Pakistan J. Zool., vol. 51(1), pp 383-386, 2019. **DOI**: http://dx.doi.org/10.17582/journal.pjz/2019.51.1.SC8

Short Communication

Effect of Diarrhea on Hematocrit and Serum Biochemical Profile in Foals

Muhammad Ijaz[1], Shahid Hussain Farooqi[1], Rahmatullah[2], Amjad Islam Aqib[1], Sadaqat Ali[3,*], Awais Ghaffar[1], Ahmad Ali[1] and Sehrish Saleem[1]

[1]*Department of Clinical Medicine and Surgery, University of Veterinary and Animal Sciences, Lahore 54600*
[2]*Department of Livestock and Dairy Development, Punjab*
[3]*University College of Veterinary and Animal Sciences, The Islamia University of Bahawalpur, Bahawalpur 63100*

Article Information
Received 02 November 2017
Revised 13 December 2017
Accepted 27 January 2018
Available online 11 January 2019

Authors' Contributions
MI designed and executed the study. RU did sampling. RU and SHF processed the samples. AIA, AG, AA and SS compiled and analyzed the data statistically. AG, SHF and SA wrote the manuscript. MI, AIA, SHF and SA reviewed the manuscript.

Key words
Diarrheic foals, Hematocrit, Serum electrolytes, Serum trace and ultra-trace elements.

ABSTRACT

The aim of this study was to assess the effects of diarrhea on hematocrit and various serum biochemical parameters (serum electrolytes, trace and ultra-trace elements) in foals. A total of 105 diarrheic foals (n = 35 horse foals, n = 35 donkey foals, and n = 35 mule foals) were selected regardless of their etiologies. Additionally, 12 healthy foals (n = 4 horse foals, n = 4 donkey foals, and n = 4 mule foals) as control animals were selected for this study. Packed cell volume (PCV) and serum electrolytes, trace and ultra-trace elements were measured in both groups (diarrheic and healthy). The results showed significant (p< 0.05) increase of PCV% in diarrheic foals of all species. The sodium, calcium, copper, iron and lithium concentrations significantly (p < 0.05) decreased while the potassium concentration significantly (p < 0.05) increased in diarrheic foals. Hence, this can be concluded that diarrhea in foals have significant correlation with PCV, serum electrolytes, and trace and ultra-trace elements.

Diarrhea is a common health problem in newly born foals and is a significant cause of morbidity and mortality in the neonatal foal (Haq *et al*., 2018; Knottenbelt *et al*., 2005; Cohen, 1994). Numerous parasites, non-infectious factors and microorganisms, have been associated with this condition (Magdesian, 2005). In a study of foal diarrhea which was carried out over period of 3-years, *Rotavirus*, *Clostridium perfringens*, *Cryptosporidium* species, and *Salmonella* species, were associated significantly with diarrhea whereas, *Clostridium perfringens* and *Salmonella* species were associated with fatal illness (Netherwood *et al*., 1996).

In horses diarrhea usually results in severe body fluid and electrolytes imbalance which requires instant medical attention. The required therapy alterations in serum electrolyte and blood gas values must be known which act as useful indicator. The severity of the fluid imbalance varies with duration and intensity of diarrhea, patient's ability to compensate for induced abnormality and etiology. There are few studies regarding evaluation of diarrhea in horses with respect to the association of nature of acid base disturbances and duration of diarrhea (Carlson, 1979). In order to evaluate the serum electrolytes as well as the acid-base imbalance, there is a need of laboratory tests. Although it is well understood that serum concentrations of potassium (K) and sodium (Na) are very much necessary with reference to the fluids composition used for treatment purpose (Rucker *et al*., 2008). However, information about changes of copper, iron and lithium is lacking. Keeping in mind the importance of equines, this study was designed to assess the effects of diarrhea on various serum biochemical parameters (serum electrolytes and trace elements) in foals. This is the first study up to our knowledge regarding the assessment of trace elements in diarrheic foals.

Materials and methods

This study was conducted at various public and private sector veterinary hospitals as well as equine stud farms in and around Lahore district, Pakistan. A total of 105 foals (n = 35 horse foals, n = 35 donkey foals and n = 35 mule foals) which were affected clinically with diarrhea regardless of the etiology and 12 apparently healthy foals (n = 4 horse foals, n = 4 donkey foals and n = 4 mule foals) were selected as control group for this study.

