Short Communication



Haemato-Biochemical Aspects of Foot and Mouth Disease in Cattle in Chittagong, Bangladesh

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Abstract | During an outbreak of Foot and Mouth Disease (FMD) blood samples were collected from clinically diseased (n=10) and healthy (n=10) cattle and analysed for various hemato-biochemical parameters. Results indicated a significant increase in packed cell volume (PCV) (28.6 \pm 0.98%, P=0.036), lymphocytes (70.2 \pm 6.20%, P=0.0132), monocytes (8.2 \pm 1.08%, P=0.0132) and serum glucose (104.2 \pm 15.59 mg/dL, P=0.0053) whereas a significant decrease in the concentration of hemoglobin (7.17 \pm 0.07 gm/dL, P=0.0004), basophils (0.3 \pm 0.16%, P=0.0241), eosinophils (16.5 \pm 1.83%, P=0.0097), calcium (8 \pm 0.41 mg/dL, P=0.0125) and total protein (6.2 \pm 0.19 mg/dL, P=0.0058) was recorded in FMD infected cattle. Non-significant (P >0.05) difference was noted between healthy and FMD infected animals with respect to total erythrocyte count (TEC) (P=0.6066), total leukocyte count (TLC) (P=0.3169), neutrophils (P=0.2358) and serum inorganic phosphorous (P=0.2507). It was concluded that FMD had major impact on hemato-biochemical profile of affected animals.

Keywords | FMD, Haemato-biochemical, PCV, TEC, TLC

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Noot and Mouth Disease (FMD) is a highly communicable viral disease concerned primarily with cloven hoofed animals, caused by RNA viruses of the family Picornaviridae belonging to genus Aphthovirus (Belsham, 1993). FMDV exists in seven distinct serotypes namely O, A, C, Asia 1, SAT1, SAT2, and SAT3 (Domingo et al., 2003). FMD serotype A and O viruses, responsible for an outbreak in Southeast Asia, are endemic in this region (Rweyemamu et al., 2008b). FMDV produces widespread vesicular lesions on the lips, gums, tongue, dental pad, feet and also in the udder (Lubroth et al., 2002; Rweyemamu et al., 2008b). Mortality in adult animals due to FMDV infection is rare, but other defects associated with FMD are weight loss, decreased milk production and loss of draft power (Blacksell et al., 2008), while treatment cost and time spent caring for sick animals, the farmer has to face considerable losses (Rushton et al., 2002). The international trade of animal products is constrained by FMD. To protect the livestock industries of developed as well as the developing countries livelihoods and income generation where FMD remains as endemic (Rweyemamu et

al., 2008b). Social and economic consequences of FMD were reduced milk and meat production as a result of high morbidity and loss of market value (Sangare et al., 2004). FMDV in cattle is usually obvious in the non-vaccinated herds. Sometimes due to vaccine failure, it occurred in vaccinated flock (Abubakar et al., 2014). FMDV may circulate undetected in vaccinated herds and in some indigenous breeds in areas in which FMD is common (Kitching, 2002). Frequent outbreaks noticed around the globe since the first outbreak of FMD in America in 1870 (Sumption et al., 2008; Gibbs, 2003). Due to the infection of FMDV, the livestock industry faces severe economic losses by mortality in young animals and morbidity in adult. Abubakar et al. (2012) added that FMD has constrained the trade of animals and by products from countries where FMD is prevalent.

Clinical signs are mostly associated to the progression and rupturing of vesicles at the coronary band and in the oral cavity. Vesicles and ulcerations also occur on the mammary gland. Adult animals usually recover within 8-15 days. It

was more familiar in the agro-climatic zones than in hilly areas (Abubakar et al., 2012). Animals with a history of vesicular disease, FMDV detection in samples of epithelial tissue, vesicular fluid, milk, esophageal and pharyngeal (OP) sample, or blood is enough to launch a diagnosis. FMD virus had also been shown to persist in a non-reflective form in the lymph nodes (Juleff et al., 2008). According to Rweyemamu et al. (2008a), movement or shipment of animals and people around the infected farms was restricted; however, these measures proved inadequate to prevent FMDV spread.

In Bangladesh, FMD is an endemic viral disease of cattle with financially viable losses. To control any infectious disease, identification of the risk factor is a major concern. This will help the planners to formulate an effective plan to control the disease. With a view of above description, the study was undertaken to identify the hematological and biochemical risk factors which are associated with the disease.

