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CASE REPORT

Companion or pet animals

Juvenile primary acquired hypothyroidism in a dog with suspected renal dysplasia

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Abstract

A 1-year 3-month-old, female, neutered crossbreed dog presented for investigation of polyuria, polydipsia and azotaemia. Subsequent biochemistry, endocrine testing and abdominal ultrasound identified hypercholesterolaemia, thyroglobulin autoantibody-positive hypothyroidism, and IRIS stage II non-hypertensive, non-proteinuric suspected renal dysplasia, with a concurrent *Escherichia coli* urinary tract infection. The patient responded well clinically to levothyroxine treatment, with improvement in hypercholesterolaemia, azotaemia and demeanour at home. At follow-up biochemistry assessment, hyperphosphataemia and hypercalcaemia (total and ionised) were documented, presumed to be driven by renal insufficiency and renal secondary hyperparathyroidism, although this aetiology was not definitively proven. Normalisation of these values was noted following renal diet implementation and addition of phosphate binders. This case highlights acquired primary hypothyroidism with evidence of thyroiditis in a juvenile dog with suspected congenital renal dysplasia.

KEYWORDS

endocrinology, hypothyroidism, renal disease, renal dysplasia

BACKGROUND

Hypothyroidism is a common endocrinopathy affecting dogs wherein there is an inadequate level of thyroxine (T4). Clinical signs of hypothyroidism can be variable, with dermatological, metabolic, neurological, cardiac and ocular systems among those affected.[1] Diagnosis can be challenging, and misdiagnosis is not uncommon, with one study reporting 52.2% of dogs with non-thyroidal illness having low total T4 (tT4) concentrations.[2] The presence of decreased tT4 in isolation does not confirm disease but should prompt the clinician to assess further for signs of non-thyroidal illness, review the patient history for recent drug administration that could result in diminished T4, and perform additional blood tests for assessment of thyroid function. Frequently utilised commercially available tests to support a true diagnosis of hypothyroidism include thyroid-stimulating hormone (TSH), free T4 (fT4) by equilibrium dialysis, and thyroglobulin autoantibodies (TgAA). Congenital and acquired versions of hypothyroidism exist, both of which can be caused by central (pituitary or hypothalamus) or primary (thyroid) forms of hypothyroidism, resulting from different aetiopathogeneses and possessing different diagnostic criteria.[3–6]

Acquired hypothyroidism is more common, with most cases being primary, either due to lymphocytic thyroiditis or thyroid gland atrophy. It is usually slowly progressive and diagnosed later in life. Classic clinical signs of hypothyroidism include symmetrical alopecia, weight gain and lethargy. Diagnosis of acquired hypothyroidism can be achieved by documenting low tT4 and fT4, accompanied by an elevation in TSH,[3,4] although it should be noted that TSH can be within normal limits (in approximately 25% of cases).[3] Diagnosis of lymphocytic thyroiditis can be achieved through documentation of a positive TgAA titre, although a negative titre may be seen in later stages of disease.[7]

Renal dysplasia is a developmental disorder of the kidneys, which can lead to early-onset azotaemia and progressive renal dysfunction. While histopathology of affected kidneys is required for definitive diagnosis,[8–10] in cases with appropriate signalment and clinicopathologic abnormalities, ultrasound findings are often deemed adequate,[11] and reportedly corroborate with severity on histopathology.[12]

When a patient presents with multiple biochemical and/or physical abnormalities, thorough assessment is critical to ensure no opportunity to optimise patient health is missed, as both congenital and acquired conditions can occur concurrently. Clinicians should bear in mind that dogs diagnosed with one congenital disease are commonly diagnosed with concurrent congenital illnesses,[13–15] and exploration of these should be considered if there are appropriate clinicopathologic abnormalities. We herein present a dog diagnosed with suspected congenital renal dysplasia and juvenile-acquired hypothyroidism.

