The Pharma Innovation Journal 2021; SP-10(7): 424-425

www.ThePharmaJournal.com

The Pharma Innovation

ISSN (E): 2277- 7695
ISSN (P): 2349-8242
NAAS Rating: 5.23
TPI 2021; SP-10(7): 424-425

www.thepharmajournal.com
Received: 25-04-2021
Accepted: 27-05-2021

RKR Sangamitra
Ph.D. Scholar, Department of Veterinary Physiology, Madras Veterinary College, TANUVAS, Chennai. Tamil Nadu, India

G Suganya
Assistant Professor and Head, Department of Veterinary Physiology and Biochemistry, Veterinary College and Research Institute, TANUVAS, Theni, Tamil Nadu, India

V Leela
Professor and Head, Department of Veterinary Physiology, Madras Veterinary College, TANUVAS, Chennai. Tamil Nadu, India

M Balagangadhara Thilagar
Assistant Professor, Department of Veterinary Clinical Medicine, Madras Veterinary College, TANUVAS, Chennai. Tamil Nadu, India

Corresponding Author:
RKR Sangamitra
Ph.D. Scholar, Department of Veterinary Physiology, Madras Veterinary College, TANUVAS, Chennai. Tamil Nadu, India

Activated partial thromboplastin time: An indicator for coagulation disorder in dogs

RKR Sangamitra, G Suganya, V Leela and M Balagangadhara Thilagar

Abstract
The study was conducted to evaluate the Activated thromboplastin time in dogs affected with various hepatic disorders. The study group consisted of control and experimental dogs. The experimental dogs further classified into biliary, neoplastic and parenchymal source of hepatic dysfunction based on ultrasonography. The study was conducted for a period of one year and the citrated plasma sample collected was evaluated for activated thromboplastin time. APTT values in dogs with parenchymal, neoplastic and biliary disorders showed significant increase (P<0.01) when compared to the control group of dogs. Alteration of APTT values in the experimental group indicates significant alteration in the coagulation mechanism in dogs affected with hepatic dysfunction of different etiology. Thus, the results of this study suggest that dogs affected with parenchymal, biliary and neoplastic type of hepatic disorders showed significant prolongation in APTT indicating defect in the coagulation cascade of both intrinsic and common pathway resulted in impaired clotting.

Keywords: hepatic disorder, haemostasis, APTT, canines

Introduction
Hepatic physiology is closely related to hemostasis. The hepatic parenchyma is the site of synthesis of most of the clotting factors which are involved in the coagulation cascade to maintain hemostasis. They are also associated with the regulation and also proper removal of activated clotting and fibrinolytic factors of coagulation (Prater, 2000; Niles *et al*., 2001) [9, 10]. Coagulation abnormalities in hepatic disorders occurs due to improper functioning of liver, impaired vitamin k synthesis and defect in clotting factors synthesis. Patients with hepatic insufficiency showed disseminated intravascular coagulopathies which may leads to bleeding conditions (Webster and Centre, 1995; Mijovski, 2019) [6, 7]. Estimation of Coagulation parameters has prognostic significance in acute and chronic liver dysfunctions in both human and Veterinary patients (Shih *et al*., 2007) [12]. Activated partial thromboplastin time is one of the screening test done to identify the coagulation changes in dogs affected with hepatic disorders (Geschen, 2009; Sumathi *et al*., 2012 and Lakshmi *et al*., 2018) [13]. Hence the study is conducted to measure the APTT in dogs with hepatic disorders of various etiology.

Materials and Method
Sample collection
The study was conducted in dogs presented to Madras veterinary college teaching hospital clinical complex in the year 2019. Twenty apparently healthy dogs formed the control group and twenty dogs showing symptoms of ascites, pectechial or echimotic haemorhage, jaundice where selected and further classified as parenchymal, biliary and neoplastic disorders as per WVASA guidelines formed the experimental group. Citrated plasma was collected from the dogs selected for the study by centrifugation at 3000 RPM for 30 minutes and APTT was measured with two hours of sample collection.

Methodology
APTT was estimated using commercial kits supplied by Agappe Diagnostics as per the standard protocol (Biggs and Macfarlane, 1962) [1]. Reagent 1 ($CaCl_2$) was pre-warmed at 37 ˚C prior to use. First one hundred microliter of test plasma was pipetted into a test cuvette and one hundred microliter of pre-warmed reagent 2 (APTT reagent) was pipetted into the test cuvette and the contents were incubated at 37 ˚C for 3 minutes. One hundred microliter of pre-warmed reagent 1 ($CaCl_2$) was added into the test cuvette. Immediately after addition of reagent 1, the clotting time in seconds was recorded using semi-automated coagulation instrument (Mispa Clog).

