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ETIO-PATHOLOGY AND THERAPEUTICS OF PICA IN DROMEDARY CAMELS

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ABSTRACT

In the present study, a very high incidence (51.66%) of pica was recorded in camels of all age groups at an organised farm. Camel calves of less than one year of age showed vices of licking the manger and corral walls along with eating floor soil. Pica was more pronounced in active growing camels between 1-5 years of age. Common vices observed were osteophagia, geophagia, lithophagia and coprophagia depending upon the availability of that particular object in the vicinity of the camels. General weakness as reflected by thinness of the hump, roughness of the hair coat, anaemia and emaciation were commonly observed symptoms. High faecal silica content in the pica affected camels suggested high silica consumption by the pica affected camels. There was a significant decrease in mean erythrocyte count, haemoglobin concentration, packed cell volume, total serum protein, serum globulin, serum calcium and phosphorus concentrations in the pica affected camel, as compared to healthy and treated camels. Decrease in zinc and iron levels were also observed in the pica affected camels as compared to healthy camels. No significant difference in serum copper, cobalt and selenium concentrations were recorded in pica affected and healthy camels. Treatment using specially designed mineral mixture at the rate of 50 gm per day per animal for 60 days was found satisfactory in terms of improving haemato-biochemical and serum mineral profiles in camels. Mineral mixture supplementation was also found satisfactory in reducing the symptoms of pica.

Key words: Camel, etipathology, pica, threrapeutics

Pica or allotriophagia associated with parasitism and deficiencies of phosphorus, salt or protein (Smith, 2015) and has been widely reported in cattle in Egypt (Elshahawy *et al,* 2016) and Iraq (Mosa *et al,* 2020). Minerals like copper, zinc, and cobalt, has been implicated in the aetiology of pica and fleece dietary pattern in sheep (Fahmy *et al*, 1980). Imbalance between the minerals either due to deficiency or interaction lowers immune status of the animals which ultimately affects its production (Judson *et al*, 1987). In camels these ailments are chronic and difficult to treat medically because of unknown etiology. Gautam and Bansal (1972) reported disease of camels known as "Mitti Khana" in India, due to heavy infections of gastro-intestinal worms with deficiencies of minerals such as calcium and phosphorus and of total proteins.

The most common cause of intestinal obstruction in camels having pica is eating hairs or plant fibres, leading to formation of phytobezoars and trichobezoars that may reach the intestine causing obstruction (Tanwar, 1985; Tharwat, 2012).

Present study was planned to record the incidence, clinical signs, serum minerals and haematological changes in camels suffering from pica along with its treatment using, specially prepared mineral mixture supplement*.

Materials and Methods

A total of 300 camels of an organised herd were included in the present study. These were given drinking water dewormers and antitrypanocidal drugs at regular intervals. These camels were maintained in the open housing system with stall feeding and allowed browsing and grazing daily for five hours in the rain fed demarcated area of the farm.

Feeding treatment trial of the selected animals: Out of these 300 camels 12 severely affected male camels aged between 2-4 years were divided into two groups comprising six animals in each group. In the feeding treatment trial six camels of group-1 were fed with specially designed mineral mixture* daily at the rate

* The composition of the designed mineral mixture (per 100 kg) was as follows: dicalcium phosphate (Ca_2PO_4) 59.00 kg; calcium carbonate ($CaCO_3$) 40.50 kg; zinc sulphate ($ZnSO_4$) 0.230 kg; copper sulphate ($CuSo_4$) 0.160 kg; manganese sulphate ($MnSO_4$) 0.030 kg and cobalt sulphate ($CoS0_4$) 0.050 kg.

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of 50 gram per animal per day, whereas 6 animals of group-2 were fed with the same basal fodder for 60 days. All these camels were observed for one hour during day time for eating of non-feed items in order to know the incidence of pica. These were also examined for associated signs and symptoms of rough hair coat, anaemia and weakness.

Two bolus (35-40 gm) of fresh faecal samples were collected separately for each camel in duplicate in polyethylene bags both prior to start of the treatment and 60 days post treatment. These faecal samples were transported to the laboratory, in a cool transport thermocol box, for further examination.

Qualitative examination of all the faecal samples was carried out to record the gastrointestinal parasitic infestation in the experimental camels by using centrifugal floatation and sedimentation techniques (Soulsby, 1982). None of the camel was severely infected therefore quantitative examination to know the eggs per gram faeces was not carried out. This might be due to the regular deworming schedule adopted at the farm.

Estimation of silica and sand content in the faecal samples: Ash is the inorganic residue left after ignition of a faecal sample in the muffle furnace at 550-600°C for 2-3 hours. The residue left after dissolving inorganic portion of total ash represented acid insoluble ash (AIA), majority containing sand and silica (Sastry *et al*, 1999).

