Improve Veterinary Education

Laboratory Evaluation in the Vomiting Dog and Cat: Interpreting Haematology and Biochemistry in Clinical Context

This handout explains how laboratory data support assessment of vomiting in dogs and cats, helping differentiate gastrointestinal from extra-gastrointestinal causes. It reviews key haematology, biochemistry, electrolyte and pancreatic markers, integrated with history, examination, urinalysis and imaging.

Clinical purpose of laboratory evaluation in vomiting patients

Laboratory testing in vomiting dogs and cats is most valuable when it is used to distinguish primary gastrointestinal disease from systemic or extra-gastrointestinal causes of vomiting. In many cases, bloodwork provides the earliest objective indication that the vomiting is driven by gastrointestinal pathology rather than renal, hepatic, endocrine, infectious, inflammatory or other systemic disease. Laboratory findings also contribute to assessment of severity, but they must always be interpreted in conjunction with the physical examination to reach clinically sound conclusions.

The minimum diagnostic database and baseline investigations

In veterinary medicine, the “minimum database” refers to a standardised set of diagnostic tests performed alongside a physical examination to provide a rapid and consistent baseline assessment. Although the precise components should be tailored to the individual patient’s history and clinical findings, several elements are routinely appropriate in vomiting cases.

A practical minimum database includes a complete blood count to assess red blood cells, white blood cells and platelets, and a serum chemistry profile that includes renal values, hepatic values, glucose and electrolytes. In vomiting patients, it is particularly important to include electrolytes and to obtain urine assessment. Where there are no urinary signs, a full urinalysis may be substituted by urine specific gravity, but urine evaluation remains clinically important for interpretation of renal parameters and hydration status. In cats, FeLV/FIV status should be known, and in senior cats with vomiting, total T4 is an appropriate addition to the minimum diagnostic evaluation. In some settings, particularly emergency practice, abdominal radiographs

and measurement of blood pressure on intake are also considered part of the baseline assessment.

Haematology clues: red blood cells and haemoconcentration

Red blood cell parameters can immediately shift diagnostic reasoning. Anaemia commonly suggests chronic disease and/or blood loss, including gastrointestinal ulceration or parasitism, but it may also indicate haemolysis. Dogs with immune-mediated haemolytic anaemia may present with vomiting and anorexia, and anaemia in a vomiting patient should prompt consideration of both gastrointestinal and systemic differentials.

When anaemia is present, it is clinically useful to distinguish regenerative from non-regenerative patterns, while remembering that bone marrow response is delayed. In acute haemorrhage, such as bleeding from a splenic mass, anaemia may initially appear non-regenerative because the marrow has not yet mounted a reticulocyte response.

At the opposite end of the spectrum, haemoconcentration should be evaluated through integration of laboratory data with the physical examination and urine specific gravity. Haemoconcentration may reflect dehydration, but it can also occur in other clinical contexts. In haemorrhagic gastroenteritis, for example, patients may have markedly increased haematocrit (commonly in the range of approximately 60–65%) and be profoundly dehydrated. In this scenario, tacky mucous membranes and increased skin turgor would support dehydration as the driver of haemoconcentration. If a patient has a similar haematocrit but is febrile, tachycardic and markedly painful on abdominal palpation, the same numerical finding becomes more concerning for severe systemic disease such as sepsis, severe acute pancreatitis or other causes of an acute abdomen.

Haematology clues: white blood cells, inflammation and systemic risk

White blood cell changes can further refine differentials. Inflammatory leukograms are typically characterised by neutrophilia and may include a left shift. A left shift does not, in itself, prove infection; it can be seen with inflammatory processes such as enteritis and pancreatitis, and it may also accompany neoplastic disease. Conversely, severe inflammatory disease states such as pyometra and septic abdomen may be associated with pronounced inflammatory leukograms.

Leukopenia is an important red flag in a vomiting patient, raising concern for sepsis and, depending on species and context, viral enteric disease such as canine parvovirus infection or feline panleukopenia. This pattern is often a haematological clue indicating the need for more thorough and potentially more invasive diagnostics and escalation of the diagnostic plan.

Eosinophilia may be associated with parasitism, allergic disease, hypereosinophilic syndromes, and neoplastic processes such as lymphoma. In addition, hypoadrenocorticism (Addison's disease) may present with eosinophilia. Clinically, the absence of an expected inflammatory leukogram in a severely unwell vomiting dog, particularly when accompanied by hyperkalaemia, should increase concern for Addison's disease and prompt appropriate endocrine testing.

Chemistry profile patterns suggesting gastrointestinal disease

Unlike renal disease, where creatinine and SDMA often serve as prominent biochemical indicators, there is no single marker that reliably identifies intestinal disease. Dogs and cats with chronic gastrointestinal disease and chronic vomiting may have entirely normal bloodwork. Nevertheless, several recurring biochemical patterns can provide useful clues.

