RED LIGHT, GREEN LIGHT
PEER REVIEWED

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Acute Liver Disease in a Dog

FRASIER, A 5-YEAR-OLD NEUTERED CHIHUAHUA, presented for acute-onset epistaxis and hemorrhagic diarrhea. The patient was dull and dehydrated, and generalized abdominal pain was noted on physical examination. CBC and serum chemistry panel results revealed moderate anemia, mild thrombocytopenia, marked ALT elevation, panhypoproteinemia, hyperphosphatemia, and moderate azotemia. Urine specific gravity was 1.013, and PT/PTT were prolonged. Hepatic dysfunction was confirmed on in-house ammonia tolerance testing. The dog's clinical coagulopathy was suspected to result from acute liver failure, which has been linked to spontaneous bleeding.[1,2] Abdominal ultrasonography revealed bilateral hyperechoic renal cortices and a large hypoechoic liver, and FNA was performed with a 22-gauge needle[3,4]; cytology suggested hepatocellular necrosis. Leptospirosis, fungal disease, toxin ingestion, cholangiohepatitis, and neoplasia were among the primary differentials for presumptive acute liver failure in this dog.[4]

Which of the following drugs would be appropriate for this patient?

Based on the information provided, how would you grade the following drugs and why?

Turn the page and compare your results ▶

RED = do not use YELLOW = proceed with caution GREEN = safe

	RED	YELLOW	GREEN
Ampicillin–sulbactam			
Buprenorphine			
Carprofen			
Chlorpromazine			
Famotidine			
Maropitant			
N-acetylcysteine			
Phytonadione (vitamin K1)			
Prednisone			
Sulfamethoxazole–trimethoprim			

FNA = fine-needle aspiration, PT = prothrombin time, PTT = partial thromboplastin time

Did you answer?

The following represents the best responses based on drug metabolism, pharmacokinetics, species, diagnostic differentials, clinical and laboratory data, and other pertinent findings.

Ampicillin–sulbactam

CORRECT RESPONSE

Antibiotic therapy for possible leptospirosis while awaiting results of polymerase chain reaction (PCR) testing and a microscopic agglutination test (MAT) is warranted. Penicillins and their derivatives are the preferred treatment to eliminate leptospiremia, but they do not clear the carrier state.[3] Injectable penicillins are safe and often considered early in therapy, particularly for nauseated patients unable to tolerate oral antibiotics. There is no evidence of a link between this drug and liver injury in animals; however, cholestatic hepatitis has been reported as a rare adverse reaction in humans.[4]

Buprenorphine

CORRECT RESPONSE

Opioid analgesics are indicated for this patient's abdominal pain. Buprenorphine causes less respiratory depression and sedation than other injectable opioids and is less likely to compromise monitoring for hepatic encephalopathy.[5]

Carprofen

CORRECT RESPONSE

Pain control is necessary for this patient, but NSAIDs should be avoided in the face of dehydration and hypovolemia because of the risks for nephrotoxicity and GI ulceration.[6] Carprofen has been shown to cause rare idiosyncratic hepatotoxicity and thus should be avoided in patients with liver disease.[5]

Chlorpromazine

CORRECT RESPONSE

Antiemetic medications are indicated in patients with severe GI signs, even without vomiting. However, because of the risks for hypotension and decreased perfusion pressure associated with phenothiazine use, phenothiazines should be administered only after correction of dehydration.[5] Lower doses may be required in patients with hepatic dysfunction.[5] When phenothiazines are combined with opioids such as buprenorphine, CNS depression may occur more frequently.[5] Chlorpromazine should be used with caution in this patient and only after rehydration.

Famotidine

CORRECT RESPONSE

GI ulceration risk increases with liver failure because of decreased gastrin and histamine metabolism and compromised mucosal blood flow caused by

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potential portal hypertension.[3] Famotidine is a safe H_2-receptor antagonist that has no significant drug interactions.[5]

Maropitant

CORRECT RESPONSE

Maropitant is a safe, approved antiemetic shown to reduce visceral pain with typically minimal adverse effects. However, it should be used with caution. Because it is metabolized by the liver, dose reductions (either lower dose or prolonged interval) should be considered in patients with hepatic dysfunction.[5]

N-acetylcysteine

CORRECT RESPONSE

Acute liver failure is frequently caused by toxins such as xylitol and aflatoxins. *N*-acetylcysteine reduces hepatocyte damage and restores glutathione by acting as a thiol donor, reducing damage caused by free radicals formed from various hepatotoxins.[3,6] Because hepatotoxicity is a likely diagnosis, this patient would probably benefit from this drug's antioxidant properties.

Phytonadione (vitamin K1)

CORRECT RESPONSE

Acute liver failure leads to decreased production and increased consumption of clotting factors.[1] Parenteral administration of vitamin K1 is indicated for this patient's clinical hemorrhage caused by prolonged clotting times.[1,3,6]

Prednisone

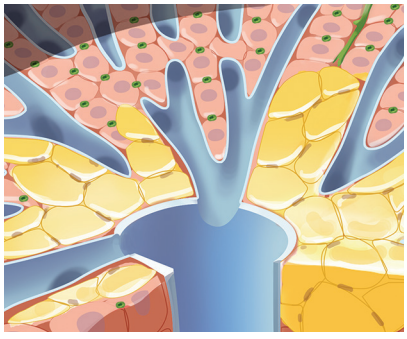
CORRECT RESPONSE

Indications for steroid use in patients with liver failure are limited to few diseases, including lymphocytic–plasmacytic cholangiohepatitis.[3] Glucocorticoids should not be used unless a definitive diagnosis confirms their need, as they exacerbate underlying infectious diseases (eg, leptospirosis), worsen hepatic encephalopathy, and increase risk for gastric ulceration.[3]

Sulfamethoxazole–trimethoprim

CORRECT RESPONSE

Sulfonamide antibiotics can cause severe, potentially fatal idiosyncratic hepatic necrosis.[5] This drug combination should not be used in patients with acute liver failure.[5,6]



▲ *Front cover artwork by Kip Carter, MS, CMI*

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