquired due to diseases, such as chronic kidney disease (CKD) and urinary tract infection (UTI).[1,2,4-14] We describe 2 cats with a hyperkalemic RTA and AR comparable to acquired PHA in humans.

2 | CASE DESCRIPTION

Cat 1 was a 7-year-old, female spayed domestic shorthair that was initially presented to the referring veterinarian for evaluation of stranguria, pollakiuria, and hematuria. Cystoliths were diagnosed, and a cystotomy was performed. Over the next 9 days, the cat became progressively hyporexic, lethargic, and painful, prompting emergent evaluation. Physical examination revealed the cat to be 7 to 10% dehydrated with diffuse abdominal discomfort. The cystotomy incision was healed with no infection. Serum chemistry revealed International Renal Interest Society (IRIS) grade IV (http://www.iris-kidney.com/guidelines/grading.html) acute kidney injury ([AKI], blood urea nitrogen [BUN], >130 mg/dL; reference interval [RI], 15-32 mg/dL; and creatinine, 7.3 mg/dL; RI, 1.0-2.0 mg/dL) with normal electrolytes (potassium, 4.4 mmol/L; RI, 3.5-4.8 mmol/L; and sodium, 157 mmol/L; RI, 146-157 mmol/L). Serum chemistry and urine specific gravity (USG) were normal 5 weeks before the cystotomy. Abdominal ultrasonographic examination identified changes suggestive of acute nephritis and ureteritis with no evidence of ureteral obstruction, focal cystitis, right renal medullary mineralization, a right nephrolith,

Abbreviations: AKI, acute kidney injury; AR, aldosterone resistance; BUN, blood urea nitrogen; CCD, cortical collecting duct; CKD, chronic kidney disease; ECO_2, serum enzymatic carbon dioxide; GFR, glomerular filtration rate; IRIS, International Renal Interest Society; MR, mineralocorticoid receptor; PC, renal principal cell; PHA, pseudohypoaldosteronism; RI, reference interval; RTA, renal tubular acidosis; TCO_2, total carbon dioxide; UO, urethral obstruction; USG, urine specific gravity; UTI, urinary tract infection.

and bilateral decreased renal corticomedullary definition. The cat was hospitalized and rehydrated. A negative urine culture was obtained at the time of cystotomy, and antibiotics were not prescribed. The cat had complete clinical recovery on day 3 after the AKI, but remained azotemic with normal serum enzymatic carbon dioxide (ECO_2) and electrolytes (BUN, 36 mg/dL; RI, 15-32 mg/dL; creatinine, 3.2 mg/dL; RI, 1.0-2.0 mg/dL; ECO_2, 24 mmol/L; RI, 16.0-25.0 mmol/L; potassium, 4.1 mmol/L; RI, 3.5-4.8 mmol/L; and sodium, 157 mmol/L; RI, 146-157 mmol/L).

On day 39 after the AKI, the azotemia stabilized at IRIS stage 2 (http://www.iris-kidney.com/guidelines/staging.html) CKD, nonproteinuric, normotensive. Serial chemistry panels revealed a stable creatinine between 2.2 and 2.5 mg/dL (RI, 1.0-2.0 mg/dL) with normal electrolytes over the subsequent 60 days. On day 111 after the AKI, serum chemistry revealed progressive hyperkalemia (8.6 mmol/L; RI, 3.5-4.8 mmol/L), hyponatremia, (135 mmol/L; RI, 146-157 mmol/L), and acidosis (ECO_2, 13 mmol/L; RI, 16.0-25.0 mmol/L). The anion gap was 22 mmol/L (RI, 17-27 mmol/L), and the chloride was 109 mmol/L (RI, 111-124 mmol/L). The urine pH increased from 5.5 to 6.5 with a USG of 1.016. The azotemia remained unchanged (BUN, 54 mg/dL; RI, 15-32 mg/dL; and creatinine, 2.2 mg/dL; RI, 1.0-2.0 mg/dL). An ultrasonographic examination showed the earlier changes suggestive of nephritis, ureteritis, and cystitis had been resolved, but there was persistent nephrolithiasis, medullary mineralization, and decreased renal corticomedullary definition. The cat was diagnosed with a hyperkalemic RTA but remained asymptomatic. An ACTH stimulation test was performed with measurement of serum cortisol and aldosterone before and 60 minutes after intravenous administration of ACTH according to previously established protocols.[15] Aldosterone was measured using a commercially available radioimmunoassay (Coat-a-Count aldosterone assay, Siemens Medical Solutions Diagnostics, Los Angeles, CA). The resting serum cortisol and ACTH-stimulated serum cortisol were 2.67 and 6.68 μg/dL (RI, 1-10 μg/dL), respectively. The resting and ACTH-stimulated serum aldosterone levels were greater than 4577 pmol/L (resting aldosterone RI, 194–388 pmol/L; ACTH-stimulated aldosterone RI, 277–721 pmol/L). The cat was diagnosed with AR comparable to acquired PHA in humans. Furosemide (Furosemide oral solution USP, West-Ward Pharmaceuticals Corp., Eatontown, NJ; 1.22 mg/kg q12h PO) therapy was initiated. One month later (day 141 after the AKI), serum chemistry showed normal potassium (4.4 mmol/L; RI, 3.5-4.8 mmol/L), near normal sodium (145 mmol/L; RI, 146-157 mmol/L), and resolved acidosis (ECO_2, 19 mmol/L; RI, 16.0-25.0 mmol/L). The azotemia improved to IRIS stage 1 (http://www.iris-kidney.com/guidelines/staging.html) CKD (BUN, 37 mg/dL; RI, 15-32 mg/dL; and creatinine, 1.3 mg/dL; RI, 1.0-2.0 mg/dL), nonproteinuric, normotensive. Subsequent rechecks with the cat receiving oral furosemide over the following 12 months revealed unchanged IRIS stage and electrolytes.