Peripheral blood was collected aseptically from puncture of jugular vein into 5 mL vacuum plain tubes and 5 mL vacuum tubes containing ethylene diamine

* Corresponding author: sadaqat.ali@iub.edu.pk
0030-9923/2019/0001-0383 $ 9.00/0

tetraacetic acid (EDTA) separately. Plain tubes were kept undisturbed for 30 min to clot the blood samples. Plain tubes were centrifuged for 12 min at 3,000 rpm after incubation (37ºC, 60 min). Serum was withdrawn into 1.5ml Eppendorf tubes and frozen at −80ºC for later biochemical analysis. Tubes containing EDTA were used for determination of PCV using microhematocrit method. The serum samples were analyzed by atomic absorption spectrophotometer. Macro minerals (calcium, potassium and sodium) and trace elements (copper and iron), and ultra-trace elements (lithium) were estimated as described by Basaki *et al*. (2012), Tajik and Nazifi (2013) and Akhter *et al*. (2007) at Quality Operations Laboratory, UVAS, Lahore. The values were compared with references values.

Data obtained from comparative study of two groups (*i.e.* diarrheic and healthy foals) on hematocrit and serum biochemical profile were analyzed by student *t*-test using IBM® SPSS (Statistical Product and Service Solutions) Statistics® version 22.0. The probability (*p*) value less than 0.05 were considered significant.

Table I.- Effect of diarrhea on the average packed cell volume, serum macro minerals, trace and ultratrace elements in Foals.

Parameter	Species	Diarrheic foals	Healthy foals
PCV (%)	Horse	56.00 ± 4.40*	40.75 ± 3.59
	Donkey	57.00 ± 9.80*	45.25 ± 0.95
	Mule	59.00 ± 3.90*	42.00 ± 2.90
Sodium (mmol/L)	Horse	109.1 ± 6.22*	142.7 ± 5.17
	Donkey	103.8 ± 6.50*	139.2 ± 2.04
	Mule	102.1 ± 3.63*	137.0 ± 2.61
Potassium (mmol/L)	Horse	06.20 ± 0.73*	03.73 ± 0.31
	Donkey	06.30 ± 0.75*	04.14 ± 0.15
	Mule	05.90 ± 0.65*	03.53 ± 0.15
Calcium (mmol/L)	Horse	02.20 ± 0.12*	02.87 ± 0.26
	Donkey	02.20 ± 0.11*	02.79 ± 0.16
	Mule	02.20 ± 0.11*	02.94 ± 0.21
Copper (µg/dL)	Horse	70.00 ± 21.0*	142 ± 17.00
	Donkey	68.00 ± 13.0*	135 ± 09.00
	Mule	59.00 ± 19.0*	130 ± 05.00
Iron (µg/dL)	Horse	102.0 ± 18.0*	220 ± 27.00
	Donkey	095.0 ± 13.0*	205 ± 19.00
	Mule	098.0 ± 14.0*	224 ± 14.00
Lithium (µg/dL)	Horse	06.00 ± 1.00*	14.0 ± 4.00
	Donkey	05.00 ± 2.00*	12.0 ± 2.00
	Mule	06.00 ± 1.00*	12.0 ± 2.00

* Significant difference $p < 0.05$.

Results and discussion

PCV in all diarrheic foals of horses, donkey and mules was significantly higher (p-value<0.05) compared to their healthy counterparts. Average PCV (%) levels in diarrheic foals of horses, donkeys, and mules were revealed 56, 57, and 59, respectively (Table I). PCV was slightly higher in mule foals, followed by donkey and horse foals. Concentration of sodium, potassium, calcium, copper, iron and lithium were evaluated in the serum sample of diarrheic as well as control group of equine foals. Serum sodium, copper, iron and lithium levels in diarrheic foals of all species in the study were decreased significantly ($p< 0.05$) when matched with healthy ones. The serum potassium values were however significantly increased ($p< 0.05$) in all diarrheic foals comparing with healthy foals.

Increased PCV in all diarrheic foals compared to healthy foals was consistent with the findings of Frederick *et al*. (2009), who reported increased PCV in non-survivor compared to survivor diarrheic foals. A case of an adult horse with acute diarrhea having high PCV stated by Oliver and Stampfli (2006) also confirmed these results. PCV increases in diarrhea due to the loss of the water from plasma consequently cellular part of blood converts to major portion.