CASE PRESENTATION

A 1-year 3-month-old, female, neutered, crossbreed dog presented for investigation of pollakiuria, suspected recurrent urinary tract infections, dilute urine and azotaemia of 2-month duration. No polydipsia, urinary incontinence or stranguria was reported. Two months before referral, the patient had received a course of amoxicillin-clavulanate (Synulox, Zoetis UK) for suspected vaginitis, at which time the urine specific gravity (USG) was 1.010, with a subsequent documented USG of 1.002; there was no other significant medical history or drug therapy reported. The patient had also been placed onto a renal therapeutic diet (Hill's k/d, Hill's Pet Nutrition) in response to azotaemia, although information on diet before this is not available. The dog was imported at 4 months of age from Bosnia.

INVESTIGATIONS

Physical examination was unremarkable, except for an increased body condition score (7/9) and an immature, recessed vulva. Systolic blood pressure was 128 mmHg. Haematology parameters fell within normal limits. Serum biochemical abnormalities of note included hypercholesterolaemia (25 mmol/L; reference interval [RI]: 3.8–7.0 mmol/L), hypertriglyceridaemia (2.25; RI: 0.57–1.14 mmol/L), elevated creatinine (174 μmol/L; RI: 22–115 μmol/L) and mild hyperalbuminaemia (35.7 g/L; RI: 26–35 g/L). Urinalysis, collected via cystocentesis, revealed hyposthenuria (1.006; RI: >1.030), an unremarkable dipstick evaluation, with rod bacteria identified on sediment evaluation (Sedivue, Idexx). Subsequent urine culture on the same sample was positive for a non-haemolytic *Escherichia coli*, sensitive to all the antibiotics tested on a standard panel. A urine protein:creatinine ratio was also submitted on the same sample and found to be normal (0.05; RI: <0.2). Hypoadrenocorticism as a cause of hyposthenuria and azotaemia was excluded on the basis of an appropriate ACTH stimulation test (pre-stimulation cortisol 43.3 nmol/L; RI: 20–230 nmol/L, post-stimulation cortisol 339 nmol/L). Hypothyroidism was considered as a cause of the hypercholesterolaemia, and a diagnosis was reached based on low total thyroxine (<13 nmol/L; RI: 15–48 nmol/L), low free T4 (equilibrium dialysis <2 pmol/L; RI: 7–40 pmol/L) in the presence of increased TSH (2.02 ng/mL; RI: 0–0.5 ng/mL). A thyroglobulin autoantibody assay was later performed and confirmed immune-mediated thyroiditis, consistent with juvenile-acquired primary hypothyroidism.

To further investigate the azotaemia, dilute urine and evaluate the kidney for changes consistent with pyelonephritis, an ultrasound of the urogenital tract was performed under supervision from a Diplomate in diagnostic imaging. Reported changes included marked bilateral loss of cortico-medullary definition and irregularity of the cortices. The left kidney (Figure 1) was moderately reduced in size at less than 4 cm in length, and the right (Figure 2) was mildly increased in size at 6.5 cm in length. There was no evidence of pelvis dilation or retroperitoneal free fluid. Based on imaging findings, congenital renal dysplasia was considered most likely, and other differentials for these changes were not listed on the ultrasound report. The urinary bladder and adrenals were within normal limits (left adrenal measures 0.45 cm, and right measures 0.52 cm at the caudal poles).

Due to the patient's travel history and previous documentation of vaginitis, *Brucella canis* serology was performed; the results were negative (<1/25).

LEARNING POINTS/TAKE-HOME MESSAGES

- Acquired hypothyroidism, and other acquired endocrinopathies, should not be excluded in young dogs with appropriate clinical and clinicopathologic abnormalities.
- Thorough investigation into co-morbidities should be performed, especially in cases where all clinicopathologic abnormalities cannot be explained by one diagnosis alone.
- Canine hypothyroidism can decrease glomerular filtration rate and give rise to pre-renal azotaemia, and should therefore be considered in dogs with appropriate clinical or clinicopathological findings.
- This case demonstrates the importance of considering concurrent, potentially reversible diseases (in particular endocrinopathies) that may be contributing toward pre-renal azotaemia.

