Approach to Polyuria and Polydipsia in the Dog

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INTRODUCTION

Polyuria and polydipsia (PU/PD) 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--termed 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 (antidiuretic hormone) 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 < 1.008), 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 can enable the clinician to distinguish in the first instance between polyuria and urinary incontinence, nocturia or pollakiuria. 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. Nocturia (voluntary desire to urinate at night) may be found in older dogs with senile changes. 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. Incontinence and pollakiuria can be exacerbated in any polyuric dog.

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 of 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 owners attempt to measure the water intake at home for a few days. Upon their return, the owners 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 SGs are above 1.030. Bear 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 happy, 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: an enlarged liver in dogs with diabetes mellitus; Cushing's disease or hepatic neoplasia; renomegaly in conditions such as pyelonephritis or renal neoplasia; small and misshapen kidneys 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 probably the most important initial step in the evaluation of PU/PD cases. Hypersthenuric urine (SG >1.030) makes PU/PD very unlikely. The presence of constantly isosthenuric urine (SG 1.008-1.012) is highly suggestive of chronic renal failure. Hyposthenuric (SG <1.008) 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 differential diagnoses. Urine culture should be considered, even when the urine sediment is unremarkable, as 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 could suggest glomerulonephritis.

Other Tests

From here on, the clinician should perform the test that she thinks will yield the most information for the 'diagnostic currency' that the client provides. At this stage, many disorders would have been ruled out or made very unlikely by the signalment, history, clinical examination and urinalysis.

A **full blood count** can increase the suspicion of pyometra or hyperadrenocorticism. A **biochemical profile** which includes electrolytes can be highly suggestive of renal failure, hypercalcaemia, hypokalaemia, hyper/hypoadrenocorticism or hepatic disease.

Abdominal radiographs/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, lymph node 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 contra-indicated 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, orally 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. Some authors suggest that the distinction between PPD and CDI can be made by measuring plasma osmolality which should be low in the former and high normal or high in the latter. Conversely, a DDAVP trial should show a quick response in CDI, while PPD cases are unlikely to respond in a meaningful way. Given the time, expenses, danger and frequent equivocal results obtained from water deprivation tests, these latter two approaches might be much more useful to the practitioner.

Differential Diagnosis for Polyuria and Polydipsia

Primary Polydipsia

- Behavioural (psychogenic)
- 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 diabetes insipidus (NDI)

Primary Nephrogenic Diabetes Insipidus (Rare)

Secondary Nephrogenic DI

- Chronic renal failure
- Renal medullary washout
- Pyelonephritis
- Pyometra
- Liver disease
- Hyperadrenocorticism
- Hypoadrenocorticism
- Hypercalcaemia
- Hypokalaemia
- Hyperviscosity
- Drugs--phenobarbitone, furosemide, glucocorticoids
- High salt diet

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SPEAKER INFORMATION

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