Pakistan J. Zool., vol. 48(5), pp. 1569-1571, 2016.

Short Communication

Hemato-Biochemical Analysis and Treatment Response to Enrofloxacin in Cats Affected with Feline Hemotropic Mycoplasma

M. Saqib,[1] G. Abbas,[1,4] I. Khan,[2] M. N. Mughal,[1] A.U.R. Sial,[1,5] M. Ijaz[3,*] and M. Avais[3]

[1]*Department of Clinical Medicine and Surgery, Faculty of Veterinary Sciences, University of Agriculture, Faisalabad-38040, Punjab, Pakistan*

[2]*Section of Epidemiology and Public Health, College of Veterinary and Animal Sciences, Jhang, Pakistan*

[3]*Department of Clinical Medicine and Surgery, University of Veterinary and Animal Sciences, Lahore*

[4] *The Equine Center, 4850 Davenport Creek Road, San Luis Obispo, CA 93401, USA*

[5]*The Department of Clinical Studies, Faculty of Veterinary and Animal Sciences, PMAS-Arid Agriculture, University, Rawalpindi*

ABSTRACT

This study documents hemato-biochemical analysis and treatment response to enrofloxacin in five domestic cats affected with FHM. Diagnosis of FHM was arrived after detecting *H. felis* on Diff-Quick stained peripheral blood smear. All the cats underwent hemato-biochemical analysis pre- (day 0) and post treatment (day 15). Mean values of WBC, RBCs, HCT, MCV, MCHC, MCH, AST, ALT and total bilirubin at day 0 were found to be 20.6 x 10^9/L, 3.53 x 10^{12}/L, 21.6%, 46.3 fL, 34.2 g/dL, 19.4 pg, 238 U/L, 219 U/L and 2.38 mg/dL, respectively. Similarly, mean values of WBC, RBCs, HCT, MCV, MCHC, MCH, AST, ALT and total bilirubin at day 15 were found to be 10.8x10^9/L, 6.56 x 10^{12}/L, 38.2%, 50.9 fL, 33.1 g/dL, 14.2 pg, 35.2 U/L, 39.8 U/L and 0.35 mg/dL, respectively. Additionally, a slightly elevated WBC count (all cats) with mild basophilia (8% in cat 4) and monocytosis (11.3% in cat 4) was also appreciated. All the affected cats were treated successfully using a combination of enrofloxacin (10mg/kg, IM) and prednisolone (1mg/kg b.wt, IM) for 21 days. The goal of present study was to assess the clinical cases of FHM diagnosed in Pakistan, with a view to ascertain the hemato-biochemical findings, clinical signs and response to treatment.

Article Information

Received 2 October 2015
Revised 2 December 2015
Accepted 12 December 2015
Available online 1 August 2016

Authors' Contributions

MS, GA, AURS designed the study, compiled data and wrote the article. MNM, IK, MI and MA analyzed the results.

Key words

Feline hemotropic mycoplasmosis, anemia, hemato-biochemical analysis, enrofloxacin.

Feline hemotropic mycoplasmosis (FHM) formerly known as haemobartonellosis or feline infectious anemia (FIA), is caused by *Haemobartonella* (*H*) *felis*, a gram negative unculturable rickettsial epierythrocytic parasite that causes hemolysis and sequestration of feline red blood cells (Jensen *et al*., 2001). Haemobartonellosis manifest in 3 forms *viz.*, acute, chronic and latent. Severity of clinical signs associated with FHM solely depends on degree of hemolysis caused by *H. felis* after being attached to erythrocytes (Skyes, 2014). In other recently reported prevalence studies, male cats were reported to be more likely to be infected than were female cats (Tasker *et al*., 2003). Host factors, FHM species, and strain-associated virulence factors may affect the clinical outcome of infection (Willi *et al*., 2006a). Under microscopic examination of Giemsa-stained blood smears, *H. felis* seems to be non refractive small basophilic coccoids, strings or ring-forms on the surface of erythrocytes. Clinical findings, hemato- biochemical analysis, identification of *H. felis* in stained blood smear and serological testing are the mainstays of diagnosis. Polymerase chain reaction (PCR) has improved our ability to detect subclinical and clinical FHM infections in cats and has also been useful in documenting the global distribution of HM infections in domestic and wild felids (Fujihara *et al*., 2007; Willi *et al*., 2006b, 2007). In the present communication hemato-biochemical analysis and treatment response to enrofloxacine among 5 cats, naturally infected with *H. felis* has been described.