All these faecal samples were estimated for silica content. Two boluses (35-40 gm) of faecal sample collected were taken in the pre weighed petridishes. These faecal samples were dried by keeping the petridishes in the hot air oven at 70°C for 12 hours. Five gram of the dry faecal sample was taken in the pre-weighed silica basins. These basins were kept in the muffle furnace for ignition at 550-600°C for 2-3 hour to obtain total ash. Then in each silica basin about 10 ml of dilute (1:2) HCl was added. Then the contents of silica basins were transferred to 25 ml beakers. This content was boiled for 5-10 minutes on hot plates and then filtered using Whatman no.1 filter papers in volumetric flasks (250 ml). Contents of the beaker and residue on the filter paper were made acid free by repeated hot water washing of the beaker and then pouring the residue on the filter paper. Filter paper with retained residue was transferred to a pre-weighed silica basin. Contents of these basins were again dried in the hot air oven at 70±2°C for 12 hours and then subjected to ashing in the muffle furnace at 550-600°C for 1-2 hour for decarburisation. Finally these basins were cooled in desiccators and weighed with left back AIA. In this way silica and sand content of the faecal samples were estimated by the following equation: AIA (%) = b-a/w× 100 (Where, b= weight of silica basin with AIA, a= empty weight of silica basin, w= weight of moisture free sample taken for ashing).

Collection of blood and serum samples: Blood samples from all the 12 experimental camels were collected on day 0 (pre treatment) and then on day 15, 30, 45 and 60 (post treatment) by jugular vein puncture in sterile vacuutainers with and without anticoagulant for estimation of haematological, serum biochemical and serum mineral profiles. Blood and serum samples of 5 apparently healthy male camels of the same age group of the same herd were also collected to know the normal haemato-biochemical and mineral profiles of the herd.

Haematological examination: Blood samples were analysed for haemoglobin (Hb), packed cell volume (PCV), total erythrocyte count (TEC), total leukocyte count (TLC) and differential leukocyte count (DLC) using standard methods (Jain, 1986).

Biochemical analysis of serum samples was made to ascertain liver function by estimating serum total protein, serum albumin, serum globulin, alkaline phosphatase (ALKP), serum aspartate aminotransferase (SGOT) and serum alanine aminotransferase (SGPT), by the Vet Test Chemistry Analyser using kit supplied by Chema Diagnostics, Monsano, Italy. Serum globulin concentration was estimated as a difference between total protein and albumin. Albumin and globulin ratio (A: G) was derived by dividing albumin concentration with globulin concentration in g/dl.

Estimation of serum minerals (Ca, P, Zn, Cu Co, Fe): Serum samples were digested as per the procedure described by Kolmer *et al* (1951). Serum calcium, phosphorus, zinc, copper, iron, and cobalt were estimated in these digested samples by Inductively Coupled Argon Plasma Spectrometer (iCAP 7000Series) by Thermo Fisher Scientific Inc.

Statistical analysis: Mean, standard error, percentages and comparison of differences between means were calculated as per the standard statistical procedures suggested by Snedecor and Cochran (1994).

Results

A very high incidence (51.66%) of the pica was recorded in the herd studied. Vices of licking the

manger and corral walls along with eating floor soil started in camel calves of less than one year of age. Pica was more pronounced in active grower camels between 1-5 years of age (60.81%). These camels were picking and chewing long bones for longer times and were even engulfing the pieces of construction bricks. Coprophagy was also more pronounced in these age group camels (Table 1). Male camels were more affected (54.86%) as compared to female camels (49.73%) in the herd examined (Table 2).

Table 1. Incidence of pica as per age group of the camels.

Age of the camel	Total No. of the camels	Pica affected camels	Percentage of the affected camels
< 1year (young)	33	18	54.54
1-5 years (Active growers)	148	90	60.81
>5 years (Adults)	119	47	39.50
Total	300	155	51.66

Table 2. Incidence of pica as per sex of the camels.

Sex	Total No. of camels	Pica	Percentage (%)
Male	113	62	54.86
Female	187	93	49.73
Total	300	155	51.66

Table 3. Vices recorded in a total of 300 camels.

Abnormal materials	Total No. of camels	Percentage (%)
Chewing of bones 'osteophagia'	85	28.33
Sand/ soil/ mud eating 'geophagia'	47	15.67
Brick/ concrete wall eating 'lithiphagia'	28	9.33
Faeces eating 'coprophagia'	13	4.33
More than one object eating	18	6.00
Total pica affected camels	155	51.66

Common vices observed were osteophagia (28.33%) followed by geophagia (15.67%), lithophagia (9.33%) and coprophagia (4.33%), depending upon the availability of the particular objects in the vicinity of the animals. Six per cent of camels were observed to be eating more than one abnormal material (Table 3). These combinations observed were mainly of coprophagia and geophagia, osteophagia and geophagia, lithophagia and geophagia. Poor body condition as reflected by thinness of the hump, roughness of the hair coat, anaemia and emaciation were more common in pica affected compared to unaffected camels (Table 4).

Table 4. Clinical signs observed in pica affected and unaffected camels.