DIFFERENTIAL DIAGNOSIS

For the azotaemia, acute and chronic causes were considered. Potential acute renal causes included pyelonephritis, hypoadrenocorticism and toxic insult (although this was excluded based on history). It should be noted that hypoadrenocorticism could be multifactorial in its contribution to azotaemia, and may also have a pre-renal component. Other pre-renal causes, such as hypotension arising from hypothyroidism, were also later considered. Chronic renal causes, such as chronic kidney disease arising from renal dysplasia, were also a possibility in light of the patient's age. Post-renal causes, such as urinary obstruction, were considered less likely in this dog based on the history.

While not included on the final ultrasound report, the renal changes described could be consistent with renal dysplasia, chronic kidney disease or chronic pyelonephritis. The disparity in size could reflect unilateral hypertrophy in compensation to dysfunction.[16]

For hypertriglyceridaemia, differentials include postprandial effects, familial dyslipidaemia, and certain drugs (such as glucocorticoids and phenobarbital). Endocrine causes include hypothyroidism (congenital or acquired), hyperadrenocorticism, diabetes mellitus (of which, the latter two had no evidence in this dog). Pancreatitis is another possible cause of hypertriglyceridaemia, but considered unlikely in this dog with no relevant clinical signs. Glomerular disease was also considered, and a urine protein:creatinine ratio was performed to better assess this.

The subsequently identified concurrent hyperphosphataemia and hypercalcaemia were considered to be related

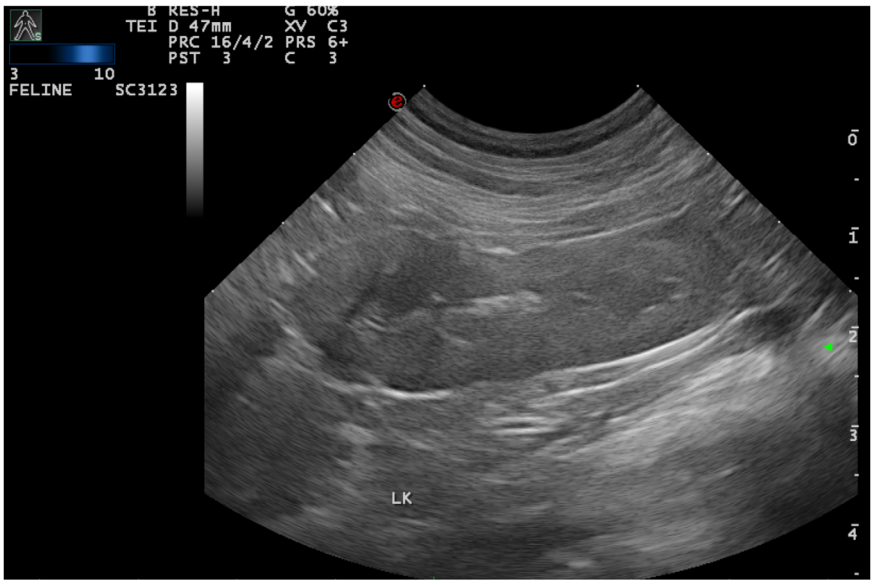

FIGURE 1 Ultrasound image of longitudinal sections of left kidney. The left kidney was reported reduced in size (length <4 cm).