Cat 2 was a 4-year-old, male neutered, domestic shorthair that presented to a local emergency clinic and was diagnosed with a urethral obstruction (UO). Serum chemistry revealed an IRIS Grade V (http://www.iris-kidney.com/guidelines/grading.html) AKI (BUN, >130 mg/dL; RI, 16-36 mg/dL; creatinine, >13.0 mg/dL; RI, 0.8-2.4 mg/dL; and potassium, 10.0 mmol/L; RI, 3.5-5.8 mmol/L). Three urinary catheters were placed and replaced throughout hospitalization. Urinalysis revealed a pH of 7.0, USG of 1.019, marked proteinuria, hematuria, struvite crystalluria, and epithelial cell rafts. At discharge, the azotemia had resolved, and the electrolytes were normal (BUN, 26 mg/dL; RI, 15-32 mg/dL; creatinine, 1.6 mg/dL; RI, 1.0-2.0 mg/dL; potassium, 4.0 mmol/L; RI, 3.5-5.8 mmol/L; sodium, 162 mmol/L; RI, 150-165 mmol/L; and chloride, 124 mmol/L; RI, 112-129 mmol/L). Urine culture returned negative.

The cat was presented 6 days later for evaluation of persistent lethargy, anorexia, and pollakiuria. Physical examination revealed 12% dehydration and dull mentation, but no UO was present. Serum chemistry revealed an IRIS Grade II (http://www.iris-kidney.com/guidelines/grading.html) AKI (BUN, 77 mg/dL; RI, 16-36 mg/dL; and creatinine, 2.4 mg/dL; RI, 0.8-2.4 mg/dL), hyponatremia (135 mmol/L; RI, 150-165 mmol/L), hyperkalemia (7.3 mmol/L; RI, 3.5-5.8 mmol/L), and acidosis (ECO_2, 13 mmol/L; RI, 16.0-25.0 mmol/L). Abdominal ultrasonographic examination revealed evidence of cystitis. Urinalysis identified rod bacteriuria, pyuria, hematuria, hyaline casts, and a pH of 8.0. Ampicillin-sulbactam ([Unasyn (UNASYN {ampicillin sodium/sulbactam sodium}, Pfizer Inc., New York, NY)], 30 mg/kg IV q8h) was prescribed. Despite rehydration, the hyperkalemia, hyponatremia, and metabolic acidosis persisted during hospitalization. Baseline serum cortisol and aldosterone levels (Coat-a-Count aldosterone assay, Siemens Medical Solutions Diagnostics, Los Angeles, CA) were obtained. The owners declined mineralocorticoid treatment while awaiting hormone results and took the cat home after 3 days of hospitalization with resolved azotemia (BUN, 16 mg/dL; RI, 16-36 mg/dL; and creatinine, 1.1 mg/dL; RI, 0.8-2.4 mg/dL), but persistent hyperkalemia (potassium, 6.7 mmol/L; RI, 2.9-4.2 mmol/L), hyponatremia (135 mmol/L; RI, 147-162 mmol/L), and acidosis (ECO_2, 15 mmol/L; RI, 16.0-25.0 mmol/L). The anion gap was 13.7 (RI, 10-27 mmol/L), and the chloride was 113 mmol/L (RI, 112-129 mmol/L). The cat was sent home with amoxicillin–clavulanic acid ([Clavamox (Clavamox Chewable Tablets {62.5 mg}, Zoetis Canada Inc., Kirkland, QC, Canada)], 13.39 mg/kg PO q12h). Electrolytes were rechecked 24 hours after hospital discharge and showed resolved hyperkalemia and acidosis (potassium, 4.1 mmol/L; RI, 2.9-4.2 mmol/L; and ECO_2, 21 mmol/L; RI, 16.0-25.0 mmol/L), and improved hyponatremia (sodium, 142 mmol/L; RI, 147-162 mmol/L). The anion gap was 11 (RI, 11-27 mmol/L), and the chloride was 115 mmol/L (RI, 112-129 mmol/L). Baseline serum cortisol was 5.1 μg/dL (RI, 1-10 μg/dL), and baseline serum aldosterone was 5548 pmol/L (RI, 194-388 pmol/L). Cat 2 was diagnosed with transient AR comparable to transient acquired PHA in humans.