Symptoms	Pica affected camels (n=155)	Per cent	Pica unaffected camels (n=145)	Per cent
Weakness	89	57.42	12	8.27
Rough hair coat	25	16.12	6	4.14
Anaemia	22	14.19	0	0.00
Emaciation	9	5.81	2	1.38

Faecal samples examination for gastrointestinal parasites: Initial pretreatment faecal samples examination of the 12 experimental camels revealed minor parasitic infestations in two camels, one was positive for trichuris egg and second for strongyle egg. This reflected that pica was not due to parasitic infestation. Regular deworming schedule adopted for the farm was effective in taking care of gastrointestinal parasites. Examination of faecal samples 60 days post treatment also did not reflect any parasitic infestations in these camels.

Silica content in faecal samples: On 60 days post treatment, faecal dry matter content was observed to be higher whereas ash, acid insoluble ash and silica content of ash were lower in the treated group reflecting lower consumption of silica in the treated group compared to control group (Table 5).

Table 5. Mean+S.E of Silica content in faeces of treated and untreated control group of camels on 60 days post treatment.

Attribute	Treated group (n=6)	Untreated Control group (n=6)	P Value
Faecal DM (%)	29.11±1.19	25.32±1.01	0.067
Ash content (%)	18.47±0.52	22.65±2.36	0.174
Silica content (%)	8.51±0.60	13.43±2.97	0.200
Silica% of ash	45.72±2.04	55.22±5.63	0.210

Haematological parameters (Table 6): Significant increase in the mean TEC was recorded in the treated group compared to control group on day 30, 45 and 60 ($P \leq 0.05$), post treatment. Significant increase in mean value of TEC count was also observed on day 30, 45 and 60 post treatment compared to day 0 ($P \leq 0.05$), in the treated group. No significant difference in the mean Hb concentration was recorded between treated and control group on day-0. Mean Hb concentration in the treated group increased significantly compared to control group on day 15, 30, 45 and 60 ($P \leq 0.05$), post treatment. Significant increase in Hb was also observed on

Table 6. Mean±SE values of haematological parameters at different intervals of treatment.

Parameter	Treated group					Untreated control group				
	0 day	15 day	30 day	45 day	60 day	0 day	15 day	30 day	45 day	60 day
Erythrogram										
TEC (10^6/ mm^3)	8.06± 0.02^{a}	8.30± 0.27	8.40± 0.19^{Ab}	8.90± 0.20^{Ab}	9.20± 0.21^{Ab}	7.80±0.18	7.80± 0.20	7.70± 0.20^{B}	7.90± 0.21^{B}	8.08± 0.20^{B}
Hb (gm%)	7.30± 0.18^{a}	7.80± 0.15^{Ab}	8.00± 0.22^{Ab}	8.40± 0.22^{Ab}	8.70± 0.20^{Ab}	7.20±0.21	7.00± 0.14^{B}	7.20± 0.12^{B}	7.50± 0.17^{B}	7.50± 0.17^{B}
Range	7.0- 8.0	7.2- 8.2	7.2- 8.8	7.8 -9.4	8.0- 9.4	6.4- 7.8	6.4- 7.4	7.0- 7.8	6.8-8.0	6.8- 7.8
PCV (%)	34.16± 0.70	36.16± 0.60	36.50± 0.22	36.60± 0.42	37.50± 0.34	32.80± 0.60	34.00± 0.86	34.30± 0.61	33.80± 0.40	33.62± 0.33
TLC (10^3/mm^3)	14.38± 0.92	14.83± 0.78	13.38± 0.73^{A}	13.45± 0.64^{A}	13.95± 0.52^{A}	17.31± 2.43	18.45± 2.00	19.11± 0.98^{B}	18.95± 1.02^{B}	18.28± 0.54^{B}
DLC (%)										
Lymphocyte	53.33± 0.71	52.16± 0.65^{A}	49.0± 0.36^{A}	57.33± 1.05^{A}	55.16± 1.22^{A}	54.33± 0.61	54.33± 0.56^{B}	54.16± 0.60^{B}	63.0± 1.63^{B}	63.0± 1.26^{B}
Neutrophils	38.83± 0.60^{a}	38.8±1.05	42.66± 0.12A^{b}	40.16± 1.02	42.33± 1.05^{Ab}	37.00± 1.39	37.66± 0.33	37.50± 0.56^{B}	33.83± 1.49	33.00± 1.41^{B}
Eosinophils	5.3± 0.33^{a}	4.5± 0.50^{b}	3.8± 0.31^{b}	0.66± 0.33^{b}	0.33± 0.21^{b}	4.66± 0.33	4.0± 0.26	4.66± 0.21	2.16± 0.70	2.66± 0.49
Basophils	0.5± 0.22	0.6± 0.21	0.5± 0.22	0.16± 0.17	0.16± 0.17	1.16± 0.17	1.0± 0.00	0.83± 0.17	0.33± 0.21	0.5± 0.22
Monocytes	4.6± 0.49^{a}	3.6± 0.33	4.0± 0.26^{A}	1.6± 0.33^{Ab}	1.8± 0.60^{b}	3.6± 0.33	3.0± 0.26	2.8± 0.17^{B}	0.6± 0.21^{B}	0.8± 0.31

Note: - 1. Mean±SE bearing different superscript (A, B) between treated and control group differed significantly ($p<0.05$).

