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APPREACH TO THE DOG WITH POLYURIA AND POLYDIPSIA

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Introduction

Polyuria and polydipsia are frequent presenting complaints in small animal practice. Polyuria is defined as a daily urine output of greater than 50 ml/kg per day, while polydipsia is defined as a fluid intake of more than 100 ml/kg/day. Healthy dogs generally consume between 50-60 ml/kg/day, depending on the moisture content of their diets, the ambient temperature and humidity and their level of activity. Normal urine production is approximately 20 – 40 ml/kg /day or put differently, 1-2 ml/kg/hour.

The balance between water loss and water intake results from interactions between the hypothalamus, the pituitary gland and the kidney and is maintained by thirst and renal excretion of water and salt.

Pathophysiology of disorders of water balance

Most disorders of water balance are due to the inability of the kidney to conserve water – thus primary polyuria. In these cases polydipsia represents a compensatory mechanism to maintain total body fluids within normal limits. Much less frequently, polydipsia is primary with a compensatory polyuria to excrete the excess water load. Primary polyuria is either due to osmotic (solute) diuresis, ADH deficiency or renal insensitivity to ADH. Primary polydipsia, in turn, is caused by certain behavioural or neurological disorders with prolonged intake of large amounts of water resulting in renal medullary washout and the production of large amounts of dilute (SG < 1.005), solute-free urine.

Renal medullary hypertonicity is maintained by the efflux of large concentrations of sodium, chloride and urea from the loop of Henle and collecting ducts into the renal medullary interstitium. Loss of this osmotic gradient in, for example, cases of hypoadrenocorticism with chronic sodium wasting, results in inadequate urine concentration, despite the presence of adequate amounts of circulating ADH.

In many cases the pathophysiology of polyuria is multifactorial, or may be changed by complicating factors during the course of the disease. Liver failure, for example, results in decreased production of urea (thus causing decreased renal medullary hypertonicity) and increased levels of corticosteroids that inhibit the release of ADH (thus causing a degree of central diabetes insipidus).
Diagnostic approach

Signalment

Some causes of PU/PD are more prevalent in certain breeds: for example small terrier breeds are predisposed to Cushing’s disease, whereas Dobermann pinchers might suffer from chronic active hepatitis and older female dogs from anal sac adenocarcinoma, causing paraneoplastic hypercalcaemia and resultant PU/PD.

History

An accurate history is very informative and enables the clinician to distinguish in the first instance between polyuria and urinary incontinence, nocturia or pollakiuria. Nocturia (voluntary desire to urinate at night) may be found in older dogs with senile changes. Urinary incontinence typically presents in middle-aged, large breed, spayed bitches and is characterised by the passive leakage of urine whilst the bitch is lying down or sleeping. Pollakiuria (increased frequency of urination) is generally caused by disorders of the lower urinary tract that compromise the normal function or filling capacity of the bladder. Bear in mind that incontinence and pollakiuria can be exacerbated in polyuric dogs.

Further history should include questions relating to the dog’s general health, diet, appetite (dogs with diabetes mellitus and hyperadrenocorticism are often polyphagic), behavioural changes, reproductive abnormalities and importantly, recent or current drug administration (anticonvulsants and glucocorticoids can inhibit the release of ADH and diuretics such as furosemide can also cause polyuria).

If the history is inconclusive it is advisable that the owner attempts to measure the water intake at home for a few days. Upon return to the practice, the owner should also present the clinician with randomly collected urine samples so that the SG could be verified. It is unlikely that a dog is polyuric if the majority of its urine SG’s is above 1.030. It should also be borne in mind that the urine SG in the normal dog can range from 1.001 – 1.050 depending on physiological conditions and water intake.

Clinical examination

This is imperative for increasing or decreasing the index of suspicion for certain disorders. As such, dogs with diabetes insipidus or primary polydipsia are generally bright and alert, whereas dogs with Addison’s disease or pyometra are generally unwell.

The clinical examination should be thorough and systematic and include careful palpation of the abdomen that could reveal the following: The liver is often enlarged in dogs with diabetes mellitus, Cushing’s disease or hepatic neoplasia. The kidneys could be enlarged in conditions such as pyelonephritis or renal neoplasia and small and misshapen in chronic interstitial nephritis or congenital renal dysplasia. The uterus is often distended in cases of a closed-cervix pyometra. A pendulous abdomen is encountered frequently in dogs with Cushing’s disease.