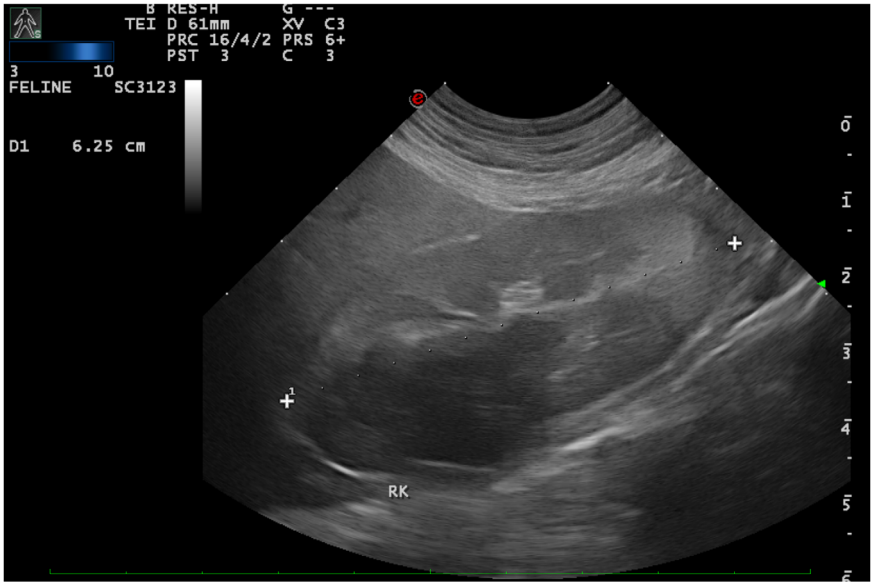

FIGURE 2 Ultrasound image of longitudinal sections of the right kidney. The right kidney size was mildly increased (length 6.5 cm).

to secondary renal hyperparathyroidism, and response to management of the renal disease supported this. However, other causes of hypercalcaemia include primary hyperparathyroidism, hypoadrenocorticism, hypervitaminosis D, granulomatous disease, osteolytic disease and certain cancers (lymphoma, apocrine gland of the anal sac adenocarcinoma, multiple myeloma—all considered unlikely in this dog). Spurious lab results were also considered, but repeated consistent results made this less likely.

It should be considered that a multifactorial aetiology could be contributing to the biochemical abnormalities identified in this dog.

TREATMENT

The patient was ultimately diagnosed with hypothyroidism, a urinary tract infection and non-hypertensive, non-proteinuric IRIS II chronic kidney disease due to possible renal dysplasia.[17] Based on bacterial antibiotic sensitivity, treatment with amoxicillin-clavulanate (12 mg/kg orally [PO] every 12 hours for 14 days; Synulox, Zoetis UK) was prescribed. Levothyroxine (Leventa, MSD Animal Health UK) at a starting dose of 10 μg/kg (PO every 12 hours) was also prescribed. In terms of nutritional guidance, management of the marked hypercholesterolaemia was prioritised, and the dog was transitioned on to a specific low-fat diet with restricted phosphate levels (Hill's w/d, Hill's Pet Nutrition).

OUTCOME AND FOLLOW-UP

The dog responded to levothyroxine, with a tT4 assessment 4 weeks after initiation of treatment measuring 43.1 nmol/L (RI: 13–51 nmol/L). The client reported an overall more 'youthful' demeanour. The cholesterol levels improved (8.76 mmol/L; RI: 3.2–6.2 mmol/L), consistent with hypercholesterolaemia secondary to hypothyroidism. The most recent cholesterol level was 10.17 mmol/L (RI: 3.2–6.2 mmol/L), with a total T4 at this time of 44.1 nmol/L (RI: 13–51 nmol/L).

Subsequent serial assessment of the patient's renal disease identified improvement in creatinine (range 93–113 μmol/L; RI: 44–133 μmol/L), and a persistently mild increase in