Keeping in view the control group, reduced serum sodium concentrations in all diarrheic foals were in agreement with previous studies (Oliver and Stampfli, 2006; Gomez *et al*., 2013), which disclosed hyponatremia in adult horses with acute diarrhea. Diarrhea leads to loss of isotonic fluid (hypovolemia). Excessive loss of gastrointestinal water, sodium, chloride, and bicarbonate diminish the extracellular portion of these electrolytes. Kidney response of fluid loss is to accelerate proximal tubule's reabsorption sodium, chloride, bicarbonate and water. Normal plasma sodium to chloride (1.4:1) is decreased so it is called hyperchloremic metabolic acidosis. Sodium reabsorption results in sodium delivery at distal tubule for H^+ exchange exacerbating the acidosis (Wilson and Green, 1986).

Higher serum potassium values in all three categories of diarrheic foals supported the results of case report about diarrhea in non-suckling horses (Wilson and Green, 1986). Grinberg *et al*. (2009) reported hyperkalemia in hospitalized foals affected with cryptosporidiosis are also in close agreement with this study. Contradictory to this, hypokalemia were reported by various authors (Oliver and Stampfli, 2006; Navarro *et al*., 2005; Gomez *et al*., 2013). Underlying whole body potassium deficit might be the reason of hypokalemia in previous studies.

Consistent low serum calcium levels in all diarrheic foals during the study coincided with earlier work (Navarro

et al., 2005) in which both total and ionized calcium levels were decreased in colic horses with diarrhea. Another study revealed lower total, ionized and adjusted ionized calcium levels in horses with diarrhea (Kolk *et al*., 2002) are also accorded with our results. Likewise, Dart *et al*. (1992) also described lower serum ionized calcium concentration in horses with surgical managed gastrointestinal disease. Previously, other studies also stated hypocalcemia in diarrheic calves (Cabello *et al*., 1977; Groutides and Michell, 1990; Michell *et al*., 1992). Possible reasons of hypocalcemia might be alterations in the levels of ionized calcium due to modifications in blood pH and plasma albumin levels, infection, inadequate food intake, and intravenous fluid therapy without calcium supplementation, and ileus (Navarro *et al*., 2005).

Decreased serum concentration of copper and zinc in diarrheic people (Kilic *et al*., 2003; Arora *et al*., 2006) were congruent to hypocupremia of present study in diarrheic foals. Copper is required for hemoglobin formation, iron transport, and ceruloplasmin synthesis. Ceruloplasmin (ferroxidase) is a glycoprotein enzyme produced in the liver and essential in iron oxidation, permitting it to bind with the iron transport protein transferrin (Jain, 1993). Copper is generally thought to be readily absorbed in the upper gastrointestinal tract including stomach, duodenum and jejunum (Sternlieb, 1967). Hypocupremia may be inherited and acquired copper deficiency. The causes of latter include dietary deficiency, protein deficiency, high amount of iron, zinc or ascorbic acid, diarrhea, inflammatory bowel disease, short bowel syndrome, stoma, fistula, and celiac disease in humans (Sandstead, 1982; Rodriguez *et al*., 1985; Spiegel and Willenbucher, 1999). So, it can be concluded that hypocupremia leads to serum iron deficiency consistent with findings of this study. Iron deficiency anemia were reported in children affected with acute gastroenteritis due to *Entamoeba histolytica* and *Giardia lamblia* in Gaza (Al-Laham *et al*., 2015), latter may be cause of diarrhea in our foals. Relation of iron deficiency anemia and celiac disease in was well described which included impaired duodenal mucosal uptake of iron due to reduced surface absorptive area, blood loss due to ulceration and neoplasia, immune-mediated hemolysis, and reduced expression of regulatory proteins involved in iron uptake (Freeman, 2015).

Serum lithium levels in all diarrheic foals were decreased versus healthy foals. Unfortunately, there was no published work on diarrheic effect on lithium body reserves. Lithium is assimilated in the alimentary tract and expelled by urinary system entirely (Schrauzer, 2002; Linakis, 2007; Shahzad *et al*., 2016). Lithium is distributed in total body water and does not bind to serum proteins (Okusa and Crystal, 1994; Shahzad *et al*., 2016). Lithium competes with cations, especially Na^{+}, K^{+}, Mg^{++} and Ca^{++}. It may also interact with ammonia groups (including biogenic amines) (Bunney and Mandell, 1979). It interacts with cyclic-AMP-mediated processes, notably in the kidney, thyroid, and CNS (Ebstein and Belmaker, 1979). It has effects on membranes (*i.e.,* red cell membrane), neurotransmitter systems and serotonin synthesis (Mandell and Knapp, 1976; Rosenthal and Goodwin, 1982). Lithium deficiency may likely be attributed to the lithium-poor nutrition and mothers of the diarrheic foals, not the diarrhea. It was reported in goats that lithium-poor nutrition decreased feed intake in growing youngones, lower birth weight of kids, lesser conception rate, increased abortion rates, delivery of more female kids, decreased milk production, life expectancy and serum enzyme activity (isocitric acid dehydrogenase, malate dehydrogenase, aldolase, and glutamine dehydrogenase), except stress indicator enzyme (creatinine kinase), and skin lesions (Anke *et al*., 1983, 1991; Mueller *et al*., 2010).

