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Clinical observations of renal failure in dogs

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Abstract
In this study total 13060 dogs were presented in Veterinary Clinical Complex, PGIVER and Veterinary Polyclinic Jaipur. Total 161 dogs were found positive for renal failure on the basis of observation of classical clinical signs and azotemia [creatinine concentration more than 2mg/dl and BUN more than 30mg/dl] and out of 161 dogs, 30 dogs were selected randomly for research work and divided into 3 treatment groups on the basis of age. Randomly 10 healthy dogs negative for renal failure (after clinico-haemato-biochemical examination) were assigned as control group. Vomiting was the most common clinical sign (73.30%) while necrosis of the tip and lateral margins of the tongue was least common (40%). Decline in rectal temperature, increased pulse rate and respiration rate was found to be highly significant in dogs suffering from renal failure. Systolic blood pressure was also found significantly elevated in dogs with renal failure in comparison to control group.

Keywords: renal failure, dogs, BUN, creatinine

Introduction
Dogs serve us not only as companion but also as co-workers. In a number of tasks, they have demonstrated to be invaluable, including guide dogs for the blind, helping dogs for the handicapped, sniffer dogs used by police, army, railways, antiterrorist squad for security and customs, and protecting dogs used for farm job (Maruti, 2015) [27].
Dogs, in their life span, suffer from many bacterial, viral, protozoal, nutritional deficiency and systemic diseases (Chandler *et al.*, 1979) [3]. Kidney diseases are still one of the most common life disorders in adult and older dogs (Patil, 2011) [33].
Kidney diseases are common in dogs and cats, and are often associated with poor prognosis in later stages (Polzin, 2011) [34]. Acute kidney injury (AKI) is associated with a sudden onset of renal parenchymal damage with subsequent impairment of renal function. Severe injury can lead to acute renal failure (ARF), which is characterised by the highest morbidity and mortality rates. The high mortality associated with ARF is caused by delayed detection of this condition due to insensitive diagnostic tests (Cowgill and Langston, 2011) [6]. In ARF, clinical signs are non-specific and can include lethargy, vomiting, diarrhea, abdominal pain and decreased appetite. Initially affected animals urinate large amounts, followed by a decline in urine output (Pradhan and Roy, 2012) [36].
Chronic renal failure (CRF) is a common problem in aged dogs and is associated with significant morbidity and mortality. Several risk factors are attributed for CRF including old age, specific breeds, smaller body size, periodontal disease and obesity (O'Neill *et al.*, 2013) [31]. It results from a progressive and irreversible loss of functioning nephrons and the cause is often difficult to determine. Dogs with CRF exhibit polyuria, polydipsia, anorexia, vomiting, weight loss, pallor of mucous membrane, oral ulceration, halitosis and acute blindness (McGrotty, 2008) [28]. Most common clinical signs of CRF include polyuria and polydipsia along with nonspecific signs like anorexia, vomiting, lethargy, depression, fatigue, weakness, dehydration, diarrhea, tremor, ataxia (Pradhan and Roy, 2012) [36], lumber and abdominal pain, oral ulceration, halitosis and weight loss (Robinson *et al.*, 1989) [40].
Kumari and Haque (2014) [25] also reported subnormal temperature, elevated pulse and respiration rate in dogs with renal failure which was accordance to Mugford *et al.* (2013) [29], Kumar *et al.* (2011) [24], Ross *et al.*, (2006) [41] and Cowgill and Francy (2004) [9]. Hypertension was observed in renal failure (Koenhemsi and Gonul, 2019) [22].

Materials and Methods

Animals

In this study 13060 dogs were presented in Veterinary Clinical Complex, PGIVER and Veterinary Polyclinic Jaipur during study period. Out of these 13060 dogs, 161 dogs were diagnosed with renal failure on basis of observation of classical clinical signs and azotaemia (creatinine concentration more than 2 mg/dl and BUN more than 30 mg/dl). Out of these 161 dogs, 30 dogs irrespective to age, sex and breed were selected randomly for research work. 10 dogs were apparently healthy found negative for renal failure (after clinical and haemato-biochemical analysis) brought in canine outdoor of VCC for general health check-up constituted control group. The dogs (30) positive for renal failure were classified according to age group having 10 animals in each i.e. below 5 years of age dogs were included in group I, dogs between 5 to 10 years of age were included in group II and dogs above 10 years of age were included in group III.