SDMA according to the laboratory reference interval[18] (range 16–19 μg/dL; RI: 1–14 μg/dL), which to date remains stable confirming a current non-hypertensive, non-proteinuric IRIS stage II classification, on the basis of the most recent SDMA value (see Table 1). At the follow-up, the serum phosphate and ionised calcium increased to a maximum of 1.81 mmol/L (RI: 0.8–1.6 mmol/L) and 1.5 mmol/L (RI: 1.25–1.45 mmol/L), respectively. A chitosan and calcium carbonate-based phosphate binder (Ipakitine, Vetoquinol) was initiated, but no significant improvement was documented in phosphate (1.71 mmol/L; RI: 0.8–1.6 mmol/L) or total calcium (3.01 mmol/L; RI: 2.36–2.84 mmol/L) 6 weeks later. Calcium oxalate crystals were seen on urine sediment, and the USG was 1.044. Secondary renal hyperparathyroidism was considered as a possible mechanism driving the hypercalcaemia and hyperphosphataemia, although it should be noted that a parathyroid hormone assay was not performed. As such, the dog was transitioned to a renal prescription diet (Hill's k/d, Hill's Pet Nutrition), which it was fed before referral. After 1 month, phosphate levels had improved (1.58 mmol/L; RI: 0.8–1.6 mmol/L), although not to within the target laid out in the treatment guidelines for IRIS stage II chronic kidney disease (<1.5 mmol/L).[19] The patient was transitioned to a different phosphate binder (Pronefra, Virbac UK), and 2 months later ionised calcium, total calcium and phosphate were normal (see Table 1). Creatinine was 100 μmol/L (RI: 44–133 μmol/L) and SDMA 18 μg/dL (RI: 1–14 μg/dL). The USG at this time was 1.010, with no crystalluria, a negative urine culture and an unremarkable dipstick evaluation.

DISCUSSION

If it had not been for the identification of marked hyperlipidaemia and the desire to exclude diseases that can cause secondary hyperlipidaemia, hypothyroidism would have not been considered a differential in this young dog. The presence of the hyperlipidaemia and the azotaemia could prompt the clinicians to consider hypothyroidism more readily than was the case in this patient. It should also be noted that other differentials for hyperlipidaemia were considered and excluded at the time of presentation.[20,21] Interestingly, serum cholesterol levels did not increase to pre-levothyroxine treatment values after the patient was later transitioned to a renal prescription diet, supporting hypothyroidism as the main contributor to hypercholesterolaemia.

Initially, in light of the free and total T4 levels and the patient's age, congenital hypothyroidism was considered. Congenital hypothyroidism is uncommonly reported in dogs. Typically, clinical signs will manifest during early life development, with classic signs of disproportionate dwarfism and delayed growth,[22] mental dullness, lethargy and weight gain.[23] The pathogenesis is not fully understood in dogs, but proposed mechanisms include central dyshormonogenesis and synthesis failure at the level of the thyroid gland.[4,6] Central congenital hypothyroidism is confirmed in patients with appropriate clinical signs and with low tT4 and associated inappropriately low TSH.[6] The increased TSH excluded congenital central hypothyroidism in this dog, and similarly the patient had no other physical exam findings to support concurrent central endocrinopathies, such as pituitary

TABLE 1 Trends of relevant biochemical parameters over time related to day of diagnosis of hypothyroidism and renal dysplasia.