During an outbreak of FMD in the surrounding area of CVASU in July 2013, a total of ten clinically diseased animals from 1 to 2.5 years old were randomly selected for the collection of blood samples. Ten healthy animals of the same age were also selected as controls for analysis and comparison. Blood samples of clinically diseased and healthy animals were collected from the jugular vein in sterile vacutainers with (1mg/mL) and without EDTA for hematological and biochemical studies respectively. Non coagulated blood samples were analysed for determination of various hematological parameters as described previously (Jain, 1986). Serum was separated from coagulated blood samples and analysed for determination of glucose, total protein, calcium and phosphorous.

Statistical analysis was performed by using SPSS (version 17.0) software. Significance of difference was determined by using chi square test. Value of P < 0.05 considered as statistically significant.

Plasma samples of both healthy and infected cattle were analyzed and following observation were recorded. Significantly (P <0.05) increased packed cell volume (28.6±0.98%); lymphocytes (70.2±6.20%) and monocytes (8.2±1.08%) whereas significantly (P<0.05) decreased hemoglobin concentration (7.17±0.07gm/dL); eosinophils (16.5±1.83%) and basophils (0.3±0.16%) were recorded in FMD infected animals. Non-significant (P >0.05) difference was recorded between FMD infected and healthy animals with respect to total erythrocyte count, total leukocyte count and neutrophils. The results are presented in Table 1. In disease condition, there is a fever, which might cause the destruction of RBC and causing anemia. The result of lower blood hemoglobin was supported by Krupa-

karan et al. (2009). The PCV value of infected group was significantly (P=0.036) higher than the control group that agreed with Anim et al. (2013). In infected animals group, TLC was non-significantly higher (P=0.3169) than the control group which supported the earlier observation of Krupakaran et al. (2009). In case of viral diseases the body tried to compensate to contain the infection resulting increase number of leukocytes. In viral disease, there was always increased of lymphocytes, which substantiated in this case. In case group the monocytes value was significantly (P=0.0028) higher than the control group (Table 1) which was agreed with Alsaad et al. (2012). The Neutrophil value in case group was higher (P = 0.2358) than the control group which was agreed with Alsaad et al. (2012). The neutrophils and other phagocytic cells considered as the potent cells of immune response against viral and microbial infections. Those cells generated large amounts of Reactive Oxidative Stress (ROS) and Reactive Nitrogen Species (RNS) that considered as the main cause of lipid peroxidation supported by Bozukluhan et al. (2013) because the viral infection activates the immune system.

Table 1: Hematological parameters of healthy and FMD infected cattle

Parameter	Healthy Mean ± SE	FMD infected Mean ± SE	p-value
Hb (gm/dL)	7.97±0.15	7.17±0.07	0.0004
PCV (%)	25.2±1.14	28.6±0.98	0.036
ESR (1st hour)	0	0	0
TEC ($\times 10^6/\mu L$)	6.9±0.28	6.7±0.25	0.6066
$TLC~(\times 10^3/\mu L)$	8.8±1.39	10.7±1.14	0.3169
Lymphocyte (%)	49.2±4.30	70.2±6.20	0.0132
Neutrophil (%)	26±1.55	30.4±3.16	0.2358
Eososinophil (%)	23.1±1.33	16.5±1.83	0.0097
Monocyte (%)	3.4±0.89	8.2±1.08	0.0028
Basophil (%)	0.8±0.13	0.3±0.16	0.0241

Values at *P*<0.05 are statistically significant; **Hb**=Hemoglobin, **PCV**=Packed cell volume, **ESR**=Erythrocyte sedimentation rate, **TEC**=Total erythrocyte count, **TLC**=Total leukocyte count

Table 2: Biochemical parameters of healthy and FMD infected cattle

Parameter	Healthy Mean ± SE	FMD infected Mean ± SE	p-value
Glucose (mg/dL)	46.9±6.70	104.2±15.59	0.0053
Total protein(mg/dL)	9.7±0.98	6.2±0.19	0.0058
Ca (mg/dL)	9.4±0.28	8±0.41	0.0125
P (mg/dL)	5.1±0.8	5.9±0.63	0.2507

Values at *P*<0.05 are statistically significant; **Ca**=Calcium; **P**=Phosphorous

