Polyuria and Polydipsia

A PowerPage Presented By

Polyuria (PU) and polydipsia (PD) are very common presenting signs in dogs, less common in cats. Some owners may only notice one of the two even when both are present. In some instances, PU/PD may need to be differentiated from incontinence since nocturia is common with both. In most cases, the primary disorder is one causing polyuria resulting in compensatory polydipsia to prevent dehydration. The major exceptions to this rule are psychogenic polydipsia and polydipsia secondary to hyperthermia. Many of the important individual causes of PU/PD have a dedicated PowerPage including diabetes mellitus, hyperadrenocorticism, hypoadrenocorticism, renal insufficiency, and hyperthyroidism. Therefore, instead of a detailed discussion of each disease, this PowerPage presents a logical approach to the evaluation and diagnosis of a dog or cat patient presenting with PU/PD.

Definitions

- Polydipsia - Increase from normal water consumption
  - Can be measured by owners over at least 48 hours
  - Normal is up to 50-60 ml/kg/day
  - **Polydipsia is defined as >100 ml/kg/day**

- Polyuria - Increase from normal urine output
  - Much more difficult to measure without hospitalization and catheter placement
  - Normal is < 50 ml/kg/day

Differential Diagnoses for PU/PD

This is a fairly complete list of differential diagnoses for an animal presenting for PU/PD:

- Common:
  - **Diabetes mellitus**
  - **Chronic renal insufficiency**
  - **Hyperadrenocorticism (Cushing's)**
  - **Neoplasia (especially lymphoma, anal sac adenocarcinoma, multiple myeloma, and pheochromocytoma)**
  - **Hypercalcemia**
  - **Diabetes insipidus (Central or nephrogenic)**
  - **Liver Failure**
  - **Hyperthyroidism (mainly cats)**
  - **Endotoxemia (most commonly pyometra, prostatic abscesses)**
  - **Hypoadrenocorticism (Addison's)**
  - **Iatrogenic (administration of steroids, diuretics, levothyroxine, high salt diet)**
  - **Pyelonephritis**

- Uncommon:
  - Psychogenic polydipsia
  - Hyperthermia, stress, exercise, heat
  - Fanconi's syndrome
  - Acromegaly
  - Primary renal glucosuria

- Hypokalemia
- Post obstruction diuresis after urethral blockage

Diagnostic Clues

Paying close attention to the additional information given to you about a patient may provide a key tip toward the diagnosis:

- Polyphagia
  - Suggests diabetes mellitus, hyperadrenocorticism (or iatrogenic steroid administration), or hyperthyroidism
- Weight loss
  - Not specific but could be seen with renal or hepatic failure, neoplasia, diabetes mellitus or hyperthyroidism most commonly
- Skin/coat changes
  - Suggestive of hyperadrenocorticism (dogs) or hyperthyroidism (cats)
- Vulvar discharge
  - Suggestive of pyometra
- Abdominal palpation abnormalities
  - Small kidneys, small or large liver
- Rectal exam
  - Mass suggestive of anal sac carcinoma
- Bilateral cataracts (dogs)
  - Suggests diabetes mellitus
- Lymphadenopathy
  - Suggestive of lymphoma or other neoplasia

Using Your Minimum Database to Maximum Yield

A minimum database should go a long way toward ruling in or out most causes of PU/PD:

- Complete blood count and biochemical profile:
  - Anemia
    - Not specific but may be seen with chronic renal insufficiency
  - Eosinophilia, lymphocytosis
    - Occasionally seen with hypoadrenocorticism
  - Blood glucose
    - Used to help rule in/out diabetes mellitus and differentiates diabetes mellitus from renal glucosuria
  - Serum calcium
    - Can rule in/out hypercalcemia
    - If elevated now need to investigate causes of hypercalcemia
  - Liver function parameters (BUN, albumin, glucose, cholesterol, bilirubin)
    - First 4 often decreased with liver failure, bilirubin elevated
  - Liver enzymes (ALT, ALP, AST, GGT)
    - Often elevated (especially ALP) with hyperadrenocorticism
    - Elevations are nonspecific and usually indicate ongoing damage in liver and may not be elevated in liver failure
  - Renal values (BUN, Creatinine)

  - Elevations suggest renal insufficiency, although prerenal azotemia must be ruled out with urinalysis

- Urinalysis:
  - Specific gravity **>1.035** should only be seen in PU/PD animals with marked glucosuria
  - Hyposthenuria (**urine SG <1.008**) makes renal insufficiency less likely
  - Glucosuria suggests diabetes mellitus or primary renal glucosuria. Absence largely rules them out

- Diagnostic imaging (Radiographs, Ultrasound)
  - Can be used to evaluate:
    - Adrenal gland size (increased with hyperadrenocorticism, decreased with hypoadrenocorticism)
    - Evidence of neoplasia
    - Liver and kidney size
    - Evidence of pyometra, prostatomegaly

- Additional screening tests that may be indicated:
  - T4 levels (cats)

Further Diagnostic Workup

If all of the above tests are normal and you have acquired a reasonably accurate history to know the animal does not have an iatrogenic cause or post obstructional diuresis, you have effectively ruled out all of the causes listed above except:

- Diabetes insipidus
- Hyperadrenocorticism (very difficult to rule out)
- Hypoadrenocorticism
- Neoplasia
- Psychogenic polydipsia

Depending on what other clinical signs, if any, are present, a logical plan towards a diagnosis might be:

1) Rule in/out hyperadrenocorticism and/or hypoadrenocorticism with an ACTH stimulation test and/or a low dose dexamethasone suppression test.
2) While you can rarely entirely rule out neoplasia, a thorough physical exam and lack of identifiable abnormalities on your other diagnostic tests including normal calcium levels makes it much less likely as a cause of PU/PD.
3) If everything is still normal, you have narrowed it down to diabetes insipidus versus psychogenic polydipsia.
4) To assess for central diabetes insipidus (CDI), a trial therapy with vasopressin (dDAVP) can be performed. If PU/PD has been longstanding, it may take several days to a week to overcome medullary washout of the kidneys before an effect is seen. Advanced imaging of the brain/pituitary is an expensive but reasonable way to further assess the possibility of CDI.
5) To rule in psychogenic polydipsia, a modified water deprivation test (WDT) can be performed. Many internists feel this test is overused in some practices and can be a risk to the patient if not performed very carefully. It should only be performed once a thorough search to identify other causes has been exhausted. It is unlikely that you would be asked about the details of performing a WDT, but briefly:

1. Deliberately withhold water and food and frequently monitor hydration status and USG to detect if animal can concentrate urine (>1.030).
2. If it can concentrate urine, this is consistent with psychogenic polydipsia. If not, it is consistent with DI.
3. You should give dDAVP to assess whether the kidneys can respond. If urine becomes concentrated, this confirms CDI. As stated above, it is possible that an immediate response to dDAVP may not occur due to medullary washout.

- Primary nephrogenic DI is thought to be quite rare. A WDT should NOT be performed in animals that are ill, azotemic, dehydrated, or hypercalcemic!