Materials and methods

In the present study, 5 cats presented at Veterinary Medical Teaching Hospital (VMTH), University of Agriculture Faisalabad, Pakistan from December 2013 to August 2014 with the complaint of progressive weakness, were evaluated. Varying degree of clinical manifestations including anemia, jaundice and anorexia with or without fever were recoded. All the cats were suffering from FHM, as *H. felis* was detected in Diff-Quik stained blood smears (Fig. 1). Blood samples were drawn in EDTA coated and non-coated tubes (Improvacuter®, Hamberg, Germany), respectively for hematology and serum biochemistry. Additionally, a questionnaire was also designed in order to define risk factors associated with

* Corresponding author: mijaz@uvas.edu.pk
0030-9923/2016/0005-1569 $ 8.00/0

clinical suspicion of FHM including gender, age, sex, house holding conditions, deworming, vaccination, preceding disease history, cat bite, anemia and parasitic infestation. Hemato-biochemical analyses along with microscopic evaluation of Diff-Quik stained blood smears for the presence of *H. felis* were repeated 15 days post treatment. All the FHM affected cats in this study were treated with enrofloxacin (Enrotil®, 10mg/kg b.wt, SC, q 24h) for 21 days. Additionally, all the cats received prednisolone (Solucortef®, 1mg/kg b.wt, q 12 h, for 4 days) and fluid therapy based upon dehydration status.

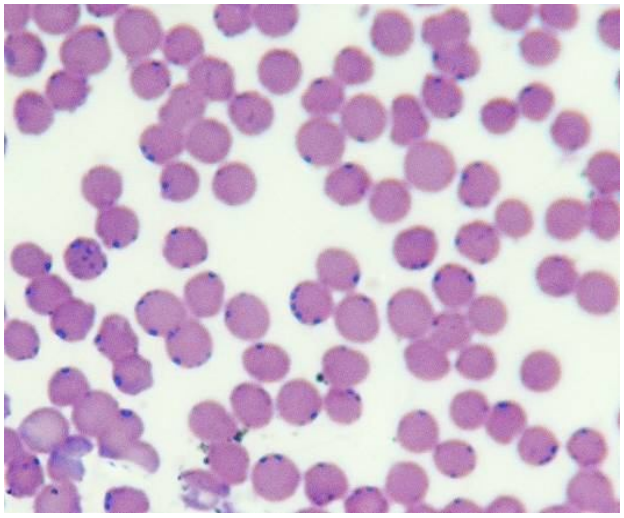

Fig. 1. Diff Quick stained blood slide of the cat affected with *Haemobartonella felis.*

Results and discussion

Results of pre- (day 0) and post treatment (day 15) hemato-biochemical analysis along with questionnaire data have been summarized in Tables I and II. Treatment with enrofloxacine was discontinued after 15 days as all the patient were recovered except cat 3, who died 2 days post initiation of treatment, and no clinical relapse was observed within 3 months post treatment. Anemia was the most commonly encountered laboratory aberrations in correlation with FMH. This anemia could be attributed to (1) decreased hematopoiesis, such as inflammatory disease associated anemia, FeLV infections and chronic kidney failure, (2) tumors, external parasites and hemostatic flaw associated blood loss, (3) hemolysis. Among cats, hemolytic anemia is usually associated with some acquired disorders such as FHM, FeLV infections, feline infectious peritonitis (FIP), oxidative damages and immune mediated processes (Kohn *et al*., 2006; Norris *et al*., 2005). Less often, hereditary defects cause hemolysis (Giger, 2005). Among infectious agents, *H. felis* is regarded as the major cause of haemolytic anemia (Tasker and Lappin, 2002).