Time from diagnosis of hypothyroidism and renal dysplasia (days)	Creatinine (μmol/L)	SDMA (μg/dL)	Phosphorous (mmol/L)	Ionised calcium (mmol/L)	Total calcium (mmol/L)	Cholesterol (mmol/L)	Total T4 (nmol/L)	Free T4 (pmol/L)	TSH (ng/mL)
–17	175 (RI: 44–133)	18 (RI: 1–14)	1.6 (RI: 0.8–1.6)	[a]	2.83 (RI: 2.36–2.84)	>18 (RI: 3.2–6.2)	9.2 (RI: 13–51)	[a]	[a]
0	174 (RI: 22–115)	[a]	1.3 (RI: 0.9–2.0)	1.38 (RI: 1.15–1.5)	2.99 (2.3–3.0)	25 (RI: 3.8–7.0)	<13 (RI: 15–48)	<2 (RI: 7–40)	2.02 (RI: 0–0.5)
18	113 (RI: 44–133)	16 (RI: 1–14)	1.81 (RI: 0.8–1.6) 1.77 (RI: 0.9–1.6)	1.5 (RI: 1.25–1.45)	2.98 (RI: 2.36–2.84) 3.01 (2.2–3.0)	8.76 (RI: 3.2–6.2)	43.1 (RI: 13–51)	[a]	[a]
60	100 (RI: 44–133)	19 (RI: 1–14)	1.71 (RI: 0.8–1.6)	[a]	3.01 (RI: 2.36–2.84)	[a]	58.4 (RI: 13–51)	[a]	[a]
88	93 (RI44–133)	16 (RI: 1–14)	1.57 (RI: 0.8–1.6)	[a]	2.76 (RI: 2.36–2.84)	9.7 (RI: 3.2–6.2)	47.8 (RI: 13–51)	29.5 (RI: 7–40)	1.11 (RI: 0–0.5)
186	100 (RI: 44–133)	18 (RI: 1–14)	1.37 (RI: 0.8–1.6) 1.38 (RI: 0.9–1.6)	1.36 (RI: 1.25–1.45)	2.84 (RI: 2.2–3.0) 2.8 (2.36–2.84)	10.17 (RI: 3.2–6.2)	44.1 (RI: 13–51)	[a]	[a]

Note: For phosphorous, total calcium, there were on occasion values from two laboratories acquired on the same blood sample. Both have been included for reference. Levothyroxine was initiated on Day 1 at a dose of 10 μg/kg (orally every 12 hours). An 11% total dose reduction in levothyroxine was made on Day 66. Diet modification to Hills w/d (Hill's Pet Nutrition) was from Day 1. On Day 119, the patient was transitioned onto Hills k/d (Hill's Pet Nutrition). Ipakitine (Vetoquinol) was started on Day 53. This was discontinued, and Pronefra (Virbac UK) initiated on Day 120.

Abbreviations: RI, reference interval; TSH, thyroid-stimulating hormone.

[a]Not assessed.

dwarfism,[7,24] which have been reported concurrently with central congenital hypothyroidism.[6] A genetic component to T4 dysgenesis has been postulated in certain breeds, including the toy fox[25] and rat terriers,[26] wherein low tT4 is accompanied with increased TSH, the same thyroid hormone changes as in this dog. However, this would be considered highly unlikely in this dog that did not show clinical signs of congenital hypothyroidism.

To better understand the aetiology of hypothyroidism in this young dog, TgAA titre was performed. While not all dogs with a positive TgAA will go on to develop clinical hypothyroidism, this finding supported the diagnosis of lymphocytic thyroiditis and acquired primary hypothyroidism. Contrary to our case, acquired hypothyroidism is typically a disease of adult dogs,[27–29] perhaps owing to the slow destruction of functional thyroid tissue. Previous studies in Borzoi dogs found histopathological evidence of lymphoid thyroiditis in the test subjects as young as 2.5 years old. However, in contrast to the case described, these dogs did not demonstrate clinical or clinicopathological features of hypothyroidism at this age.[30]

In a recent review of a breed surveillance programme, non-clinical Eurasian dogs were presented for thyroid screening between the ages of 8 and 160 months initially. At first presentation, 26/118 TgAA-positive dogs were found to be hypothyroid (defined as low serum tT4 and high TSH) without reported clinical signs.[7] The median and mean ages of TgAA-positive hypothyroidism in this cohort were 1 and 2.23 years, respectively. With the absence of clinical signs reported in this group, this may further support that clinicopathological changes of hypothyroidism can pre-date typical clinical disease.[31] Out of the previously reported breeds,[32,33] the diagnosis of juvenile, clinically relevant hypothyroidism, as exhibited by the dog described here, remains rare within the literature.