Clinical observations

A complete clinical observation was done which included classical clinical signs (polyuria, polydipsia, vomiting, progressive weight loss, hair coat changes, alertness, halitosis, necrosis of the tip and lateral margin of the tongue and gradual loss of urine concentrating ability), temperature, pulse rate, respiratory rate, colour of mucous membrane and systolic blood pressure. All parameters were recorded.

Statistical analysis

The data obtained in research work were statistically analysed and compared as per the standard statistical procedures suggested by Snedechor and Cochran (1994) [44].

Results

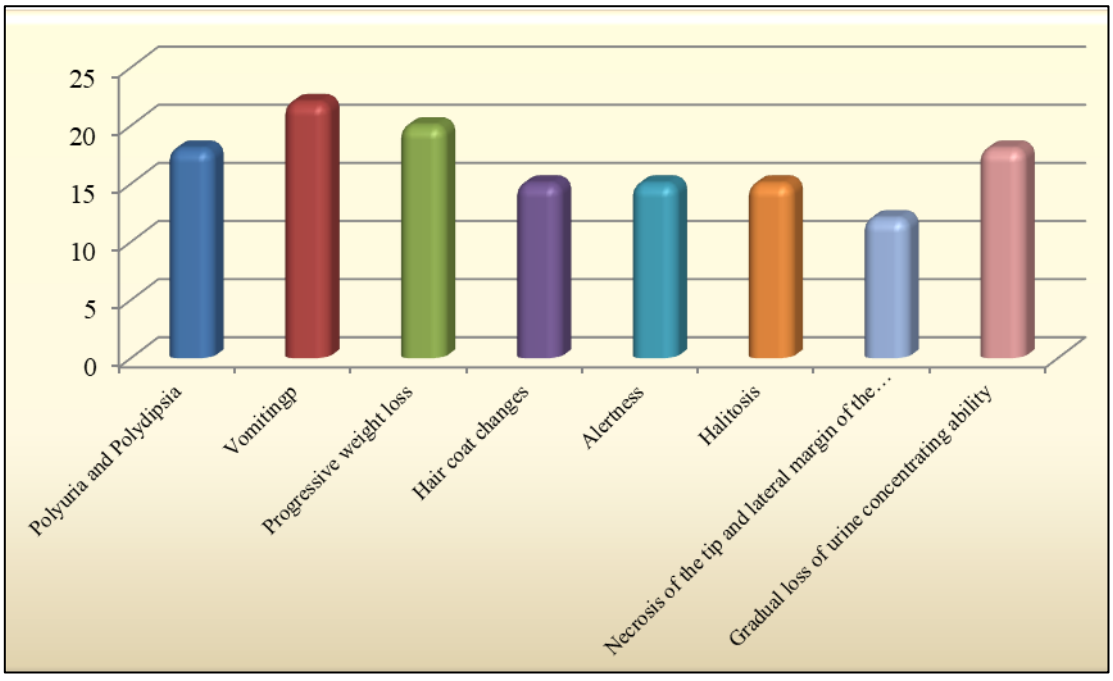
Clinical observations

A. Clinical signs

The percentage of dogs exhibiting different clinical signs at presentation and their discussion was portrayed below (Table No.1).

Table 1: Different frequencies of clinical signs observed in dogs suffered from renal failure

S. No.	Clinical signs	Frequency
1	Polyuria and Polydipsia	18 (60.0 %)
2	Vomiting	22 (73.3 %)
3	Progressive weight loss	20 (66.6%)
4	Hair coat changes	15 (50.0 %)
5	Lack of Alertness	Dull and depressed 15 (50.0%) Severely dull and depressed 6 (20.0%)
6	Halitosis	15 (50.0%)
7	Necrosis of the tip and lateral margin of the tongue	12 (40.0%)
8	Gradual loss of urine concentrating ability	18 (60.0 %)



Graph 1: Different frequencies of clinical signs observed in dogs suffered from renal failure

In present study polydipsia and polyuria were recorded in 60% of renal failure dogs. Vomition was observed to be pronounced clinical finding in dogs suffered from renal failure with a frequency of 73.3%.