Conclusion

It can be concluded that diarrhea in foals lead to changes in blood hematocrit, serum minerals, trace elements and ultra-trace elements. With diarrhea there is an increase in PCV% and concentration of potassium, while the concentration of sodium, calcium, copper, iron and lithium decreased.

Acknowledgements

The authors are grateful to Higher Education Commission, Pakistan and Medicine Laboratory, University of Veterinary and Animal Sciences, Lahore for the provision of laboratory and technical assistance for the smooth run of this research study.

Statement of conflict of interest

Authors have declared no conflict of interest.

References

Akhter, M.Z., Khan, A., Sarwar, M. and Javaid, A., 2007. *Asian-Aust. J. Anim. Sci*., **20**: 393-398.

Al-Laham, N.A., Elyazji, M.S., Al Haddad, R.J. and Ridwan, F.N., 2015. *Annls. Med. Hlth. Sci. Res.,* **5**: 292-298.

Anke, M., Arnhold, W., Groppel, B., Krause, U., 1991. In: *Lithium in biology and medicine* (eds. G.H. Schrauzer and G.H. Klippel). VHC, pp. 149-167.

Anke, M., Groppel, B., Kronemann, H., Grim, M., 1983. In: *Spurenelement Symposium – Lithium* (eds. M. Anke, W. Baumann, H. Braunlich and C. Bruckner). Universitat Leipzig, Universitat Jena, pp. 58-65.

Arora, R., Kulshreshtha, S., Mohan, G., Singh, M. and Sharma, P., 2006. *Biol. Trace Elem. Res*., **114**: 121-126. https://doi.org/10.1385/BTER:114:1:121

Basaki, M., Saeb, M., Nazifi, S. and Shamsaei, H.A., 2012. *Biol. Trace Elem. Res*., **148**: 161-164. https://doi.org/10.1007/s12011-012-9360-6

Bunney, WE. and Mandell, A.J., 1979. In: *Lithium: Controversies and unresolved issues* (eds. T.B. Coopr, S. Gershon, N.S. Kline and M. Schou). Int. Congr. Ser. No. 478, Amsrerdam-Oxford-Princeton ExetMeic, pp. 984; pp. 673-674.

Cabello, G., Michel, M.C., Foucher, C. and Lafarge, C., 1977. *Annls. Rech. Vet*., **8**: 203-211.

Carlson, G.P., 1979. *Vet. Clin. North Am. Large Anim. Pract*., **1**: 313-329. https://doi.org/10.1016/S0196-9846(17)30187-8

Cohen, N., 1994. *J. Am. Vet. Med. Assoc*., **204**: 1644-1651.

Dart, A.J., Snyder, J.R., Spier, S.J. and Sullivan, K.E., 1992. *J. Am. Vet. Med. Assoc*., **201**: 1244-1248.

Ebstein, R.P. and Belmaker, R.H., 1979. *Lithium and brain adenylate cyclas*. Amsterdam: Experta Medica, pp. 703-729.

Frederick, J., Giguere, S. and Sanchez, L,C., 2009. *J. Vet. Intern. Med*., **23**: 1254-1260. https://doi.org/10.1111/j.1939-1676.2009.0383.x

Freeman, H.J., 2015. *World J. Gastroenterl*., **21**: 9233.

Gomez, D.E., Arroyo, L.G., Stampfli, H.R., Cruz, L.E. and Oliver, O.J., 2013. *J. Vet. Intern. Med*., **27**: 548-553. https://doi.org/10.1111/jvim.12071

Grinberg, A., Pomroy, W.E., Carslak, H.B., Shi, Y., Gibson, I.R. and Drayton, B.M., 2009. *N.Z. Vet. J*., **57**: 284-289. https://doi.org/10.1080/00480169.2009.58622

Groutides, C.P. and Michell, A.R., 1990. *Br. Vet. J*., **146**: 205-210.

