Daisy: An IDEXX Cystatin B Test case study

DAISY CASE SUMMARY

+ Presented for post-discharge recheck following hospitalization for vehicular trauma
+ Although Daisy appeared stable and was recovering well, her IDEXX Cystatin B Test result indicated evidence of active kidney injury
+ Readmitted for hospitalization for active kidney injury with a readmission plan to administer IV fluids, monitor diagnostics every 24 hours, and recheck her cystatin B concentration in 3 days
+ Daisy's condition continued to improve, and she was discharged after 3 additional days of hospitalization

Patient

Daisy, 3-year-old, spayed female greyhound

Presenting reason

Post-discharge recheck following hospitalization for vehicular trauma

History

Daisy returned to the veterinarian for a recheck visit after being hospitalized for injuries suffered when she was struck by a vehicle approximately 1 week earlier. Daisy's initial injuries included pneumothorax and superficial abrasions. tFAST and aFAST (thoracic and abdominal focused assessment with sonography in trauma) revealed pulmonary contusions and hemoabdomen secondary to a lacerated spleen. Her bladder was intact. Her laboratory results revealed mildly elevated glucose and blood urea nitrogen (BUN), as well as an elevated alanine aminotransferase (ALT), a mildly decreased phosphorus, and a slightly low lymphocyte count, all consistent with trauma. The rest of Daisy's laboratory results were within normal limits. Because Daisy's blood work was run in-clinic, IDEXX Cystatin B Test was not included in her initial results. Daisy was treated for her injuries and following 3 days of hospitalization, her clinical signs and laboratory results improved, and she was discharged to her owners. Daisy's owners were instructed to restrict Daisy's activity, complete her course of oral medications as directed, and return for recheck blood work in 2–3 days.

Physical examination upon recheck 3 days post-discharge

Daisy was bright, alert, and responsive. Her owners stated she had been quieter than usual the first day home, but since then was doing well. Her appetite was good, but her owners thought she might be drinking a little more than usual. Her discharge medications included carprofen and gabapentin for analgesia, and amoxicillin/clavulanic acid for her abrasions. Her temperature, pulse, and respiration were all within normal limits, and the remainder of her physical exam was unremarkable.

Diagnostic plan

A brief blood screen and urinalysis were ordered, and while Daisy's liver enzymes remained slightly elevated, they had continued to improve since her initial injury. Her BUN also remained slightly elevated, and all other renal functional biochemistry markers were within reference limits. Daisy's urinalysis revealed dilute urine with a quiet sediment. An IDEXX Cystatin B Test was performed for the first time and was > 500 ng/mL. A cystatin B concentration that is > 100 ng/mL indicates that active kidney injury may be present. Although Daisy had been recovering well following her recent discharge from the hospital, her owners elected to admit her for renal monitoring and urine output due to concerns of subclinical kidney injury.

Upon admission, Daisy was placed on intravenous maintenance fluid therapy and was scheduled to have her functional kidney values with SDMA, electrolytes, and urine output (UOP) monitored every 24 hours. She was also scheduled to have her cystatin B concentration rechecked within 2 days. During her first overnight, Daisy's UOP decreased to below normal at 0.37 mL/kg/h (normal is 1–2 mL/kg/h), and she became tachycardic. Her fluid rate was increased and monitoring continued. If her condition had deteriorated in the face of fluid therapy, diuretics would be considered. Over the next 24 hours, Daisy's liver values and BUN continued to improve, and her urine output normalized. On day 3, Daisy's recheck cystatin B concentration was < 50 ng/mL (range 0–100 ng/mL), indicating resolution of her underlying kidney injury. Daisy was discharged from the hospital with instructions to develop a follow-up plan with her regular veterinarian.

Diagnosis

Hospital-acquired acute kidney injury secondary to trauma

Discussion

Acute kidney injury (AKI) is a leading cause of mortality in dogs with case fatality rates as high as 60% despite advances in management, including renal replacement therapy. Until recently, hospital-acquired AKI, as defined by an increase in serum creatinine, was not considered a leading etiology of AKI in veterinary medicine. However, more sensitive criteria, increased awareness, and the development of guidelines have led to higher prevalence and awareness of hospital-acquired AKI in animals.[1] The term "acute kidney injury" is defined as an abrupt decrease in kidney function, and it has largely replaced the term "acute renal failure" (ARF) to better reflect the broad spectrum of changes seen with acute renal pathology.[2,3]

Acute kidney injury can be divided into 4 phases: initiation, extension, maintenance, and recovery.[3] It is not unusual for clinical signs or laboratory abnormalities to be absent during the first 2 phases, making early diagnosis of kidney injury challenging for the veterinarian. Until now, veterinarians have relied upon surrogate markers of glomerular filtration rate (GFR), such as creatinine and SDMA, combined with urinalysis and urine output to describe kidney injury in their patients. Reliance upon azotemia or decreased urine output to identify patients with kidney injury can result in delayed treatment potentially resulting in irreversible changes in kidney function.

The International Renal Interest Society (IRIS) has devised a grading system for AKI that reflects the dynamic nature of kidney injury.* This system provides guidance on earlier recognition, appropriate therapeutic intervention, and prognosis for patients diagnosed with kidney injury. Changes in creatinine and urine output in response to fluid therapy or renal replacement therapy have been the cornerstones of monitoring response to treatment in cases of AKI. Kidney injury biomarkers have been effective in identifying subclinical injury in human medicine, but until now, they have not been available to veterinarians. The IDEXX Cystatin B Test is the first kidney injury biomarker that has been validated in both dogs and cats for detecting kidney injury at the cellular level.

The IDEXX Cystatin B Test is a urine-based test that provides quantitative results to veterinarians when kidney injury may be present. Increased concentrations of cystatin B in the urine indicate active kidney injury and can help detect acute kidney injury—even in patients where clinical signs are not apparent or functional markers like creatinine or SDMA remain unchanged.[4-6] Cystatin B may also help distinguish stable from progressive chronic kidney disease in dogs diagnosed with early chronic kidney disease.[7] Cystatin B testing is indicated in unwell dogs and cats as kidney injury may be present even in cases with nonrenal disease. Additionally, cystatin B monitoring is recommended in patients diagnosed with chronic kidney disease (CKD), as cystatin B may identify progression even in patients with stable functional markers.

The IDEXX Cystatin B Test is offered globally through IDEXX Reference Laboratories. The reportable range for the test is 50–2,500 ng/mL, and cystatin B concentrations ≥ 100 ng/mL indicate an increased risk of kidney injury. Animals without kidney injury are expected to have concentrations that are < 100 ng/mL.

The IDEXX Cystatin B Test was validated in canine and feline urine by evaluating precision, accuracy, potential sample interferants, and sample stability on multiple lots across a full range of expected cystatin B concentrations.[8]

*Complete IRIS AKI staging guidelines are available at iris-kidney.com.

References

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