Progressive weight loss was recorded in 66.6% renal failure dogs while hair coat changes were recorded in 50% of renal failure dogs. Dullness and depression were observed in 50% dogs and 20% dogs were severely dull and depressed suffering from renal failure.

Halitosis due to uremic breath was observed in 50% renal failure dogs. Necrosis of the tip and lateral margin of the tongue was recorded in 40% dogs and gradual loss of urine concentrating ability was recorded in 50% dogs suffering from renal failure.

Discussion

Result for polyuria and polydipsia resembles with the findings of Robinson *et al*., (1989) [40] who reported polyuria and polydipsia in 33% and 67%, respectively and IRIS (1998) [19], which reported both in 58.3% renal failure dogs. Polyuria and polydipsia has been reported as first clinical sign of CRF (Haller, 2002) [16]. Foster *et al*., (2013) [14] reviewed that common clinical signs of CKD include polyuria and compensatory polydipsia. Common clinical signs of renal failure include polyuria/polydipsia were also observed by O'Neill *et al*., (2013) [31], Rusenov *et al.,* (2014) [42], Devipriya *et al.,* (2018) [12] and Koenhemsi and Gonul, (2019) [22].

Suggestive reason of polyuria might be the direct, sublethal injury to the renal tubular cells which may perhaps cause loss of structural integrity, results in disorder of the actin cytoskeleton, disruption of cell polarity and loss of transporters. This dysfunction lead to massive solute loss in the urine, which produces an osmotic diuresis which can be manifested at any point during renal injury, but also during renal tubular cell recovery from sublethal injury or repopulation of tubules in which cells have undergone necrosis or apoptosis (Eatroff, 2012) [13]. Possible causes for polydipsia in renal failure includes vomiting, diarrhoea (Robinson *et al*., 1989) [40], chronic interstitial nephritis (Osborne *et al*., 1972) [32] and polyuria (Polzin, 2010) [35].

Result for vomiting resembles with the findings of Vidyadhar (2010) [47] who reported vomiting in 75% cases of renal failure in dogs. Gastrointestinal signs of renal failure include vomiting was also reported by Foster *et al*., (2013) [14], O'Neill *et al*., (2013) [31], Rusenov *et al.,* (2014) [42], Devipriya *et al*., (2018) [12] and Koenhemsi and Gonul, (2019) [22].

Reason of vomiting in renal failure might be uremic gastropathy and retention of uremic toxins (Quéau, 2012) [37], which have direct effect on dopaminergic receptors in the chemoreceptor trigger zone (Washabau and Elic, 1995) [48], other than this antibiotics and anti-inflammatory drugs may induce vomiting in some dogs (Quéau, 2012) [37].

Result for weight loss resembles with the findings of IRIS (1998) [19] reported weight loss in 69% of renal failure dogs. Common clinical sign of renal failure include weight loss was also reported by Haller (2002) [16], Polzin, (2011) [34], Foster *et al*., (2013) [14], O'Neill *et al*., (2013) [31], Rusenov *et al.,* (2014) [42], Mc grotty, (2014) and Devipriya *et al.,* (2018) [12] and Koenhemsi and Gonul, (2019) [22].

Possible reason of progressive weight loss was anorexia due to oral ulceration, gastritis and enteritis.

Result for hair coat change resembles with the findings of Vaden (2000) [46] who worked on renal failure and was reported clinical sign including thinness and poor coat quality were more common in CRF. Haller (2002) [16] also reported clinical symptom a poor condition of hair coat was common in renal disease in dogs. Common clinical sign of CKD include poor hair coat was observed in dogs with kidney damage (Polzin, 2011) [34].

Result for lack of alertness resembles with the findings of IRIS (1998) [19] who reported profound depression in 33.33% of renal failure dogs. Robinson *et al.,* (1989) [40] and IRIS (1998) [19] reported letharginess in 53% and 62.1% of renal failure dogs, respectively. Foster *et al*., (2013) [14], O'Neill *et al*., (2013) [31], and Rusenov *et al.,* (2014) [42] also reported letharginess in dogs with CKD.

