Coagulation abnormalities were studied in 45 cats with liver disease, including neoplasia ($n = 9$), inflammatory ($n = 12$), hepatic lipidosis ($n = 13$), and other degenerative diseases ($n = 11$). Parameters included platelet count; prothrombin time (PT); activated partial thromboplastin time (APTT); thrombin time; fibrinogen concentration; activities of antithrombin, protein C, plasminogen, and $\alpha_2$-plasmin inhibitor; $D$-dimer concentration; and activity of factors (F) II, V, VII, X, and XIII. One or more coagulation abnormalities were found in 44 of the 45 cats.

Of the global coagulation tests, prolonged APTT was present in 40% of cats. PT was prolonged in 18% of cats and shortened in 9%. FXIII was the most commonly decreased clotting factor, seen in 78% of cats with neoplasia, 75% with inflammatory liver disease, 31% of cats with hepatic lipidosis, and 64% of cats with degenerative liver disease. Fibrinogen was elevated in 36% of all cats and 67% of cats with hepatic inflammation. Compared with controls, fibrinogen was significantly increased in cats with hepatic neoplasia, inflammation, and lipidosis. FV activity was increased in 40% of all cats and 54% of cats with hepatic lipidosis. Protein C activity was below normal reference values in 44% of all cats and 58% of cats with inflammatory liver disease. Forty percent of all cats had increased $\alpha_2$-plasmin inhibitor activity. Increased $D$-dimer concentration was present in $\sim$50% of all cats and 83% of cats with inflammatory liver disease.

**Commentary**

Previous studies have shown that mainline coagulation tests (PT/APTT) often fail to portray the true disposition of feline liver patients. Vitamin-K supplementation will not assist because FII, FV, FVII, and FX are unaffected. In this study, cats with severe hepatic inflammatory diseases were found to have high levels of $D$-dimers, suggesting that some may be driven into disseminated intravascular coagulation by inflammatory mediators. Other patients, particularly those with hepatic lipidosis, had deficiencies of FXIII, which is routinely unmeasured but can lead to spontaneous bleeding. Another finding was that regulatory molecules (eg, protein C) are inhibited in many of these patients. A standard approach to coagulopathies can miss the mark; for patients with inflammatory hepatic disease, testing for $D$-dimers and fibrin degradation products as well as thromboelastography may be better first-tier diagnostics than those reliant on the clotting cascade.—Ewan Wolff, DVM

**Source**


**For More**

See Current Thoughts on Coagulopathy Testing by Drs. Deborah Silverstein & Lindsay Kellett-Gregory at cliniciansbrief.com/current-coagulopathy-testing