3 | DISCUSSION

Aldosterone resistance results in natriuresis, potassium retention, and decreased urinary acid excretion, leading to a hyperkalemic RTA.[1-3] The diagnosis of AR and PHA in humans is based on documenting marked hyperaldosteronemia with 2 or more of the following serum abnormalities: hyponatremia, hyperkalemia, and nonanion gap metabolic acidosis.[9,10,16] Cat 1 developed AR presumed secondary to tubulointerstitial inflammation and was treated with furosemide resulting in the normalization of electrolytes. Cat 2 developed AR presumed

secondary to a bacterial UTI from multiple urethral catheterizations. The hyponatremia and hyperkalemia did not resolve by restoring the glomerular filtration rate (GFR) but required 2 additional days of antibiotics. In veterinary medicine, AR has been documented secondary to medications, but not from known genetic defects or secondary to diseases (acquired PHA). Both cats in this report developed AR with a syndrome comparable to acquired PHA in humans.

Cat 1 developed AR presumed secondary to tubulointerstitial inflammation characteristic of CKD. While the inflammation, tubular atrophy, and interstitial fibrosis progress with worsening IRIS stage, even IRIS stage 2 CKD cats have significant changes.[17] Hyperkalemia in humans with CKD is typically present in advanced disease and is caused by hyporeninemic hypoaldosteronism from reduced juxtaglomerular apparatus cell mass and decreased aldosterone synthesis.[2,14,18] Some humans will exhibit hyperkalemia secondary to AR and not as a consequence of decreased GFR.[16] In humans and cats with CKD, transforming growth factor β1 is the primary mediator of tubulointerstitial inflammation and fibrosis.[17,19,20] This and other inflammatory mediators, including tumor necrosis factor α, thromboxanes, and interleukin-1 and -6, have been demonstrated to directly inhibit aldosterone's action on the MR.[10,14,21] MR antagonism can be persistent or transient if the inflammation resolves, especially in critical illness.[22]

In humans, hyperkalemic RTA is often an incidental finding unless arrhythmias are present or sodium wasting leads to hypovolemia.[7,8] In symptomatic patients, mineralocorticoid supplementation is often initiated while awaiting serum aldosterone results, and occasionally serum aldosterone measurement will only be considered when mineralocorticoid supplementation fails to reverse electrolyte changes.[1,8] Because cat 1 was asymptomatic, treatment was delayed until hormone results returned. The primary determinant of renal principal cell (PC) function, with or without aldosterone, is the delivery of sodium to the cortical collecting duct (CCD) lumen.[6,16] The cat was treated with furosemide to increase sodium delivery to the CCD, which facilitates sodium reabsorption through the available PC epithelial sodium channels, creating an electronegative potential in the CCD lumen. This allows for positively charged potassium and hydrogen ions to be excreted.[18] After furosemide was initiated, the cat's azotemia improved to IRIS stage 1 CKD. This could be a timely change secondary to continued recovery of renal function, or potentially a consequence of furosemide. With loop diuretics, natriuresis leads to volume contraction and reduced GFR secondary to activation of the renin-angiotensin-aldosterone system.[18,23] It is possible that the furosemide increased the ability of the PC to reabsorb sodium and water to improve effective circulating volume, and thus GFR, leading to improved azotemia.