Serum samples from healthy and infected cattle were

taken and recorded to follow biochemical values. There was a significant (P =0.0053) increase in serum glucose (104.2±15.59 mg/dL) whereas decreased in total protein (6.2±0.19 mg/dL) and calcium (8±0.41 mg/dL) in FMD infected cattle. Non-significant (P = 0.2507) difference was recorded between FMD infected and healthy cattle with respect to serum inorganic phosphorous. The results were presented in Table 2. It is well documented that an increase in glucose concentration was a common finding in cattle affected by the stress of a systemic disease (Turgut, 2000). Decrease protein may be caused by the utilization of glucose due to interfere it to enter into the cell. It results from hyperglycemia, and it was in the line with Yeotikar et al. (2003) and Gokce et al. (2004). Reduction in total protein concentrations (Ghanem and Abdel, 2010) had been reported to be associated with hepatic and renal damage, starvation, and enteropathies leading to protein loss, parasitic infestation and chronic organ diseases indicating abnormal hepatic function. Possible causes of the decreases in serum total protein concentrations observed in the study may be associated with lesions on the oral mucosa and inter digital regions. It is known that the protein requirement increases in the presence of any lesions on the body. It is also known that consumption of protein increases in animals with diabetes mellitus, as was detected in cattle with FMD. Therefore, diabetes mellitus may be another reason for the decrease in protein concentrations observed in this study. Stress due to febrile condition, systemic infections and general body illness increase cortisol, which is agreed with (Mostl et al., 2002) depress the calcium uptake from the gut due to inhibition of vitamin D. The low calcium concentration in this study may be associated with in-appetence and hypoproteinaemia. Therefore, in the study there was a significant decrease in serum protein levels and severe anorexia in cattle with FMD, which may be the possible explanation for the hypocalcaemia observed. Significant negative correlation between calcium and phosphorus. Hyperphosphatemia was similar to that reported by Yeotikar et al. (2003) and Ghanem and Abdel (2010). Hyperphosphatemia is due to higher ATP utilization and breakdown by the virus present in the body of affected animals. Serum phosphorus is significantly increased in the FMD group, hypocalcaemia leads to reciprocal increase in the serum phosphorus concentration.

From the present study it is concluded that Foot and Mouth disease significantly affects the hematological and biochemical profile of infected animals. Keeping in view the findings of the present study, effective supportive therapy of FMD may be suggested for better and quick prognosis.

CONFLICT OF INTEREST

Nothing to disclose

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AUTHORS CONTRIBUTION

Jotan Kar, Md. Zohorul Islam and Abdul Ahad implemented the study design and carried out the laboratory experimentation. Md. Samun Sarker and Sabuj Kanti Nath drafted and revised the manuscript. All authors read and approved the final version of manuscript.

REFERENCES

- Abubakar M, Kanwal S, Saeed A (2012). Persistence, emergence and distribution of Foot and Mouth Disease Virus (FMDV); Global and Pakistan perspectives. Pak. J. life Soc. Sci. 10: 84-90.
- Abubakar M, Jamil A, Ali Q (2014). An evaluation of FMD outbreak in relation to vaccination in a herd of cattle and buffaloes. Res. J. Vet. Pract. 2(2): 28-29. http://dx.doi.org/10.14737/journal.rjvp/2014/2.2.28.29
- Alsaad KA, AlObaidi QT, Hassan SD (2012). Clinical, haematological and coagulation studies of bovine viral diarrhoea in local Iraqi calves. Bulg. J. Vet. Med. 15(1): 44–50.
- •Anim RT, Lictira G, Hermmandez TM (2013). Standardization of procedures for nitrogen fractionation of ruminant feeds. Anim. Feed Sci. Tech. 57: 347-358.
- Belsham GJ (1993). Translation and replication of FMDV RNA. Curr. Top. Microbiol. Immunol. 288: 43-70. http:// dx.doi.org/10.1007/3-540-27109-0_3
- •Blacksell S, Khounsy S, Conlan J, Gleeson L, Colling A, Westbury H (2008). Foot and Mouth disease in the Lao People's Democratic Republic: II. Sero—prevalence estimates, using structured surveillance and surveys of abattoirs. Rev. Sci. Tech. Off. Int. Epiz. 27: 851–859.
- Bozukluhan K, Atakisi E, Atakisi O (2013). Nitric oxide levels, total antioxidant and oxidant capacity in cattle with footand-mouth-disease. Kafkas. Univ. Vet. Fak. Derg. 19(1): 179-181. http://dx.doi.org/10.9775/kvfd.2012.7244
- •Domingo E, Escarmis C, Baranowski E, Ruiz–Jarabo CM, Carrillo E, Nunez JI, Sobrino F (2003). Evolution of foot and– mouth disease virus. Virus Res. 91(1): 47–63. http:// dx.doi.org/10.1016/S0168-1702(02)00259-9
- Ghanem MM, Abdel-Hamid OM (2010). Clinical, haematological and biochemical alterations in heat intolerance (panting) syndrome in Egyptian cattle following natural foot-and- mouth disease (FMD). Trop. Anim. Health Prod. 42(6): 1167-1173. http://dx.doi.org/10.1007/ s11250-010-9543-0
- •Gibbs P (2003). The foot-and-mouth disease epidemic of 2001 in the UK: IImplications for the USA and the" War on Terror". J. Vet. Med. Educ. 30: 121-132. http://dx.doi.org/10.3138/jvme.30.2.121
- •Gokce G, Gokce HI, Gunes V, Erdogan HM, Citil M (2004).