Result for halitosis resembles with the findings of Cowgill and Francy (2005) and Kumar (2013), who reported uremic breath (halitosis) in 53.37 % cases of renal failure dogs. Haller (2002) [16] also reported clinical symptom of halitosis was common in renal disease in dogs. Common clinical sign of renal failure include halitosis was also observed by O'Neill *et al*., (2013) [31], Devipriya *et al.,* (2018) [12] and Koenhemsi and Gonul, (2019) [22].

Halitosis in renal failure results from bacterial degradation of urea to ammonia (Quéau, 2012) [37].

Result for necrosis of the tip and lateral margin of the tongue resembles with the findings of Krawiec (1996) who reported that gastrointestinal disorders in renal failure of dogs include oral ulceration and necrosis of the tip and lateral margin of the tongue in the dogs with renal failure. Foster *et al*., (2013) [14] reviewed that common clinical sign of CKD include gastrointestinal signs. Mc grotty, (2014) and Devipriya *et al*., (2018) [12] they observed oral ulceration in the dogs with renal failure. Koenhemsi and Gonul, (2019) [22] observed wounds in the mouth of dogs with renal failure.

Result for Gradual loss of urine concentrating ability resembles with the findings of Polzin, (2011) [34] who observed common clinical sign of CKD include Impaired urine concentrating ability was in dogs with kidney damage.

B. Clinical examination

Table 2: Mean±SE Values of Clinical Parameters

Groups	Rectal Temperature (°F)			Pulse Rate (beats/minute)			Respiration Rate (breath/minute)			Systolic Blood Pressure (mmHg)		
	0 day	07th day	14th day	0 day	07th day	14th day	0 day	07th day	14th day	0 day	07th day	14th day
Control group	102.51±0.08^{a}			84.2±0.48^{b}			29.1±0.37^{b}			130.3±0.42^{c}		
Treatment group-I	101.15±0.17^{bc}			90±1.085^{a}			30.2±0.32^{a}			178.5±0.56^{b}		
Treatment group-II	101.78±0.35^{b}	102 (N=3)	101.2 (N=1)	91±0.53^{a}	83.66 (N=3)	86 (N=1)	31±0.42^{a}	26.33 (N=3)	28 (N=1)	182.9±0.50^{a}	180 (N=3)	178 (N=1)
Treatment group-III	101.03±0.20^{c}	101.2 (N=1)	101.0 (N=1)	90.5±0.45^{a}	71 (N=1)	72 (N=1)	30±0.29^{ab}	26 (N=1)	27 (N=1)	178.8±0.55^{b}	178 (N=1)	176 (N=1)
Significance	**			**			**			**		