Cat 2 developed transient AR presumed secondary to a UTI diagnosed with rod bacteriuria, pyuria, hematuria, and proteinuria on his UA, which were distinctly different from his UA at the time of UO. Ultrasound of the kidneys was normal; however, pyelonephritis cannot be ruled out. In humans and dogs, ultrasonographic detection of pyelonephritis is not 100% sensitive, and, although unknown in cats, is presumed similar.[5,24,25] In neonatal and pediatric humans, acquired PHA is documented secondary to bacterial UTIs.[5,9,11,13] In those with pyelonephritis, 75% had electrolyte and acid-base anomalies, the most common being hyponatremia secondary to AR.[11] Infections are typically *Escherichia coli*, but other bacteria have been found.[9,11] The mechanisms are not completely understood but are likely caused by direct endotoxin MR antagonism, an inflammatory storm within the kidney, or decreased expression of MR receptors.[3,9,11,13] Antibiotic therapy for the infection can resolve the AR in 1 to 14 days.[12-14] Initially, the electrolyte abnormalities in cat 2 were attributed to hypertonic fluid loss; however, with euvolemic restoration, the hyperkalemia and hyponatremia persisted. This subsequently triggered testing for hypoadrenocorticism and hypoaldosteronism. With a normal baseline serum cortisol and marked hyperaldosteronemia in the face of hyponatremia, hyperkalemia, and a normal anion gap, the cat was diagnosed with AR comparable to acquired PHA in humans. Unlike cat 1, the AR was transient and resolved with antibiotics and time.

Limitations include the lack of rechecking aldosterone concentrations after resolution of the hyperkalemic RTA in both cats; however, if aldosterone levels were continuously elevated without AR, we would expect the potassium concentration to decrease dramatically, which was not seen in either cat. For cat 1, it is unknown whether the AR was persistent or transient. The patient tolerated the furosemide therapy well, and a discontinuation challenge may have led to relapsing hyperkalemia, thus was not pursued. The GFR was not measured in either cat at the time of AR diagnosis. In humans, GFR does not impact the overall potassium secretion until reduced by 85 to 90%, and they are oliguric.[6] Once serum creatinine concentration is elevated in cats, at least 75% of renal function has been lost.[23] Cat 1 stabilized at IRIS stage I CKD, and without performing a GFR study, it is difficult to know what percentage of renal function remained. Cat 2 had a resolution of azotemia, and whether there was permanent damage leading to CKD is unknown without a GFR study, and potentially a biopsy. However, it is unlikely either cat had sufficient reduction in GFR to account for the degree of hyperkalemia seen, especially in the presence of hyponatremia and elevated aldosterone levels. The urinary fractional excretion of electrolytes was not measured in either cat. The changes secondary to furosemide administration in cat 1 would be of particular interest. Finally, a venous blood gas was not performed in either cat, limiting the analysis of serum pH and bicarbonate. However, ECO_2 has shown excellent correlation to total carbon dioxide (TCO_2), and because bicarbonate is 95% of TCO_2, ECO_2 is considered a good approximation of bicarbonate and, therefore, estimation of serum pH.[26,27]

This is a report of AR in cats comparable to acquired PHA in humans. The most recognized cause of a hyperkalemic type IV RTA in cats is hypoadrenocorticism, with a rare case of isolated hypoaldosteronism reported. The presentation of a hyperkalemic RTA (hyperkalemia, hyponatremia, and nonanion gap metabolic acidosis) in a euvolemic animal with a normal cortisol should prompt measurement of serum aldosterone levels, consideration and potential removal of drugs that could cause MR antagonism, and initiation of mineralocorticoid supplementation. Aldosterone resistance should be considered if the hyperkalemic RTA does not respond to mineralocorticoid supplementation, particularly in those patients with CKD, an AKI without oligoanuria, pyelonephritis, and critical illness. If AR is suspected, the cat is symptomatic for hyponatremia or hyperkalemia, and the GFR has been restored to normal or

near normal, a furosemide trial should be considered, and, if needed, enteric potassium binders are initiated while awaiting hormone results. These 2 cats represent a novel differential diagnosis for a hyperkalemic type IV RTA and will allow for further categorization similar to humans once more cases are recognized.

ACKNOWLEDGMENTS

No funding was received for this study.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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How to cite this article: Marino CL, Foster JD. Pseudohypoaldosteronism and acquired renal aldosterone resistance with hyperkalemic type IV renal tubular acidosis in 2 cats. *J Vet Intern Med*. 2024;38(4):2344-2347. doi:10.1111/jvim.17098