2. Mean±SE bearing different superscript (a, b) within treated group differed significantly ($p<0.05$).

day 15, 30, 45 and 60 post treatment compared to day-0 (P≤0.05), in the treated group. No significant difference in the mean PCV was recorded between treated and control group on day-0. Whereas in the treated group PCV increased significantly compared to control group on day 15, 30, 45 and 60 (P≤0.05), post treatment. Significant increase in PCV was also observed on day 30, 45 and 60 post treatment compared to day-0 (P≤0.05).

In the treated group, no significant difference in the TLC was recorded between treated and control groups on day-0. Whereas in the treated group TLC increased significantly compared to control group on day 30, 45 and 60 (P≤0.05), post treatment. No significant difference in TLC was observed post treatment compared to day-0 (P≤0.05), in the treated group. No significant difference in the mean lymphocyte count was recorded on day-0 of the start of treatment between treated and control groups. Whereas in the treated group there was significant difference in the mean lymphocyte count compared to control group on day 15, 30, 45 and 60 (P≤0.05), post treatment. No significant change in mean lymphocyte count was observed on day 15, 30, 45 and 60 post treatment compared to day-0 (P≤0.05), in the treated group. Significant decrease in lymphocyte count was observed on day 60 post treatment (P≤0.05) in the treated group compared to control group. No significant difference in the mean neutrophils count was recorded on day 0, 15 and 45 post treatment between treated and control groups. Whereas significant increase in neutrophils count was recorded on day 30 and 60 post treatment in the treated group compared to control group. Significant increase in neutrophils was also observed on day 30 and 60 in the treated group compared to day-0 of the treated group (P≤0.05). No significant difference in the mean eosinophils count was recorded on day 0, 15, 30, 45 and 60 post treatment between treated and control groups. Significant difference in mean eosinophils count was recorded on day 15, 30, 45 (P≤0.05) and 60 (P≤0.01) in the treated group compared to day-0 of the treated group. No significant difference in mean basophils count was observed between treated and control groups either pre or post treatment. No significant difference in the mean monocytes count was recorded on day 0, 15 and 60 post treatment between treated and control groups. Whereas significant difference in monocytes count was observed on day 30 and 45 post treatment

Table 7. Mean±SE value of liver enzymes, protein profile camels at different intervals of treatment.

Parameter	Treated group					Control group				
	0 day	15 day	30 day	45 day	60 day	0 day	15 day	30 day	45 day	60 day
Protein profile										
T.P. (gm/dl)	6.59± 0.34^{Aa}	8.02± 0.66	7.76 ±0.56^{A}	7.50± 0.56^{Ab}	7.99± 0.56^{Ab}	7.22± 0.16^{B}	6.61± 0.43	6.26± 0.15^{B}	5.54± 0.20^{B}	6.32± 0.30^{B}
Albumin (gm/ dl)	3.19± 0.16	2.93± 0.16A	2.94± 0.12	3.29± 0.09	3.08± 0.12	2.75± 0.29	3.46± 0.12B	3.22± 0.28	3.12±0.17	3.30± 0.14
Globulin (gm/ dl)	3.40± 0.47^{a}	5.08± 0.59^{Ab}	4.81± 0.63^{A}	4.21± 0.52^{A}	4.91± 0.61^{Ab}	4.46± 0.34	3.15± 0.49^{B}	3.04± 0.37^{B}	2.43± 0.26^{B}	3.02± 0.32^{B}
A/G (ratio)	0.93^{a}	0.57^{b}	0.61	0.78	0.62^{b}	0.61	1.09	1.05	1.28	1.09
Liver enzymes										
SGOT/AST (IU/L)	65.43± 4.28^{a}	44.40± 11.50	48.54± 6.39	50.08± 2.95^{b}	40.59± 4.40^{b}	75.64± 7.11	50.57± 13.91	55.30± 4.34	48.69± 7.36	41.40± 2.44
SGPT/ALT (IU/L)	37.89± 13.49	31.04± 14.27	41.65± 17.12	14.99± 1.15	15.60± 4.50	25.34± 2.30	18.42± 3.73	24.88± 4.26	11.29± 1.64	17.95± 4.35
ALKP (IU/L)	65.24± 10.13^{A}	78.71± 22.39^{A}	50.93± 4.03	46.51± 8.18^{A}	81.75± 14.13^{A}	133.75± 22.00^{B}	170.06± 27.99^{B}	74.36± 12.86	71.38± 4.59^{B}	37.43± 7.00^{B}

Note: - 1. Mean±SE bearing different superscript (A, B) between treated and control group differ significantly ($p<0.05$).