Hypoalbuminaemia is a common finding that can reflect gastrointestinal disease, particularly protein-losing enteropathy in chronic cases where intestinal mucosal injury leads to loss of albumin into the gastrointestinal tract. This may occur with chronic enteritis, inflammatory bowel disease, intestinal lymphoma, and fungal disease such as histoplasmosis, and can also be seen with severe gastrointestinal bleeding. Malnutrition associated with chronic vomiting, chronic diarrhoea or parasitic infection, particularly in young animals, may contribute to reduced albumin.

When hypoalbuminaemia is suspected to be gastrointestinal in origin, supporting biochemical clues include globulins that are normal or low and cholesterol that is often low, particularly in protein-losing enteropathy. The combination of low albumin and low cholesterol is a classic pattern in protein-losing enteropathy. Severe lymphangiectasia, whether primary or secondary, may produce particularly low cholesterol and albumin due to severe malabsorption and maldigestion. However, low cholesterol is not specific to gastrointestinal disease and may be absent in many patients with chronic enteropathy.

A distinct gastrointestinal clue is markedly increased blood urea nitrogen (BUN) with normal creatinine, often with BUN values in the range of approximately 70–100 in the presence of a normal creatinine. This pattern is consistent with gastrointestinal bleeding, as digestion of blood increases urea production and elevates BUN. In such cases, melaena may be detected on rectal examination, and urine specific gravity should be normal to high if dehydration is contributing. Faecal occult blood testing can be used to detect blood not visible in the stool, although false positives are common and dietary factors can substantially affect results, limiting its routine utility.

It is also important to recognise that a high BUN with a comparatively lower creatinine can occur in advanced renal disease, particularly in cats, and in cachectic patients with low muscle mass, where creatinine may be misleadingly low. Physical examination findings and urine specific gravity are therefore essential for interpretation. In patients with high BUN and normal creatinine, rectal examination, assessment of hydration, and reassessment of the complete blood count for thrombocytosis may provide further context, as some patients may have increased platelets.

Mild liver enzyme elevations and reactive hepatopathy

Mild elevations in liver enzymes are frequently secondary rather than primary in vomiting patients. A mild increase in alanine aminotransferase (ALT) and alkaline phosphatase (ALP) does not necessarily indicate primary hepatobiliary disease; it is commonly associated with chronic gastrointestinal inflammation and can occur in chronic enteropathies and pancreatic inflammation. In these reactive patterns, total bilirubin is often normal, but mild liver enzyme elevations may be present. This phenomenon is often described as reactive hepatopathy, reflecting extrahepatic disease driving hepatic biochemical changes. In contrast, primary hepatocellular injury may present with more substantial increases in ALT and ALP and may be accompanied by increased total bilirubin.

Azotaemia and the importance of urine assessment

Azotaemia in a vomiting patient may be gastrointestinal in origin when dehydration produces pre-renal azotaemia, emphasising the value of urine specific gravity in interpretation. However, azotaemia may also reflect intrinsic renal failure, and post-renal obstruction can also produce azotaemia. Integrating renal parameters, urine concentration, hydration status and clinical context is therefore critical before attributing vomiting to primary gastrointestinal disease.

Electrolyte patterns and lesion localisation

Electrolyte assessment should be part of the minimum database in vomiting patients. Hypochloraemia should trigger consideration of an outflow obstruction, particularly when the obstruction is proximal. Gastric outflow obstruction, including pyloric outflow disease, tends to produce low chloride. Similarly, proximal intestinal foreign bodies, such as duodenal foreign bodies, may be associated with hypochloraemia.

Hyponatraemia can be seen in vomiting, diarrhoea, Addison's disease and abdominal effusions. Hypokalaemia is clinically important to identify and correct, particularly in anorexic patients, because hypokalaemia can contribute to intestinal ileus.

Hyperkalaemia requires prompt localisation and clinical correlation. In cats with high potassium, urinary obstruction should be a primary consideration, with urethral obstruction being especially common. Physical examination is pivotal. In dogs, hyperkalaemia can be associated with obstruction (including urethral or ureteral obstruction), renal failure, and endocrine disease such as Addison's disease. Pseudohyperkalaemia may occur due to severe haemolysis as a laboratory artefact or, in some cases, thrombocytosis. The classic electrolyte pattern in Addisonian dogs is hyperkalaemia with hyponatraemia.

Pancreatic markers and interpretation alongside imaging

Canine and feline pancreatic lipase assays are commonly used as pancreatic markers. These may be assessed as in-clinic SNAP tests yielding a normal/abnormal result, or as quantitative laboratory assays that provide a numerical value, which can be useful for estimating degree of elevation. Pancreatic lipase is typically increased in acute pancreatitis, but elevations may also occur secondary to other inflammatory or systemic disease. For example, increased pancreatic lipase may be seen in patients with septic abdomen, intestinal obstruction, or bacterial cholangiohepatitis, and it may be influenced by concurrent gastrointestinal inflammation or renal disease.