Haq, I., Durrani, A.Z., Khan, M.S., Mushtaq, M.H., Ahmad, I., Khan, A. and Ali, M., 2018. *Pakistan J. Zool.,* **50**: 381-384. http://dx.doi.org/10.17582/journal.pjz/2018.50.1.sc5

Jain, N.C., 1993. *Essentials of veterinary hematology*. Lea and Febiger, Philadelphia, pp. 173-175.

Kilic, E., Yazar, S. and Saraymen, R., 2003. *Biol. Trace Elem. Res*., **96**: 153-158. https://doi.org/10.1385/BTER:96:1-3:153

Knottenbelt, D.K., Holdstock, N. and Madigan, J.E., 2005. In: *Equine neonatology: Medicine and surgery*. Elseiver, London, England, pp. 219.

Kolk, J.V., Nachreiner, R.F., Refsal, K.R., Brouillet, D. and Wensing, T., 2002. *Equine Vet. J*., **34**: 528-531. https://doi.org/10.2746/042516402776117692

Linakis, J.G., 2007. *Toxicity, lithium*. eMedicine, 8 January 2007. http://www.emedicine.com/EMERG/topic301.htm.

Magdesian, K.G., 2005. *Vet. Clin. N. Am. Equine Pract*., **21**: 295-312.

Mandell, A.J. and Knapp, S., 1976. *Neuropsyhopharakol,* **9**: 116-126.

Michell, A.R., Brooks, H.W., White, D.G. and Wagstaff, A.J., 1992. *Br. Vet. J*., **148**: 507-522. https://doi.org/10.1016/0007-1935(92)90006-M

Mueller, R., Anke, M. and Betz, L., 2010. In: *Proceedings of the 30 Scientific symposium of industrial toxicology* (eds. A. Fargasova, M. Jambrich, M. Jambrich, V. Koprda, V. Koprda and M. Melnik). Slovakia, pp. 376.

Navarro, M., Monreal, L., Segura, D., Armengou, L. and Anor, S., 2005. *J. Vet. Intern. Med*., **19**: 871-877. https://doi.org/10.1111/j.1939-1676.2005.tb02780.x

Netherwood, T., Wood, J.L., Townsend, H.G., Mumford, J.A. and Chanter, N., 1996. *Epidemiol. Infect*., **117**: 375-383. https://doi.org/10.1017/S0950268800001564

Okusa, M.D. and Crystal, L.J., 1994. *Am. J. Med*., **97**: 383-389. https://doi.org/10.1016/0002-9343(94)90308-5

Oliver, O.E. and Stampfli, H., 2006. *Vet. Clin. N. Am. Equine Practic*., **22**: 73-84.

Rodriguez, A., Soto, G., Torres, S., Venegas, G. and Castillo-Duran, C., 1985. *Acta Pediat*., **74**: 770-774. https://doi.org/10.1111/j.1651-2227.1985.tb10029.x

Rosenthal, N.E. and Goodwin, F.K., 1982. *Annu. Rev. Med*., **33**: 555-568. https://doi.org/10.1146/annurev.me.33.020182.003011

Rucker, R.B., Fascetti, A.J. and Keen, C.L., 2008. In: *Clinical biochemistry of domestic animals*. Elsevier Inc., pp. 663-693. https://doi.org/10.1016/B978-0-12-370491-7.00022-2

Sandstead, H.H., 1982. *Am. J. clin. Nutr*., **35**: 809-814. https://doi.org/10.1093/ajcn/35.4.809

Schrauzer, G.N., 2002. *J. Am. Coll. Nutr*., **21**: 14-21. https://doi.org/10.1080/07315724.2002.10719188

Shahzad, B., Mughal, M.N., Tanveer, M., Gupta, D. and Abbas, G., 2016. *Environm Sci. Pollut. Res. Int*., **24**: 103-115.

Spiegel, J.E. and Willenbucher, R.F., 1999. *J. Parenter. Enteral. Nutr*., **23**: 169-172.

Sternlieb, I., 1967. *Gastroenterology*, **52**: 1038-1040.

Tajik, J. and Nazifi, S., 2013. *Pak. Vet. J.,* **33**: 5-8.

Wilson, E. and Green, R., 1986. *J. Equine Vet. Sci*., **6**: 321-325. https://doi.org/10.1016/S0737-0806(86)80006-5