Incidence of FHM is reported to be at peak during 4-6 years of age, affecting males predominantly (Tasker *et al*., 2003) which is in accordance with the present study. Nevertheless in dogs both sexes are at equal risk. Our study is also in line with the previous studies documenting increased incidence of FHM among outdoor and flea infested patients (Tasker and Lappin, 2002). It is problematic to compare the results of studies aimed to determine seroprevelance of FHM, as all these studies are conducted under different circumstances including either sick, anemic or some time healthy cats rendering the results less reliable for comparison (Giger, 2005). Moreover, geographical and climatic factors also influence prevalence dramatically. All the 5 cases documented herein reflect a conspicuous resemblance to the acute and chronic form of FHM (Skyes, 2014).

In the current study, hemato-biochemical analysis revealed mild normocytic normochromic regenerative anemia, normocytic hypochromic anemia and macrocytic-normochromic anemia, slightly elevated WBC count (in all cats) with mild basophilia and monocytosis. In FHM, macrocytic-normochromic response of the hematopoietic system reflects the degree of the regenerative response projected to communicate the severity of the anemia (Kurtdede and Ural, 2004). Nevertheless, if FHM exists in combination with FeLV infection, toxoplasmosis or any other chronic inflammatory condition then the expected retort is macrocytic-hypochromic and normocytic-normochromic, respectively.

The present study is also supported by the findings of previous studies reporting elevated level of AST, ALT and total bilirubin in cats affected with FHM. This might be attributed to hepatic lipidosis and hepatic hypoxia secondary to anorexia and severe hemolytic anemia, respectively. It has been reported that treatment with antimicrobial agents may reduce the degree of parasitemia (Messick and Harvey, 2012). However complete elimination of the parasite is not possible. Lamentably, in cats treatment with tetracycline may cause esophageal stricture and fever rendering enrofloxacin an efficacious alternative for cats unable to endure tetracycline antimicrobials. Glucocorticoids may be administered as adjunctive therapy in order to combat immune mediated hemolytic anemia associated with FHM and should be withdrawn as PCV increases (Liehmann *et al*., 2006). After recovery animal remains carrier for rest of life, however if PCV returns to normal limit clinical relapse is seldom observed. Prudent health management can only prevent FHM.

Table I.- Risk factors associated with clinical suspicion of FHM.

Patient	Age	Sex	Deworming status	Vaccination status	Previous disease history	Housing	Anemia	Cat bite	Flea
Cat-1	3	Male	Yes	V	NA	Outdoor/Indoor	Present	+	+
Cat-2	4	Male	No	NV	NA	Indoor	Present	-	+
Cat-3	1	Male	No	NV	NA	Indoor	Present	-	+
Cat-4	2	Male	No	NV	NA	Outdoor/Indoor	Present	-	+
Cat-5	3	Male	No	NV	NA	Outdoor/Indoor	Present	+	+

V, vaccinated; NV, not vaccinated; NA, not available.

Table II.- Hemato-biochemical analysis of cats affected with FHM.

Parameter	Cat 1		Cat 2		Cat 3		Cat 4		Cat 5		Mean		Reference Values*
	Day (0)	Day (15)	Day (0)	Day (15)	Day (0)	Day (15)	Day (0)	Day (15)	Day (0)	Day (15)	Day (0)	Day (15)	
WBC ($x10^9$/L)	21.6	11.2	22.3	8.5	24.1	NA	17.2	12.5	18.2	11	20.6	10.8	5.5-19.5
RBC ($x10^{12}$/L)	3.06	6.92	4.20	6	2.10	NA	5.03	6.13	3.27	7.2	3.53	6.56	5-10
PCV (%)	15.5	41.3	30.70	36	12.70	NA	21.3	34.5	26.3	41	21.3	38.2	30-45
MCV (fL)	40.1	52.5	48.23	50	52.43	NA	42.5	49.3	62.4	52	46.3	50.9	39-55
MCHC (g/dL)	30.8	32.5	28.25	34	42.63	NA	32.2	33.1	37.4	33	34.2	33.1	30-36
MCH (pg)	10.3	14.9	22.7	13	17.9	NA	21.4	15	23.2	14	19.4	14.2	13-17
AST (U/L)	200	27.9	270	38	300	NA	300	37	122	38	238	35.2	9.2-40
ALT (U/L)	250	48	350	27	100	NA	199	51.3	200	33	219	39.8	8.3-53
Total Bilirubin (mg/dL)	1.38	0.3	2.98	0.4	2.51	NA	1.95	0.5	3.12	0.2	2.38	0.35	0.1-0.5
H. felis	+	-	+	-	+	NA	+	-	+	-	+	-	