The hyposthenuria identified in the early stages of presentation was not readily explained by chronic kidney disease, nor sporadic cystitis. As such, chronic pyelonephritis in addition to chronic renal insufficiency was considered. Pyelonephritis can be challenging to diagnose, and although this dog lacked common features (fever, renal pain, renal pelvis dilation), the positive urine culture and azotaemia prompted the 14-day antibiotic course. The International Society for Companion Animal Infectious Diseases (ISCAID) guidelines for the diagnosis and management of bacterial urinary tract infections in dogs and cats recommends a fluoroquinolone as the first-line treatment for suspected pyelonephritis.[34] However, within the authors' institution, the use of fluroquinolones is reserved for severely ill patients. Therefore, based on the antimicrobial sensitivity test, the sporadic nature of the possible pyelonephritis, and in keeping with hospital antibiotic stewardship, amoxicillin-clavulanic acid was initiated in the first instance.[35]

Although a USG of 1.045 is not typical of CKD, it is commonly recognised that this alone cannot preclude a CKD diagnosis, given that approximately 66% of nephron function loss is expected before seeing changes in urine concentrating ability.[36] The diagnosis of CKD in this case was supported by increasing SDMA and structural changes on renal ultrasonography, in keeping with IRIS categorisation of CKD. A definitive explanation for the presence of azotaemia and ultrasonographic features of the kidneys was not reached, and it is acknowledged that in order to prove renal dysplasia, renal biopsies would be required, rather than relying solely on ultrasound changes. Other plausible explanations for the temporary nature of both the azotaemia and isosthenuria includes an acute insult to the kidneys, either from poor glomerular filtration rate (GFR) arising from hypothyroidism or pyelonephritis.

Azotaemia was noted to improve within 3 weeks of concurrent initiation of treatment with levothyroxine and amoxicillin-clavulanate. While this, in part could be due to treatment of an ascending infection, contributing to kidney damage, the authors also consider the role that treatment of hypothyroidism may have played. It has been previously documented that in promoting euthyroidism, the GFR is increased.[37,38] Similarly, in a recent study, one third (8/24) of hypothyroid dogs were azotaemic at the time of diagnosis. Of these eight dogs, all showed a decrease in serum creatinine concentration following initiation of levothyroxine treatment, and all but one were no longer azotaemic at follow-up assessment.[39] This pattern is similar to what was seen in this dog, supporting that the initial azotaemia and raised SDMA was pre-renal in origin, and not a result of renal disease.[18]

The subsequently identified total and ionised hypercalcaemia and hyperphosphataemia have not been definitively explained in this case. Causes such as hypoadrenocorticism and vitamin D toxicity were excluded based on ACTH stimulation and history.[40] Interestingly, hypercalcaemia has been reported in association with both congenital[22] and acquired[41] canine hypothyroidism. However, this was deemed unlikely given hypercalcaemia developed after initiation of levothyroxine treatment when the dog was euthyroid. Due to the response to dietary phosphate restriction and the lack of another plausible explanation for these changes, renal secondary hyperparathyroidism was considered by the clinicians to be the most likely aetiology; however, this cannot be proven without a parathyroid hormone assay, which was not performed.[42]

This case highlights that acquired endocrinopathies, particularly hypothyroidism, should not be excluded on the basis of patient age if clinical signs, biochemical abnormalities and endocrine testing support the diagnosis. Young dogs with azotaemia, with appropriate clinical or clinicopathological findings should have hypothyroidism considered as a contributor to reduced GFR, especially in the face of concurrent chronic kidney disease.

AUTHOR CONTRIBUTIONS

Patient initially seen by and management plan created by Glynn Woods and Callum Atkins. Sophie Parton then took over with supervision from Glynn Woods and Alisdair Boag. Initial plan for case report formulated by Sophie Parton and Glynn Woods. Sophie Parton, Glynn Woods, Callum Atkins and Alisdair Boag all involved in interpreting and analysing results and writing and reviewing paper.

CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

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ETHICS STATEMENT

The patient was treated according to the standards of care, and owner's consent was obtained for all investigations.

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