2. Mean±SE bearing different superscript (a, b) within treated group differ significantly ($p<0.05$).

Table 8. Mean±SE value of serum minerals in camels at different intervals of treatment

Mineral	Treated group					Control group				
	0 day	15 day	30 day	45 day	60 day	0 day	15 day	30 day	45 day	60 day
Ca (mg/dl)	6.92± 0.29^{a}	7.61± 0.41	8.45± 0.17^{Ab}	9.17± 0.27^{Ab}	10.42± 0.31^{Ab}	6.63± 0.25	7.65± 0.37	7.59± 0.29^{B}	7.66± 0.32^{B}	7.68± 0.41^{B}
P (mg/dl)	4.89± 0.20^{a}	5.25± 0.16^{b}	5.47± 0.19^{A}	5.75± 0.21^{b}	5.96± 0.18^{Ab}	4.45± 0.39	4.45± 0.33	4.27± 0.37^{B}	5.08± 0.22	4.56± 0.25^{B}
Zn (µg/dl)	37.0± 6.0^{a}	41.0± 2.0	52.0± 5.0^{b}	51.0± 4.0	58.0± 3.0^{b}	52.0± 4.0	49.0± 5.0	44.0± 2.0	52.0± 5.0	54.0± 3.0
Cu (µg/dl)	47.0± 0.4^{a}	51.0± 2.0^{b}	55.0± 1.0A^{b}	51.0± 2.2^{A}	59.0± 1.2^{Ab}	45.0± 1.5	47.0± 4.9	48.0± 2.6^{B}	45.0± 1.2^{B}	47.0± 4.0^{B}
Co (µg/dl)	0.25± 0.056^{a}	0.42± 0.19	0.48± 0.15	0.48± 0.09	0.50± 0.06^{Ab}	0.17± 0.033	0.38± 0.079	0.41± 0.079	0.36± 0.056	0.31± 0.031^{B}
Fe (µg/dl)	34.0± 4.0^{a}	53.0± 3.0^{Ab}	56.0± 3.0^{b}	60.0± 3.0^{b}	75.0± 6.0^{Ab}	32.0± 5.0	42.0± 2.0^{B}	53.0± 4.0	55.0± 5.0	50.0± 3.0^{B}
ALKP (IU/L)	65.24± 10.13^{A}	78.71± 22.39^{A}	50.93± 4.03	46.51± 8.18^{A}	81.75± 14.13^{A}	133.75± 22.00^{B}	170.06± 27.99^{B}	74.36± 12.86	71.38± 4.59^{B}	37.43± 7.00^{B}

Note: - 1. Mean±SE bearing different superscript (A, B) between treated and control group differ significantly ($p<0.05$).

2. Mean±SE bearing different superscript (a, b) within treated group differ significantly ($p<0.05$).

in the treated group compared to control group (P≤0.05).

Biochemical parameters of the camels (Table 7)**:** Significant difference was observed on day 0, 30, 45 and 60 post treatment compared to control group. Within the treated group difference was significant (P≤0.05) on day 45 and 60 compared to day-0 of the treated group. Significant difference in albumin concentration was recorded on day 15 post treatment between treated and control group. No significant difference in albumin concentrations was recorded at any other stage of the treatment. No significant difference in the globulin concentration was recorded between treated and control groups on day-0. Whereas there was significant difference (P≤0.05) on day 15, 30, 45 and 60 between treated and control groups. Within the treated group there was significant difference (P≤0.05) on day 15 and 60 post treatment compared to day-0. No significant difference in the mean AST concentration was observed between treated and control groups. This difference became significant (P≤0.05) in the treated

group on day 45 and 60 as compared to day-0, of the treated group. No significant difference in ALT concentration was recorded in both pre-treatment vs. post treatment and treated vs. control groups. There was significant difference (P≤0.05) in alkaline phosphates concentration on day 0, 15, 45 and 60 between treated and control groups. But no significant difference in treated group was recorded compared to day-0.

Estimation of serum minerals (Ca, P, Zn, Cu, Co, F) (Table 8): No significant difference in the mean serum Ca concentration was recorded on day 0 and 15 of treatment between treated and control groups. Whereas significant (P≤0.05) increase was recorded in the treated group on day 30, 45 and 60, post treatment compared to control group. Within the treated group significant (P≤0.05) increase was recorded on day 30, 45 and 60 post treatment compared to day-0. Significant (P≤0.05) increase in the mean P concentration was recorded on day 30 and 60 in the treated group compared to control group. Within the treated group significant (P≤0.05) increase was recorded on day 15, 45 and 60 post treatment compared to day-0. No significant difference in Zn concentrations was recorded in the treated group compared to control group. Within the treated group significant (P≤0.05) increase was recorded on day 30 and 60 post treatment compared to day-0. No significant difference in Cu concentrations was recorded in the treated group on day 0 and 15 compared to control group. Whereas this difference became significant (P≤0.05), on day 30, 45 and 60 post treatment compared to control group. Within the treated group significant (P≤0.05); difference was recorded on day 15, 30 and 60 post treatment compared to day-0. Significant (P≤0.05) increase in the mean Co concentration was recorded on day 60 in the treated group compared to control group. Within the treated group significant (P≤0.05) increase was recorded on day 60, post treatment compared to day-0. No significant difference in Fe concentration was recorded on day 0, 30 and 45 of the treated group compared to control group. This difference was significant on day 15 and 60 post treatment, compared to control group. Within the treated group the difference was significant (P≤0.05) on day 15, 30, 45 and 60 post treatment compared to day-0 of the treated group.

A:G ratio became low in both the treatment groups (<0.8), either because of only increase in globulin concentrations or corresponding decrease in albumin concentrations which showed rectification of compromised immunity in the treated camels. In the control groups A:G ratios were comparatively higher (>1.0) and in the healthy camels the ratio was approx. 0.8 (Table 9).

Table 9. Mean±SE of haemato-biochemical-mineral profiles of healthy camels of the herd.

Erythrogram	Healthy male camels (n=5)	
	Mean	Range
RBCs (10^6/ mm^3)	9.44±0.28	9.1-10.1
Hb (gm per cent)	9.08±0.27	8.3-9.9
PCV (per cent)	36.4±0.51	35-38
TLC ($10^3/mm^3$)	15.5±0.94	13.5-18.3
Lymphocyte	42.4±1.21	39-45
Neutrophils	51.8±1.02	49-55
Eosinophils	2.6±0.24	2-4
Basophils	0.4±0.24	0-1
Monocytes	2.8±0.37	2-3
Protein profile		
T.P. (gm/dl)	7.17±0.22	6.5-7.62
Albumin (gm/dl)	3.18±0.17	2.79-3.82
Globulin (gm/dl)	3.99±0.34	2.68-4.63
A/G (ratio)	0.79	
Liver enzymes		
SGOT/AST (IU/L)	33.58±4.23	21.73-47.73
SGPT/ALT (IU/L)	17.99±1.91	12.05-21.73
ALKP (IU/L)	43.41±5.39	31.7-57.48
Ca (mg/dl)	10.65±0.54	9.22-11.2
P (mg/dl)	6.09±0.26	5.34-6.8
Zn (µg/dl)	68.90±15.17	40.1-119.02
Cu (µg/dl)	57.12±5.62	45.77-77.0
Co (µg/dl)	0.39±0.03	0.3-0.051
Fe (µg/dl)	80.26±7.28	53.0-91.0

Efficacy of mineral mixture feeding was seen in eliminating signs of pica in camels after 60 days of treatment as these animals stopped eating non food items whereas untreated control group animals were eating non food items.

Discussion

Surprisingly very high incidence (51.66%) of the pica was recorded in the camel herd studied. Comparatively low prevalence (9.48%) has been reported in camels by Sharma (2000) in the Bikaner region. Tuteja *et al* (2018) recorded increasing incidence of pica in camels over the years, based on the surveys' carried out from 2007 to 2015, in the Rajasthan state. Kachhawaha *et al* (2013) reported 41.8 per cent camel herders of southern Rajasthan

has problem of pica in their herds. Kachhawa *et al* (2019) conducted retrospective study of diseases of camel at teaching veterinary clinical complex of RAJUVAS, Bikaner from January 2013 to December 2017. Amongst digestive disorders pica was (13.6%) a primary disorder followed by simple indigestion (12%). The high prevalence of pica and simple indigestion might have been due to poor availability of quality feed particularly deficiency of minerals like calcium and phosphorus in the region due to drought conditions.

Common vices recorded in camels were osteophagia, geophagia, lithophagia and coprophagia. Studies by previous authors report geophagia (Vernacular 'mitti khana') (Gautam and Bansal, 1972; Sharma and Satija, 1974; Shamat, 2008) and osteophagia (Dioli and Stimmelmayr, 1992; Shamat, 2008) in camels. Oesophageal obstruction in camels has been reported due to non feed items (Ramadan and Abdin-Bey, 1990).

Poor body condition as recorded by general weakness reflected by thinness of the hump, roughness of the hair coat and anaemia (pale mucous membranes) was common in pica affected camels compared to unaffected camels. Symptoms of weakness, roughness of the hair coat and emaciation in pica affected camels has already been reported (Gautam and Bansal, 1972; Kachhawaha *et al*, 2013). Shen and Li (2010) reported 'emaciation ailment' in Bactrian camels as clinical sign of pica.

Faecal dry matter was found higher in the treated camels compared to controls. Lower DM content of controls could be defense mechanism to eliminate higher contents of silica in the faeces. Consumption of some amount of soils is obvious in all the camels, being raised in the sandy arid area. Because these camels browse on desert plants with sticking sand on the plant leaves and some of the sand do remains in harvested fodders of the desert.

Significant increase in the mean TEC was recorded in the treated camels compared to untreated controls on 60 days post treatment. Decrease in TEC has been reported in pica affected camels Singh *et al,* 1986; Singh, 1993; Beniwal and Singh, 2007). Decrease of Hb concentrations in present findings in the pica affected camels was in accordance with Singh *et al* (1986) and Beniwal and Singh (2007). In pica affected camels PCV values were less than 35% whereas in healthy camels PCV values were more than 36%. In treated camels PCV values were corrected to normal (37%). Decrease in PCV in pica affected camels has also been reported (Singh *et al*, 1986; Singh, 1993; Beniwal and Singh, 2007).

In the treated group TLC count decreased significantly post treatment compared to control group. Whereas no significant difference in TLC was observed post treatment compared to day 0, in the treated group. Singh (1993) reported that in the pica affected camels TLC remained unaffected. Comparative low levels of TLC in healthy camels have been recorded (Dongre, 2000; Sharma, 2000; Mali, 2002).

In the treated group significant decrease in lymphocyte count was observed on day 60 post treatment ($P \leq 0.05$) compared to control group. Contrary to the present findings, Singh (1993) reported that in the pica affected camels lymphocytes remained unaffected. Comparative low levels of lymphocytes (<40%) in healthy camels have been recorded (Soni and Aggarwal, 1958; Banerjee *et al*, 1962; Musa and Mukhtar, 1982; Gorakhmal *et al*, 2001; Mali, 2002). In present study lymphocyte count of more than 40% was seen in healthy camels (Nassar *et al*, 1977; Rezakhani *et al*, 1997; Dongre, 2000).

Post treatment significant increase in neutrophils count was recorded in the treated group compared to control group and compared to day-0 of the treated group. Neutrophils counts of more than 50% in healthy camels have been reported (Singh *et al*, 2000; Gorakhmal *et al*, 2001; Mali, 2002). In the present study mean neutrophils count increased post treatment. Contrary to the present findings, Singh (1993) reported that in the pica affected camels neutrophils count remained unaffected.

Significant difference in mean eosinophils count was recorded on day 15, 30, 45 ($P \leq 0.05$) and 60 ($P \leq 0.01$) in the treated group as compared to day 0 of the treated group. Variations in the eosinophils counts from 1.2 to 4.53 per cent have been reported (Musa and Mukhtar, 1982; Rezakhani *et al*, 1997; Dongre, 2000; Gorakhmal *et al*, 2001; Mali, 2002) in healthy camels.

No significant difference in mean basophils count was recorded both between treated vs. control groups and treated vs. treated. Basophils count of less than one per cent in healthy camels has been reported (Nassar *et al*, 1977; Musa and Mukhtar, 1982; Singh, 1993; Rezakhani *et al*, 1997; Dongre, 2000; Gorakhmal *et al*, 2001; Mali, 2002).

Significant difference in monocytes count was observed on day 30 and 45 post treatment in the treated group compared to control group ($P \leq 0.05$). In

the treated group significant difference in monocytes count was observed on day 45 and 60 post treatment in the treated group as compared to day zero value (P≤0.05). Contrary to the present findings, Singh (1993) reported that in the pica affected camels monocytes count remained unaffected.

In the present study, mean serum total protein increased up to 7.91 gm/dl on day 60 post treatment in the treated group. Comparable protein levels in healthy Indian camels (7.17±0.22) have been reported (Bansal *et al*, 1970; Rathod, 2006). Significant decrease in total protein was recorded on day 60 post treatment compared to day-0 in the controls. Serum TP levels decrease in pica affected camels (Singh *et al*, 1986; Singh, 1993). There occurred significant increase in globulin concentrations on day 15, 30, 45 and 60 in the treated group compared to control group. Post treatment difference in TP was mainly due to increased globulin, whereas albumin concentrations remained almost unaffected.

No significant difference in the mean AST concentration was observed between treated and control groups. This difference became significant (P≤0.05) in the treated group on day 45 and 60 as compared to day-0, of the treated group. No significant difference in ALT concentration was recorded in both pre-treatment vs. post treatment and treated vs. control groups. There was significant difference (P≤0.05) in alkaline phosphates concentration on day 0, 15, 45 and 60 between treated and control groups. But no significance difference in treated group compared to day 0. The pepsinogen is a protein secreted by the stomach as precursor of the pepsin, one of the main enzyme of the digestion. An elevation in blood pepsinogen in veterinary clinics is suggestive of gastrointestinal parasitism (Faye and Bengoumi, 2018). Significant increased levels of plasma gastrin and pepsinogen in pica affected camels having sand in their third compartment of the stomach have also been reported (Kataria and Kataria, 2006).

Significant increase in Ca was recorded in the treated group on day 30, 45 and 60 post treatment compared to control and day-0 of the treated group. Serum Ca levels increased significantly and reached up to 10.42±0.31 mg/dl. Mean serum levels of more than 10 mg/dl in the healthy camels have been reported by Bhatt and Kohli (1961). In the present study, there was significant decrease in the serum Ca level in the pica affected camels. Significant decrease in serum Ca level in the pica affected camels have also been reported by Gautam and Bansal (1972) and Beniwal and Singh (2007). Mehrotra and Gupta (1989) reported seasonal variation in Ca concentration from 7.1-18.3 mg/dl in serum of Indian camels.

Serum phosphorus level increased significantly after supplementation to the level of 5.96±0.18 mg/dl. In the present study, there was significant low serum P level in the pica affected camels. Significant decrease in serum P level in the pica affected camels has also been reported (Gautam and Bansal, 1972; Beniwal and Singh, 2007). P deficiency in Australian camel was commonly manifested by bone chewing (Manefield and Tinson, 1996). Blood and Radostits (1997) mentioned that P deficiency as primary etiological factor in depraved appetite in animals.

Within the treated group significant increase in Zn concentration was recorded on day 30 and 60 post treatment compared to day-0. Comparatively high Zn levels (>80µg/dl) in healthy camels have been reported (Shekhawat, 1983; Ghosal and Shekhawat, 1992; Dongre, 2000; Dixit *et al*, 2008). Serum Zn levels become low in pica affected camels (Singh *et al* 1986; Beniwal and Singh, 2007). Manefield and Tinson (1996) mentioned that 18-20 mg of ZnSo4 per day is sufficient for good health in camels.

Cu concentration increased significantly post treatment compared to controls and day-0 of the treated group. Similar mean Cu values (60µg/dl) in camels have been reported by Faye *et al* (2005). Comparative high Cu levels (>90 µg/dl) in healthy camels have been reported (Shekhawat, 1983; Ghosal and Shekhawat, 1992; Dongre, 2000; Dixit *et al*, 2008). Decreases in serum Cu levels in pica affected camels have been reported (Singh *et al*, 1986; Beniwal and Singh, 2007). In camels mineral supplementation increased blood Cu level (Faye and Bengoumi, 1997). Mohamed (2004) observed an increase in Cu concentration with age and Cu concentrations were higher in rainy season than dry season.

Significant (P≤0.05) increase in the mean Co concentration was recorded on day 60 in the treated group compared to control group and day-0 of the treated group. In comparison to the present findings higher mean Co values (14.87±1.38 µg/dl) has been reported by Dixit *et al* (2008). Faye *et al* (2005) recorded much lower Co values (0.08µg/dl) in camels. Singh *et al* (1986) reported decrease in serum Co concentrations in pica affected camels.

No significant difference in Fe concentration was recorded on day 0, 30, and 45 of the treated group compared to control group. This difference was significant on day 15 and 60 post treatment,

compared to control group. Within the treated group the difference was significant (P≤0.05) on day 15, 30, 45 and 60 post treatment compared to day-0 of the treated group. Higher serum Fe values (>100µg/dl) in healthy camels have been reported (Dongre, 2000; Saeed *et al*, 2004; Faye *et al*, 2005; Dixit *et al*, 2008). Singh *et al* (1986) reported decrease in serum Fe concentrations in pica affected camels. Ghosal and Mathur (1992) reported five per cent of the animals having serum Fe values less than 40 µg/dl indicated subclinical deficiency in Bikaner area of Rajasthan. Haris *et al* (1995) reported large proportion of Cu circulating in plasma is combined with serum glycoprotein, ceruloplasmin, which has ferroxidase action and is required to deliver Fe to circulation, so low Fe level might result from Cu deficiency. Vegad (1995) mentioned deficiency of Cu lead to Fe deficiency anaemia which is microcytic and hypochromic.

A:G ratio becoming low in treatment group (<0.8), either because of only increase in globulin concentrations or corresponding decrease in albumin concentrations shows rectification of compromised immunity in the treated groups. In the control groups A: G ratios were comparatively higher (>1.0) and in the healthy camels the ratio was approximately 0.8.

Haematobiochemical changes in pica affected camels like eosinophilia and monocytosis along with decreased serum globulin concentrations are suggestive of bowel inflammation in pica affected camels, may be due to eating of non food items leading to inflammation which in turn may lead to disturbed absorption of other nutrients. These haematobiochemical values became normal after treatment, which is suggestive of regression of bowel inflammation, might be due to stoppage in feeding non food items. Regression of bowel inflammation and non interference of non feed items in the bowel resulted in normal absorption of various nutrients.

Mineral mixtures supplementation to pica affected camels has also been recommended by Gautam and Bansal (1972). Pica in animals is of multi-etiological origin, deficiency of essential macro or micro mineral in animals may result in low production and poor health with symptoms of pica that can be corrected by suitable supplementation of specific mineral. The concept and advantages of using area specific mineral mixture have been extensively discussed throughout the world (Prasad and Gowda, 2005; Devasena *et al*, 2010; Singh *et al*, 2016; Sahoo *et al*, 2017 and Pandey *et al*, 2018). These studies suggested that supplementation of the most deficient minerals as area specific mineral mixture improved production and reproduction of animals. Thiophanate possess a broad spectrum anthelmintic activity against Strongyle type parasites commonly associated with pica in camels (Bali *et al*, 1978).

Conclusion

A very high incidence of pica in dromedary camels of the present study along with mineral imbalance indicated importance of feeding balanced ration along with providing area specific mineral mixtures.

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