- Alterations in some haematological and biochemical parameters in cattle suffering from foot-and-mouth disease. Turk. J. Vet. Anim .Sci. 28: 723-728.
- •Juleff N, Windsor M, Reid E, Seago J, Zhang Z, Morrison IW, Charleston B (2008). Foot and—mouth disease virus persists in the light zone of germinal centres. Plos one. 3(10). e3434. http://dx.doi.org/10.1371/journal.pone.0003434
- Jain NC (1986). Schlm's veterinary helminthology. 4th Ed. Washington Square Philadelphia, PA 19106-4198, USA, Volume 2. Pp. 922-1330.
- •Kitching RP (2002). Identification of Foot and Mouth disease virus carrier and subclinically infected animals and differentiation from vaccinated animals In: Foot and Mouth disease: facing the new dilemmas (GR, Thomson, ed.). Rev. Sci. Tech. 21: 531-538.
- Krupakaran RP, Porcheziyan T, Sivseelan S (2009). Biochemical and haematological profile of Foot and Mouth disease affected crossbred cows in Karur district of Tamil Nadu. Vet. Pract. 10: 01.
- Lubroth J (2002). Foot and Mouth disease: A review for the practitioner. Vet. Clin. North. Am. Food. Anim. Pract. 18: 475
 -549. http://dx.doi.org/10.1016/S0749-0720(02)00036-1
- •Mostl E, Palme R (2002). Hormones as indicators of stress. Domest. Anim. Endocrin. 23: 67-74. http://dx.doi.org/10.1016/S0739-7240(02)00146-7
- Rweyemamu M, Roeder P, Mackay D, Sumption K, Brownlie J, Leforban Y, Valarcher JF, Saraiva V (2008a). Epidemiological

- patterns of foot-and-mouth-disease. Transbound. Emerg. Dis. 55(1): 57-72.
- Rweyemamu M, Roeder P, Mackay D, Sumption K, Brownlie J, Leforban Y (2008b). Planning for the progressive control of foot-and-mouth disease worldwide. Transbound. Emerg. Dis. 55: 73-87. http://dx.doi.org/10.1111/j.1865-1682.2007.01016.x
- •Rushton J, Heffernan CL, Pilling D (2002). Appendix 7: A literature review of livestock diseases and their importance in the lives of poor people. In: Perry BD, Randolph TF, McDermott JJ, Sones KR and Thornton PK, Investing in animal health research to alleviate poverty, ILRI (International Livestock Research Institute), Nairobi. Kenya. Pp. 129.
- •Sangare O, Dungu B and Bastos ADS (2004). Foot and Mouth disease in Mali: the current situation and proposed control strategies. Rev. Sci. Tech. Off. Int. Epiz. 23(3): 863-872.
- •Sumption K, Rweyemamu M, Wint W (2008). Incidence and distribution of foot-and-mouth disease in Asia, Africa and South America: Combining expert opinion, official disease information and livestock populations to assist risk assessment. Transbound. Emerg. Dis. 55: 5-13. http://dx.doi.org/10.1111/j.1865-1682.2007.01017.x
- Turgut K (2000). Veteriner Klinik Laboratuvar Tebhis, 2. basky, Bahyvanlar Basymsanayi, Konya, Pp. 467- 2000.
- •Yeotikal H, Said El (2003). Alteration of hematological and biochemical parameters of viral diseases. JASA. 39: 485-491.