Interpretation should therefore be cautious and should incorporate imaging and overall clinical assessment. Mild increases are often non-specific, whereas marked increases tend to have higher specificity, particularly when ultrasound demonstrates compatible findings and excludes foreign bodies or alternative causes of acute abdomen. Classic sonographic examples include an enlarged pancreas that appears hypoechoic, with hyperechoic, inflamed peripancreatic fat or mesentery.

Integrating laboratory findings with clinical context: pre-renal azotaemia, obstruction and case interpretation

A core principle throughout vomiting work-ups is that history, physical examination and laboratory trends must be integrated before concluding that vomiting is primary gastrointestinal disease or secondary to systemic pathology. For instance, a vomiting dog with a foreign body may have increased renal values, including increased creatinine, alongside a high urine specific gravity consistent with pre-renal azotaemia due to dehydration. Similarly, a cat presenting with vomiting or straining to urinate may show azotaemia that is pre-renal in nature because of obstruction; after relief of the obstruction, renal parameters can normalise, indicating that the cat was not in end-stage renal failure.

A representative clinical case involved a cat presenting through emergency with acute vomiting after previously being healthy. Sedation (gabapentin) was used to facilitate examination. The chemistry profile demonstrated increased creatinine and a markedly increased BUN, while phosphorus was not increased to the degree expected in end-stage renal failure. Importantly, the patient was hyperkalaemic. In a cat with increased renal values and hyperkalaemia, the immediate clinical priority is to palpate the bladder and assess for urinary obstruction. In this case, the bladder was normal in size, prompting imaging. Ultrasound demonstrated an enlarged kidney with a markedly distended renal pelvis consistent with hydronephrosis, and a ureteral stone was identified. The patient underwent surgery and a subcutaneous ureteral bypass was placed. Within 24 hours, creatinine decreased from 11 to 3, supporting the interpretation that vomiting was driven by dehydration and obstruction rather than irreversible end-stage renal disease. This case also illustrates the value of repeating laboratory tests after rehydration to separate dehydration artefacts from true metabolic pathology.

A further practical example addressed point-of-care blood glucose assessment in haemoconcentrated patients. When packed cell volume is very high, the plasma fraction in whole blood is reduced, and handheld glucometer readings may be falsely low. A low glucose measured under these circumstances does not necessarily indicate sepsis, and confirmation using a chemistry analyser or laboratory methodology is advisable.

Additional diagnostics to refine chronicity and systemic differentials

Beyond the minimum database, additional diagnostics may be indicated depending on clinical suspicion. In patients with azotaemia or concern for urinary tract involvement, full urinalysis with consideration of urine culture is beneficial, particularly when pyuria is present or when there is concern for renal infection.

Markers of chronic gastrointestinal disease include cobalamin and folate. Folate is absorbed in the duodenum, whereas cobalamin is absorbed in the ileum, and these parameters can be useful in chronic enteropathies. In a chronically vomiting cat with low cobalamin, the finding may suggest a long-standing process of sufficient duration to produce vitamin deficiency. Trypsin-like immunoreactivity (TLI) is a useful marker for exocrine pancreatic insufficiency.

When clinical and laboratory patterns raise concern for Addison's disease, ACTH stimulation testing is indicated. A representative scenario described a vomiting dog with a chronic history that appeared profoundly unwell and laterally recumbent. The minimum database suggested haemoconcentration, described as a packed cell volume of 58.5%, with increased creatinine reported as 277. Electrolytes were notable for potassium of 5.9 with low sodium and mild

hypercalcaemia, a combination that is classically suspicious for hypoadrenocorticism when urinary output is otherwise normal. In such a case, ACTH stimulation testing was pursued, and cortisol concentrations were reported as less than 0.2 both before and after ACTH administration, supporting a diagnosis of Addison's disease.

Key take-home messages

Bloodwork should always be interpreted in context and never in isolation. Laboratory assessment supports differentiation of primary gastrointestinal causes of vomiting from secondary systemic causes, including renal, hepatic and endocrine disorders. Recognising electrolyte patterns can help localise lesions, such as hypochloraemia suggesting proximal gastric loss or outflow obstruction and hyperkalaemia suggesting urinary obstruction or Addison's disease. Pancreatic markers should be used judiciously and interpreted alongside imaging to minimise misdiagnosis. Finally, many stable gastrointestinal causes of vomiting may present with normal bloodwork, and the absence of abnormalities can itself be clinically informative when integrated with the patient's overall stability and examination findings.