WBC, white blood cell; RBC, red blood cell; PCV, packed cell volume; MCV, mean corpuscular volume; MCHC, mean corpuscular haemoglobin concentration; MCH, mean corpuscular haemoglobin; AST, serum aspartate aminotransferase; ALT, serum alanine aminotransferase; NA, not available; Mean: mean values.

Keeping in view the existence of FHM in Pakistan, reports of novel cases of this parasite should be incessantly encouraged to achieve further information regarding prevalence, endemic areas, epidemic and zoonosis nationwide. This first communication on *H. felis* infection in Pakistan is projected to seek attention of health surveillance authorities regarding the establishment of supplementary effectual control measures for parasitic diseases of companion animals not reported hitherto in this region.

Statement of conflict of interest

Authors have declared no conflict of interest.

Reference

Fujihara, M., Watanabe, M., Yamada, T. and Harasawa, R., 2007. *J. vet. med. Sci*., **69**:1061–1063

Giger, U., 2005. In: *Textbook of veterinary internal medicine - diseases of the dog and cat* (eds. S.J. Ettinger and E.C. Feldman). 6th ed. Elsevier Saunders, Philadelphia, pp. 1886-1907.

Jensen, W.A., Lappin, M.R., Kamkar, S. and Reagan, W.J., 2001. *Am. J. Vet. Res*., **62**: 604-608.

Kohn, B., Weingart, C., Eckmann, V., Ottenjann, M. and Leibold, W., 2006. *J. Vet. Int. Med.*, **20**:159-166.

Kurtdede, A. and Ural, K., 2004. *Acta Vet. Brno*, **73**:507-512.

Liehmann, L., Degasperi, B., Spergser, J. and Niebauer, G.W., 2006. *J. Small Anim. Pract.*, **47**:476-479.

Messick, J.B. and Harvey, J.W., 2012. In: *Infectious diseases of the dog and cat* (ed. C.E. Greene), 4th ed. Elsevier-Saunders, St. Louis, Mo, USA, pp. 310-319.

Norris, J.M., Bosward, K.L., Whte, J.D., Baral, R.M., Catt, M.J. and Malik, R., 2005. *Aust. Vet. J*., **83**: 666-673.

Skyes, J.E., 2014. *Canine feline Infectious Diseases*, 1st ed. 209-223.

Tasker, S., Binns, S.H., Day, M.J., Gruffydd-Jones, T.J., Harbour, D.A., Helps, C.R., Jensen, W.A., Olver, C.S. and Lappin, M.R., 2003. *Vet. Rec.*, **152**:193–198

Tasker, S. and Lappin, M.R., 2002. *J. Fel. Med. Surg*., **4**:3-11.

Willi, B., Boretti, F.S., Baumgartner, C., Tasker, S., Wenger, B., Cattori, V., Meli, M.L., Reusch, C.E., Lutz, H., and Hofmann-Lehmann, R., 2006a. *J. clin. Microbiol.*, **44**: 961–969.

Willi, B., Tasker, S., Boretti, F.S., Tasker, S., Wenger, B., Cattori, V., Meli, M.L., Reusch, C.E., Lutz, H. and Hofmann-Lehmann, R., 2006b. *J. clin. Microbiol.*, **44**:4430–4435.

Willi, B., Filoni, C., Catão-Dias, J.L., Cattori, V., Meli, ML., Vargas, A., Martínez, F., Roelke, M.E., Ryser-Degiorgis, M.P., Leutenegger, C.M., Lutz, H. and Hofmann-Lehmann, R., 2007. *J. clin. Microbiol*., **45**:1159–1166.