Result

The mean ± SE values of APTT in the control and experimental groups are presented in Table. The mean ± SE values of APTT in control group was 35.45 ± 1.33 seconds and the mean ± SE values of APTT in parenchymal, neoplastic and biliary disorders of liver disease dogs were 130.22 ± 20.79seconds, 156.39 ± 47.60seconds and 125.69 ± 25.72 seconds, respectively. A significant increase ($P<0.01$) in APTT was observed in the dogs with parenchymal, neoplastic and biliary disorders when compared to the control group of dogs.

Table 1: Haemostatic profile of control and experimental groups

Parameters	Control group (n = 20) Mean ± SE	Experimental groups (n = 20)			F- value
		Parenchymal disorders (n = 9) Mean ± SE	Neoplastic disorders (n = 6) Mean ± SE	Biliary disorders (n = 5) Mean ± SE	
Activated partial thromboplastin time (Seconds)	35.45 ± 1.33a	130.22 ± 20.79b	156.39 ± 47.60b	125.69 ± 25.72b	11.28**

Discussion

Coagulation cascade is initiated with binding of calcium and ends with formation of clot. Coagulation cascade involves extrinsic, intrinsic and a common pathway to cause coagulation. Activated partial thromboplastin time indicates the functioning of coagulation factors involved in intrinsic coagulation cascade and common pathway factors such as factors II, V, X and fibrinogen. APTT is initiated by the exposure of contact system in the phospholipids in the endothelial tissues (Mijovski, 2019) [7].

In the present study, the APTT was prolonged in dogs affected with parenchymal, neoplastic and biliary disorders when compared with the healthy control dogs. These findings were in accordance with that of Shih *et al*. (2007) [12]; Prins *et al*. (2010) [11] and Elhiblu *et al*. (2015) [2]. In biliary tract disorders, the APTT was significantly prolonged when compared to control was similar to the findings of Neer (1992) [8]. The Prolonged APTT in biliary disorder may be due to the long standing cholestasis due to choleliths, that leads to decreased absorption of the fat-soluble vitamins resulting in a deficiency of vitamin K-dependent coagulation factors production in the hepatocytes (Neer, 1992) [8].

Dogs with neoplastic disorder showed significantly prolonged APTT when compared to the control group. Similar findings were reported by Hammer and Sikkema (1995) [4]. The prolonged APTT may be due to inadequate synthesis of coagulation factors by the liver and increased consumption of coagulation proteins by the exposed endothelial surface.

References

1. Biggs R, Macfarlane G. Human blood coagulation and its disorders. Blackwell scientific publications. 3rd Edition. Oxford 1962.
2. Elhiblu MA, Dua K, Mohindroo J, Mahajan SK, Sood NK, Dhaliwal PS. Clinico-hemato-biochemical profile of dogs with liver cirrhosis. Veterinary world 2015;8(4):487-489.
3. Gaschen L. Update on hepatobiliary imaging. Veterinary Clinics: Small Animal Practice 2009;39(3):439-467.
4. Hammer AS, Sikkema DA. Hepatic neoplasia in the dog and cat. Veterinary Clinics of North America: Small Animal Practice 1995;25(2):419-435.
5. Lakshmi K, Padmaja, Nagaraj P, Reddy AG, Prakash MG. Coagulation Profile in Hepatobiliary Disorders Affected Dogs. International Journal of Current Microbiology and Applied Sciences 2017;6(11):3975-3977.
6. Leveille-Webster CR, Center SA. Chronic hepatitis: therapeutic considerations. Kirks current veterinary therapy. Philadelphia: WB Saunders Company 1995, 749-56.
7. Mijovski MB. Advances in monitoring anticoagulant therapy. Advances in clinical chemistry 2019;90:197-213.
8. Neer TM. A review of disorders of the gallbladder and extrahepatic biliary tract in the dog and cat. Journal of veterinary internal medicine 1992;6(3):186-192.
9. Niles JD, Williams JM, Cripps PJ. Hemostatic profiles in 39 dogs with congenital portosystemic shunts. Vet Surg 2001;30:97-104.
10. Prater MR. Acquired coagulopathy II: liver disease. In: Feldman BF, Zinkl JG, Jain NC, eds. Schalm's Veterinary Hematology. Philadelphia, PA, Lippincott Williams & Wilkins 2000, 560-564.
11. Prins M, Schellens CJMM, Leeuwen MWV, Rothuizen J, Teske E. Coagulation disorders in dogs with hepatic disease. The Veterinary Journal 2010;185(2):163-168.
12. Shih JL, Keating JH, Freeman LM, Webster CR. Chronic hepatitis in Labrador Retrievers: clinical presentation and prognostic factors. Journal of veterinary internal medicine 2007;21(1):33-39.
13. Sumathi D, Selvaraj P, Nambi AP, Prathaban S, Enbavelan PA. Assessment of prothrombin and activated partial thromboplastin time in dogs. Tamilnadu Journal of Veterinary and Animal Sciences 2012;8(4):238-240.