Further pointers during the clinical examination could include peripheral lymphadenopathy (i.e. cases of multicentric lymphoma) or the presence of a bradycardia that could indicate hypoadrenocorticism or hypercalcaemia. The external genitalia should be examined for discharge (i.e. open cervix pyometra) or testicular atrophy (cases of Cushing’s disease). The detection of cataracts during ophthalmoscopic examination could point to diabetes mellitus, whereas thin, alopecic, non-elastic abdominal skin could be suggestive of hyperadrenocorticism.
Further diagnostic tests

Urinalysis

This is the most important initial step in the evaluation of PU/PD cases. Hypersthenuric urine (SG > 1.030) renders PU/PD very unlikely. The presence of constantly isosthenuric urine (SG 1.005 – 1.012) is highly suggestive of chronic renal failure. Hyposthenuric (SG < 1.005) urine is indicative of diabetes insipidus (either central of nephrogenic) or primary polydipsia, but importantly, imparts knowledge about the normality of the kidneys, i.e. it indicates that the renal tubules are able to actively dilute the glomerular filtrate and are thus functioning appropriately. Glucosuria significantly narrows the list of differential diagnoses. Urine culture should be considered, even when the urine sediment is unremarkable, because some cases of hyperadrenocorticism might have an impeded white cell response due to immunosuppression. Proteinuria, especially in the presence of dilute urine, indicates significant protein loss and is suggestive of glomerulonephritis.

Other tests

From hereon the clinician should perform the test that he/she thinks will yield the most information for the “diagnostic dollar” that the client provides. Many disorders will by now be ruled out or made very unlikely by the signalment, history, clinical examination and urinalysis.

A full blood count can increase the index of suspicion for pyometra or hyperadrenocorticism. A biochemical profile with electrolytes can be highly suggestive of renal failure, hypoadrenocorticism or hepatic disease. Abdominal radiographs and/or ultrasound may be indicated to evaluate the liver, kidneys, adrenals and uterus. Evaluation of the hypothalamic-pituitary-adrenal (HPA) axis with ACTH stimulation or low dose dexamethasone suppression testing should be performed if Cushing’s disease is suspected. If hypercalcaemia is detected, further tests to find a neoplastic process might include thoracic radiographs, lymphnode aspirates or bone marrow aspiration. After a thorough review of all test results, a cause would either be found or most causes would at least be ruled out.

If a diagnosis is still eluding the clinician a water deprivation test should be performed. The purpose of this test is to determine whether a dog can concentrate its urine in response to dehydration, i.e. whether it can release ADH and whether the kidneys are able to respond to this hormone. It is therefore important to note that this test is contraindicated in animals with renal failure. Longstanding cases of PU/PD may be complicated by renal medullary washout, rendering the kidneys unable to respond to ADH, even when they are normal. Therefore the test is often preceded by a gradual reduction in water intake over a few days. Thereafter water and food is withheld. The patient should be closely monitored (i.e. bodyweight, hydration status, serum urea and creatinine) and the test should be stopped if the patient appears dehydrated or has lost 5 % of its bodyweight. If the patient is able to concentrate its urine in response to water deprivation it most likely has psychogenic polydipsia. If it is still unable to concentrate after dehydration, administer exogenous ADH (DDAVP either i/m or intra-conjunctivally). If it is able to concentrate its urine, then it has central diabetes insipidus (CDI), if it is still unable to concentrate it has nephrogenic diabetes insipidus (NDI). Remember that primary NDI is a very rare diagnosis. Ensure, once again, that all the other causes of secondary NDI have been properly eliminated before confidently making the diagnosis.

The assessment of a random plasma osmolality could aid the differentiation between psychogenic polydipsia (which should have a serum osmolality below 280 nOsm/kg) and CDI or NDI (which should have serum
osmolalities above 305 nOsm/kg). The grey area of values between 280 and 305 mOsm/kg is unfortunately non-informative and could include a patient with any of the above-mentioned disorders.

Differential Diagnoses for Polyuria and Polydipsia

Primary polydipsia

- Behavioural (psycogenic)
- Fever
- Encephalopathy
- Pain
- Neurologic disorder

Primary polyuria

Osmotic diuresis
- Diabetes mellitus
- Primary renal glucosuria
- Fanconi’s syndrome
- Post-obstructive diuresis

ADH deficiency – Central diabetes insipidus (CDI)
- Congenital disorder
- Traumatic origin
- Neoplastic

Renal insensitivity to ADH - Nephrogenic DI (NDI)

Primary NDI

Secondary NDI
- Chronic renal failure
- Renal medullary washout
- Pyelonephritis
- Pyometra
- Liver disease
- Hyperadrenocorticism
- Hyperaldosteronism
- Hypoadrenocorticism
- Hypercalcaemia
- Hypocalcaemia
Hypokalaemia
Hyperviscosity and polycythemia
Drugs – phenobarbitone, furosemide, glucocorticoids
High salt diet

Further reading: