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USE OF DEXMEDETOMIDINE AS SEDATIVE IN MALE DROMEDARY CAMELS (*Camelus dromedarius*)

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ABSTRACT

The study was conducted to evaluate the sedative, physio-haemodynamic and haemato-biochemical effects of two doses of dexmedetomidine in camels. The experiment was conducted on 6 adult male camels in a randomised cross over design. The camels were randomly divided into two treatment groups, Dex_1 (dexmedetomidine 2.5 µg/kg b.wt) and Dex_2 (dexmedetomidine 4 µg/kg b.wt). Sedation, analgesia, physio-haemodynamic parameters and haemato-biochemical parameters were recorded at T- 0 (pre-administration) and at T -15, 30, 60, 90 and T-120 minutes post-administration. The onset of sedation was quicker (6.10 ±0.44 min) in Dex_2 than (6.85± 0.45 min) Dex_1 group. The duration of sedation and complete recovery time in Dex_2 group (40.05 ± 1.47 min and 64.32 ± 1.72 min, respectively) was significantly ($P<0.01$) longer than Dex_1 group (25.85 ±0.97 min and 43.14 ± 1.13 min, respectively). The extent of sedation and analgesia was significantly ($P<0.05$) more prominent in Dex_2 than Dex_1 group. Heart rate and pulse rate decreased significantly ($P<0.05$) in Dex_2 group at 15, 30 and 45 min. interval but no significant difference was observed between the groups. Systolic and diastolic blood pressure increased initially in both groups and later followed a decreasing trend. Haematological studies revealed non-significant changes in haemoglobin, packed cell volume, total erythrocyte count, total leukocyte count and differential leukocyte count at various time intervals in both the groups. Biochemical parameters *viz*, blood glucose, AST, ALT, ALKP, serum urea nitrogen, creatinine and cortisol showed non-significant changes at various time intervals in both groups. The changes observed in physiological and haemato-biochemical parameters were transient and within the normal range in both the groups. It was concluded that dexmedetomidine 4 µg/kg b.wt (Dex_2) produced better sedation without significant alterations in vital parameters and therefore, may be employed safely for sedation and analgesia in camels.

Key words: Analgesia, camel, dexmedetomidine, sedation

An Indian dromedary camel is reared primarily for carting, draft, agricultural operation and transportation therefore are more prone to various types of injuries and surgical disorders. Drug-induced sedation and analgesia are used commonly in dromedaries for restraining, diagnostic procedures, to perform minor surgeries and as a preanaesthetic drug. There are various class of sedatives which have been used alone or as preanaesthetic agents to induce sedation and anaesthesia in camels. The α-2 adrenergic agonist are most commonly used sedative for this purpose eg xylazine, detomidine, etc. However, these have cardiopulmonary effects after intravenous administration in camels. The duration and intensity of these effects depend on the type and dose of α-2 agonist used (Khalil *et al,* 2019). Recently dexmedetomidine has become popular in canines and other animals as a sedative due to its high α2:α1 selectivity (Kamibayashi and Maze, 2000). It is considered as a full α-2 agonist, allowing the use of relatively high doses without the unwanted vascular effects resulting from stimulation of α-1 adrenoceptors (Ebert *et al,* 2000). It causes both sedation and analgesia with negligible respiratory and cardiovascular side effects (Salarian *et al,* 2016). In dogs, it produces dose-dependent sedation and analgesia (Kuusela *et al,* 2000). Use of dexmedetomidine as sedative in dromedary camels has not been reported previously. The present study is therefore undertaken to investigate sedative, physio-haemodynamic and haemato-biochemical effects of dexmedetomidine in male dromedary camels.

Materials and Methods

The experimental trials were conducted after approval of the experimental protocol by the Institutional Ethics Committee of National Research Centre on Camel.

The study was conducted on 6 adult male camels with a mean body weight of 435.38 ± 11.22

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kg and aged between 4-5 years in a randomised crossover design with an interval of 14 days between two treatments. Food and water were withheld for 24 and 12 hours, respectively prior to experiments. Camels were kept restrained in sitting position/ sternal recumbency with both fore limbs tied together and allowed for 15 minutes to be stabilised after restraining.

Two sedative doses of dexmedetomidine *viz*. 2.5 µgm kg^{-1} b.wt and 4.0 µgm kg^{-1} b.wt for achieving satisfactory sedation with fair to good analgesia were selected during pilot trials. The study was based on doses of dexmedetomidine administered hence selected adult male dromedary camels (n=6, each group) were divided into two groups, i.e. Dex_1 (dexmedetomidine 2.5 µg/kg b.wt) and Dex_2 (dexmedetomidine 4 µg/kg b.wt). Sedation and analgesia were evaluated by subjective analysis of median scores of sedation and analgesia within group and in between groups. Quality of sedation and analgesia was evaluated by observing sedative response and analgesia based on behavioural scoring method on a 0-3 scale (Table 1). The observations were recorded at T-0 minute (pre-administration) and at T -5, 15, 30, 45, 60, 90 and 120 minutes (min), post- administration.

The physio-haemodynamic parameters, i.e. rectal temperature (RT), respiration rate (RR), heart rate (HR), pulse rate (PR), systolic and diastolic blood pressure (SBP and DBP) were recorded at T-0 minute (pre-administration) and at T- 5, 15, 30, 45, 60, 90 and 120 min post-administration of drug.

Haemato-biochemical parameters were studied from blood sample collected at T-0 minute (pre administration) and at T-5, 15, 30, 45, 60, 90 and 120 min post-administration of drug. Haematological parameters *viz*. haemoglobin, packed cell volume, total leucocyte count, total erythrocyte count and differential leucocyte count were estimated using automated haemato-analyser (IDEXX Vet Test, IDEXX Lab). Biochemical parameters *viz*. glucose, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, serum urea nitrogen and creatinine were estimated with the help of semi-auto analyser (LT-100) using commercial kits. The cortisol level as a stress marker hormone was estimated as per standard procedures.

The data were presented as mean ± standard error of mean and median (Range) in parametric and non parametric statistical analysis, respectively.

Analysis of Variance (ANOVA), and Duncan's multiple range test (DMRT) were used to compare the means at different time intervals among groups and paired t test was used to compare the means at different time interval with respective base values (Snedecor and Cochran, 1994). The subjective data generated from the sedation scores were analysed using non parametric Kruskal- Wallis test. A value of $P < 0.05$ was considered significant.

Results and Discussion

The onset of sedation in Dex_2 group (6.10 ± 0.44 min) was recorded earlier than Dex_1 group (6.85 ± 0.45 min). The duration of sedation and complete recovery time was significantly ($P<0.01$) longer in Dex_2 (40.05 ± 1.47 min and 64.32 ± 1.72 min, respectively) than Dex_1 group (25.85 ±0.97 min and 43.14 ± 1.13 min, respectively) (Fig 1). The recovery was smooth and uncomplicated in both the groups. Moderate to good sedation was recorded in camels of Dex_1 group whereas, good sedation was recorded in Dex_2 group. The Dex_2 group camels showed marked sedation with significantly higher ($P<0.05$) sedation score particularly at 15, 45 and 90 min time points than Dex_1 group. The Dex_1 group camels exhibited moderate to good analgesia. Whereas, Dex_2 group camels showed good analgesia. The extent of muscle relaxation was significantly higher ($P<0.05$) in Dex_2 group particularly at 5 and 45 min time points than Dex_1 group (Table 2). Sedative effects of dexmedetomidine are attributed to its action on supraspinal autoreceptors in pons (Ahmed *et al*, 2018). Dexmedetomidine inhibited the release of substance P from the dorsal horn of the spinal cord, leading to primary analgesic effects (Kending *et al*, 1991). Dose dependent sedation by dexmedetomidine in Dex_1 and Dex_2 groups was in accordance with the findings of previous studies in dogs (Sabbe *et al*, 1994; Santosh *et al*, 2012). Kuusela *et al* (2001) reported that time to

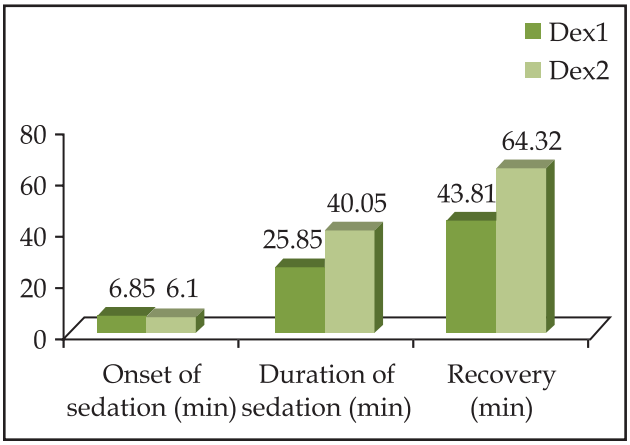

Fig 1. Onset of sedation, duration of sedation and recovery time after dexmedetomidine administration in camels.

Table 1. Description of sedation and analgesia score* in camel.

Score	Grade	Sedative response	Analgesia
0	No	Alert, normal carriage of head and neck, tongue and lower lip apposition and normal postural tone	strong reaction to bone prick
1	Poor	Slight reduction in alertness, mild head dropping, mild protrusion of tongue, Dropping of lower lip, slightly relaxed postural tone	weak reaction to bone prick
2	Fair	Moderately reduced alertness, moderate head dropping or deviation to one side, moderate protrusion of tongue and dropping of lower lip	occasional response to bone prick
3	Good	No alertness, deviation in posture, severe head dropping or deviated and rest on the back, severe protrusion of tongue, dropping of lower lip	no response to bone prick

*Adopted and modified (Khalil *et al,* 2019).

Table 2. Median values of sedation and analgesia score in camels of Dex_1 and Dex_2 group (n=6).

Parameters	Groups	Time interval							
		0	5	15	30	45	60	90	120
Sedation	Dex_1	0^{aA}	1.5^{abA}	2.5^{bA}	2.5^{bA}	1.5^{abA}	1^{abA}	0^{aA}	0^{aA}
	Dex_2	0^{aA}	2^{abB}	3^{bA}	3^{bA}	2.5^{bB}	2^{abA}	1^{abB}	0^{aA}
Analgesia	Dex_1	0^{aA}	1^{abA}	2.5^{bA}	2.5^{bA}	1^{abA}	1^{abA}	0^{aA}	0^{aA}
	Dex_2	0^{aA}	2^{acB}	3^{cA}	3^{cA}	2.5^{bcB}	1.5^{abcA}	0.5^{abA}	0^{aA}

Variables with different superscript in small letters differ significantly (P<0.05) within groups.

Variables with different superscript in capital letters differ significantly (P<0.05) among different groups.

Table 3. Mean±SE values of physio-haemodynamic parameters in camels of Dex_1 and Dex_2 group (n=6).

Parameters	Groups	Time interval							
		0	**5**	**15**	**30**	**45**	**60**	**90**	**120**
Rectal temperature (°F)	Dex_1	98.78 ± 0.56	98.76 ± 0.53	98.5 ± 0.44	97.93 ± 0.54	98.15 ± 0.49	98.28 ± 0.48	98.46 ± 0.48	98.26 ± 0.44
	Dex_2	98.45 ± 0.36	98.48 ± 0.30	98.08 ± 0.41	97.75 ± 0.45	97.33 ± 0.47	97.8 ± 0.50	97.66 ± 0.41	97.93 ± 0.42
Respiration rate (breaths min^{-1})	Dex_1	14.5 ± 0.67	14.0 ± 0.93	13.16 ± 0.79	13.5 ± 0.84	13.16 ± 0.90	13.33 ± 0.33	14 ± 0.77	14.16 ± 0.70
	Dex_2	14.16 ± 0.94	13.33 ± 0.95	12.17 ± 0.94	12.00 ± 0.77	11.67 ± 0.49	11.5 ± 0.67	12.83 ± 0.87	13.67 ± 0.84
Heart rate (beats min^{-1})	Dex_1	55.16 ± 3.14	52.16 ± 3.07	48.66 ± 2.98	48.0 ± 2.47	51.66 ± 2.47	52.00 ± 3.09	53.66 ± 2.81	55.66 ± 2.89
	Dex_2	54.66^{cA} ± 2.81	49.5^{bc} ± 2.04	43.33a ± 2.1	41.8a ± 2.34	44.33^{a} ± 2.81	46.66^{ab} ± 2.49	48.5^{abc} ± 2.18	53.16^{bc} ± 2.3
Pulse rate (beats min^{-1})	Dex_1	52.0 ± 2.59	49.16 ± 2.58	46.33 ± 2.92	46.16 ± 2.93	49.33 ± 3.25	49.66 ± 3.06	50.50 ± 3.77	52.33 ± 3.21
	Dex_2	51.83^{b} ± 3.14	46.0^{abc} ± 2.19	40.5^{a} ± 1.91	39.16a ± 1.66	42.5^{a} ± 2.43	43.67^{ab} ± 2.33	45.8^{abc} ± 2.0	50.33^{bc} ± 2.22
Blood pressure systolic (Mm Hg)	Dex_1	145.66 ± 4.97	152.33 ± 5.34	155.5 ± 5.35	145.16 ± 3.83	140.5 ± 4.16	143.0 ± 4.96	144.83 ± 4.76	145.16 ± 4.70
	Dex_2	144.33 ± 4.20	154.16 ± 5.36	156.33 ± 4.93	148.0 ± 5.68	140.83 ± 4.90	142.0. ± 5.13	143.5 ± 4.16	144.16 ± 3.51
Blood pressure diastolic (Mm Hg)	Dex_1	96.0 ± 4.80	102.83 ± 5.24	103.83 ± 5.81	94.67 ± 4.03	91.5 ± 4.0	93.5 ± 4.63	95.3 ± 4.8	95.83 ± 4.4
	Dex_2	95.33 ± 4.31	104.83 ± 5.34	106.83 ± 4.72	98.16 ± 5.64	91.83 ± 4.77	93.16 ± 4.72	94.16 ± 4.0	95.0 ± 3.6

sternal recumbency and walking were significantly prolonged in dogs treated with higher dose level, compared with the 2 lower dose levels in dogs.

Rectal temperature (RT) decreased non-significantly up to 30 and 45 min, respectively in Dex_1 and Dex_2 groups (Table 3). Decrease in rectal

Table 4. Mean ±SE values of haematological parameters in animals of Dex_1 and Dex_2 groups (n=6).

Parameters	Groups	Time interval					
		0	15	30	60	90	120
HB (g/dL)	Dex_1	12.73 ± 0.37	12.43 ± 0.32	12.31 ± 0.33	12.56 ± 0.30	12.53 ± 0.27	12.63 ± 0.41
	Dex_2	12.50 ± 0.34	12.3 ± 0.47	12.15 ± 0.30	12.10 ± 0.30	12.05 ± 0.27	12.21 ± 0.25
PCV (%)	Dex_1	27.71 ± 1.12	26.63 ± 0.92	25.93 ± 1.01	26.45 ± 1.19	26.61 ± 1.22	26.95 ± 1.00
	Dex_2	27.68 ± 0.98	27.00 ± 0.96	26.1 ± 0.89	25.66 ± 1.01	26.30 ± 0.93	26.86 ± 0.97
TEC ($10^6/mm^3$)	Dex_1	7.85 ± 0.46	7.79 ± 0.46	7.54 ± 0.52	7.30 ± 0.48	7.61 ± 0.45	7.59 ± 0.43
	Dex_2	7.93 ± 0.51	7.91 ± 0.52	7.50 ± 0.52	7.22 ± 0.49	7.12 ± 0.47	7.30 ± 0.48
TLC ($10^3/mm^3$)	Dex_1	13.26 ± 0.51	13.28 ± 0.51	13.10 ± 0.50	13.02 ± 0.54	13.21 ± 0.55	13.16 ± 0.57
	Dex_2	13.50 ± 0.45	13.49 ± 0.48	13.35 ± 0.50	13.22± 0.45	13.27 ± 0.44	13.30 ± 0.44
DLC							
N (%)	Dex_1	50.33 ± 0.80	50.67 ± 0.71	51.16 ± 1.01	50.66 ± 0.49	49.83 ± 0.70	50.00 ± 1.15
	Dex_2	51.66 ± 0.84	51.00 ± 0.44	51.83 ± 0.60	53.16 ± 1.24	52.16 ± 0.70	50.66 ± 0.49
L (%)	Dex_1	45.50 ± 1.17	45.50 ± 1.18	45.00 ± 1.41	46.50 ± 0.84	47.00 ±1.0	46.16 ± 1.47
	Dex_2	44.00 ± 0.89	44.33 ± 0.55	43.83 ± 0.60	42.50 ± 1.05	43.33 ± 0.76	44.16 ± 0.60
M (%)	Dex_1	2.83 ± 0.47	2.16 ± 0.47	2.00 ± 0.25	1.66 ± 0.21	2.00 ± 0.25	2.33 ± 0.33
	Dex_2	2.33 ± 0.33	2.00 ± 0.25	1.83 ± 0.40	2.33 ± 0.21	2.00 ± 0.25	2.33 ± 0.21
E (%)	Dex_1	2.00 ± 0.44	2.33 ± 0.33	2.83 ± 0.47	2.00 ± 0.25	2.33 ± 0.42	2.50 ± 0.42
	Dex_2	2.00 ± 0.25	2.66 ± 0.33	2.5 ± 0.34	2.00 ± 0.36	2.50 ± 0.42	2.83 ± 0.40

temperature in both groups might be attributed to reduction in muscular activity, decrease in metabolic rate, muscle relaxation with depression of thermo-regulatory system. Alpha-2 adrenergic agonist was reported to induce prolonged depression of thermoregulation (Ponder and Clarke, 1980). Similar observations after dexmedetomidine administration in dogs (Ahmad *et al,* 2011) and sheep (Monsang, 2011) were reported.

Respiration rate (RR) decreased non-significantly in animals of Dex_1 and Dex_2 groups (Table 3). Decrease in respiration rate might be due to depression of respiratory centres through stimulation of supra-spinal adrenoceptors following systemic administration of the alpha 2 agonist drug (Prado *et al,* 1999). A decreased RR results due to depressing action on respiratory centre in central nervous system (Hall *et al,* 2001). Decreased RR was also reported following administration of dexmedetomidine along with butorphanol in dogs (Surbhi *et al,* 2010) and dexmedetomidine along with propofol in dog (Singh *et al,* 2020).

In animals of Dex_1 group, heart rate and pulse rate decreased non-significantly whereas in animals of Dex_2 group, heart rate and pulse rate decreased significantly ($P<0.05$) at 15, 30 and 45 min from the base line value with a peak decrease at 30 min interval. Thereafter, heart rate and pulse rate increased significantly ($P<0.05$) at 120 min interval in animals of Dex_2 group (Table 3). Bradycardia was observed in animals of both Dex_1 and Dex_2 groups which is attributed to vasoconstriction property of alpha-2 agonists leading to reflex bradycardia (Congdon *et al,* 2011). Similarly, bradycardia was reported following dexmedetomidine administration in goats (Kastner *et al,* 2005) and camel calves (Samimi *et al,* 2020). Systolic and diastolic blood pressure increased initially at 5 and 15 min interval in both Dex_1 and Dex_2 groups and later followed decreasing trend with a peak low at 45 min. interval (Table 3).The increasing trend in the systolic and diastolic blood pressure followed by a decrease in both groups was similar to previous reports following administration of dexmedetomidine along with propofol in dog (Singh *et al,* 2020). This biphasic effect of dexmedetomidine on arterial blood pressure, with an initial rise in blood pressure followed by subsequent reduction in blood pressure might be attributed to change in plasma concentration of dexmedetomidine as reported earlier (Flaherty, 2013). Cardiovascular effects of alpha-2 agonists causes initial vasoconstriction and increased blood pressure mediated by the α2 b-subtype adrenoceptors and later causes decreased sympathetic tone and blood pressure (Kamibayashi and Maze, 2000).

In present study, complete blood count values remained within the normal clinical range in both groups of camels, however, non-significant changes

Table 5. Mean±SE values of biochemical parameters in animals of Dex_1 and Dex_2 group (n=6).

Parameters	Groups	Time interval					
		0	15	30	60	90	120
Blood glucose (gm/dl)	Dex_1	99.16 ± 6.22	117.5 ± 7.36	124.33 ± 7.55	121.16± 7.93	112.83 ± 9.7	107.16 ± 8.19
	Dex_2	102.5 ±6.08	119.83 ± 7.84	127.16 ± 8.46	127.83± 9.40	120.5 ± 9.91	111.50 ± 8.97
AST (IU/L)	Dex_1	90.82 ± 4.64	90.95 ± 6.09	92.71 ± 4.93	94.78 ± 5.96	94.63 ± 5.49	93.48 ± 5.11
	Dex_2	92.57 ± 5.92	94.08 ± 5.32	94.77 ± 5.66	95.25 ± 4.89	95.10 ± 5.27	94.55 ± 5.0
ALT (IU/L)	Dex_1	11.41 ±0.87	11.72 ± 0.92	11.81 ± 0.96	13.09 ± 0.80	12.94 ± 0.78	12.86 ± 0.77
	Dex_2	11.37 ± 0.89	11.85 ± 0.79	12.0 ± 0.70	13.36 ± 0.70	13.32 ± 0.68	13.34 ± 0.81
ALP (IU/L)	Dex_1	92.66 ± 5.89	92.83± 6.05	93.5 ± 6.13	95.5 ± 5.61	94.5± 5.64	93.83 ± 5.10
	Dex_2	94.33 ± 5.15	94.5± 5.05	96.0 ± 5.41	97.0 ± 5.24	96.5 ± 5.99	95.66 ± 6.07
SUN (mg/dl)	Dex_1	29.52 ± 1.76	29.55 ± 1.75	29.97 ± 1.89	30.78 ± 1.82	30.53 ± 1.74	29.83 ± 1.76
	Dex_2	29.46 ± 2.14	29.61 ± 2.21	29.68 ± 2.16	30.43 ± 1.98	30.61 ± 1.90	30.33 ± 1.91
Creatinine (mg/dl)	Dex_1	1.10 ± 0.15	1.13 ± 0.16	1.18± 0.14	1.20 ± 0.15	1.18 ± 0.14	1.15 ± 0.24
	Dex_2	1.06 ± 0.12	1.10 ± 0.13	1.15± 0.12	1.22 ± 0.12	1.23 ± 0.11	1.17 ± 0.12
Cortisol (µg/dL)	Dex_1	1.2 ± 0.14	1.11 ± 0.15	0.98 ± 0.12	0.90 ± 0.12	0.96± 0.09	1.06 ± 0.09
	Dex_2	1.16 ± 0.15	1.09± 0.14	0.97± 0.14	0.92 ± 0.12	0.97± 0.10	1.07± 0.08

were observed during the recording at different time periods. Haemoglobin (Hb), packed cell volume (PCV), total erythrocyte count (TEC) and toal leucocyte count (TLC) decreased non-significantly in both the groups (Table 4). It was attributed to shifting of body fluid from extravascular compartment to intravascular compartment in order to maintain normal cardiac output in the animals (Wagner *et al,* 1991). Pooling of circulatory blood cells in the spleen or other reservoirs secondary to decreased sympathetic activity might lead to a decrease in Hb, erythrocyte, PCV and TLC (Wagner *et al,* 1991). Decrease in Hb and PCV after administration of dexmedetomidine was also reported in dogs (Gupta, 2010) and sheep (Monsang, 2011). The initial increase in neutrophils and a corresponding decrease in lymphocytes recorded in present study might be associated with initial excitement due to handling of the animals and stress caused by the preanaesthetic drug and subsequent stimulation release of epinephrine leading to the release of neutrophils from bone marrow (Rosin, 1981). Similar findings were also reported following administration of dexmedetomidine in dogs (Ahmad *et al,* 2011).

In animals of Dex_1 and Dex_2 groups blood glucose increased non-significantly up to 30 and 60 min, respectively and later on, values decreased but remained above base line in both the groups (Table 5). Increase in blood glucose level observed in animals of both the groups might be attributed to increased hepatic glucose production, decreased glucose utilisation by body cells, decreased membrane transport and reduced plasma concentrations which are mediated by activation of α2-adrenoceptors present in the β-cells of pancreatic islets exerting a negative control of basal insulin release (Burton *et al,* 1977). Similar findings has also been reported following administration of medetomidine/ dexmedetomidine butorphanol followed by propofol anaesthesia in canine orthopaedic patients (Gupta, 2010), buffaloes (Malik *et al,* 2011) and sheep (Monsang, 2011). A non significant increase in aspartate amino transferase (AST), alanine amino transferase (ALT), and alkaline phosphatase (ALP) activity was observed up to 60 min in both Dex_1 and Dex_2 groups. Thereafter, values decreased but remained above base line in animals of both the groups (Table 5). Transient increase in ALT, AST and ALP levels might be associated with increased cell membrane permeability in response to haemodynamic changes induced by anaesthetic agents as result of oxidative transformation of these drugs in the liver during the process of elimination (Verma *et al,* 2018). A non significant increase was observed in serum urea nitrogen and creatinine level up to 60 min in animals of Dex_1 and up to 90 min in animals of Dex_2 group, later followed decreasing trend but remained above base line value. The increase in BUN and creatnine values in present study might be attributed to the temporary inhibitory effects of anaesthetic drugs on the renal blood flow and consequent decrease in glomerular filtration rate and increased urea production in liver (Kinjavdekar *et al,* 2000). Increased BUN and creatinine was also

reported following midazolam-dexmedetomidine in dogs (Santosh, 2011), xylazine propofol anaesthesia in dog (Surbhi *et al,* 2010) and dexmedetomidine, butorphanol and ketamine in dogs (Verma *et al,* 2018).

A non significant decrease in cortisol level was observed up to 60 min in animals of both Dex_1 and Dex_2 groups but later on followed an increasing trend (Table 5). Decrease in the level of cortisol in animals of both the groups might be attributed to direct inhibitory neuroendocrine response or indirect sedative and analgesic properties of dexmedetomidine which decreases the stress response when administered systemically as evidenced in previous studies (Raekallio *et al,* 2005). Alpha-2 agonists were known to influence the pituitary response and may decrease adrenocorticotropic hormone output (Masala *et al,* 1985). Similarly decreased cortisol level has also been reported following dexmedetomidine with etomidate and sevoflurane administration in dogs (Bisht *et al,* 2018).

It is concluded that the quality of sedation and analgesia remained dose dependent in animals of present study. Dexmedetomidine 4 µg/kg b.wt (Dex_2) produced a better sedation and analgesia. Transient changes recorded in physio-haemodynamic and haemato-biochemical profiles were within normal range, suggesting no deleterious effects on function of vital organs. Use of dexmedetomidine was found safe for sedation and analgesia in camels.

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