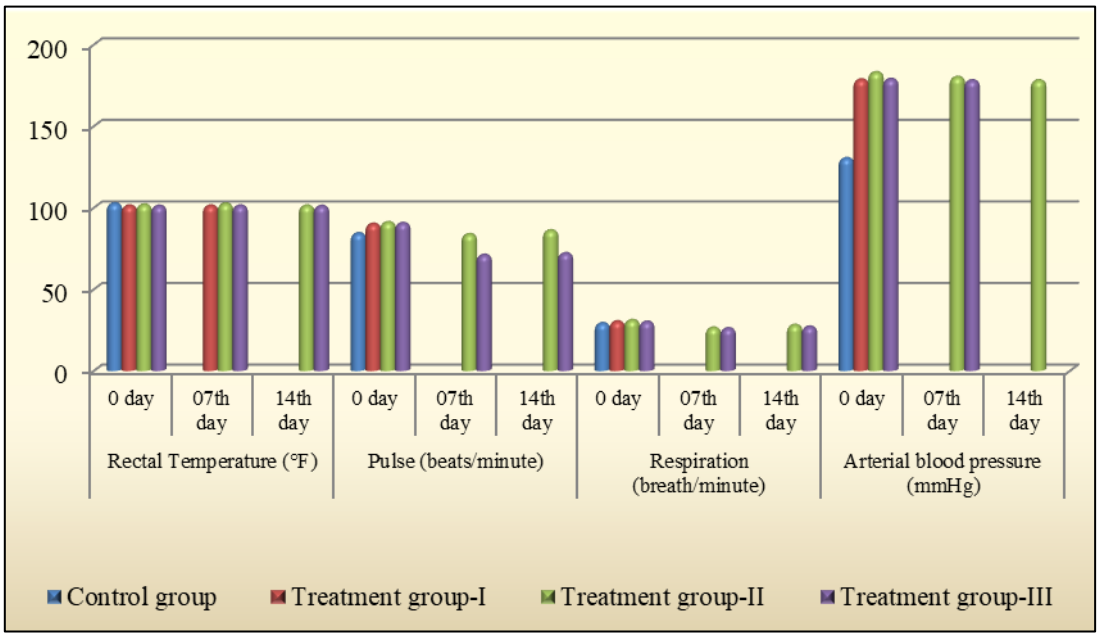
N = No. of Animal

Significant = 0.00 (Temp), 0.00 (Pulse), 0.007 (Respiration) and 0.00 (Arterial Blood pressure)

*(Significant) = P≤0.05

**(Highly Significant) = P≤0.01

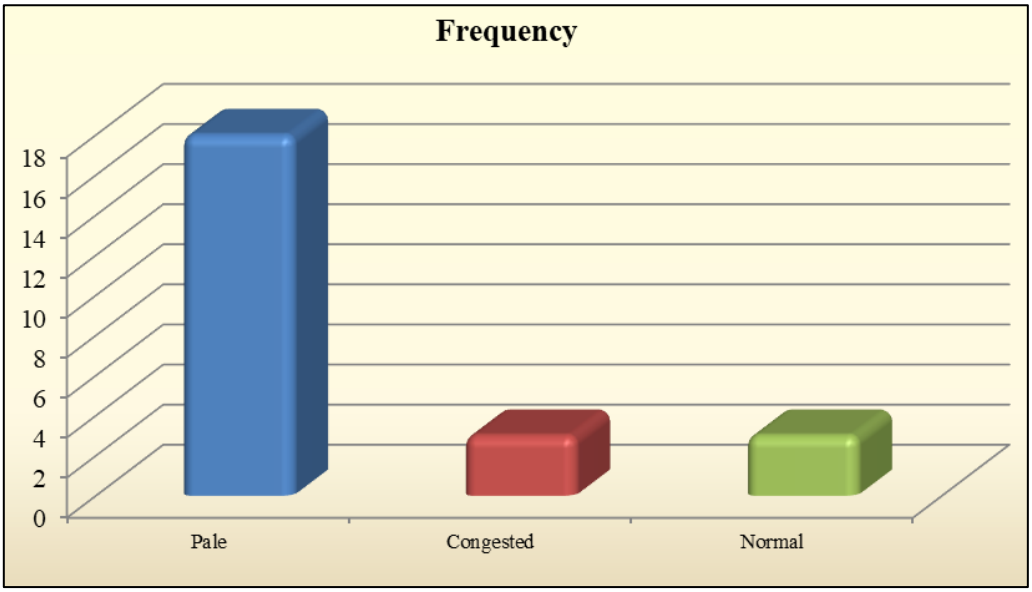
On 7th day & 14th day the significance of the difference between groups was not feasible to test due to less number of observations because of death of dogs.



Graph 2: Mean±SE Values of Clinical Parameters

Table 3: Frequency of Clinical Parameter (Colour of Mucous Membrane)

S. No.	Clinical parameter	Frequency
1	Mucous membrane (Colour)	Pale 23 (76.67%)
		Congested 5 (16.67%)
		Normal 2 (6.66%)



Graph 3: Frequency of Clinical Parameter (Colour of Mucous Membrane)

Rectal Temperature *(RT)*

In present study mean±SE values of rectal temperature (^{0}F) of dogs in control group, treatment group-I, treatment group-II and treatment group-III, were recorded 102.51±0.08, 101.15±0.17, 101.78±0.35 and 101.03±0.20, respectively on 0 day (Table No.2).

On 7th day mean values of rectal temperature (^{0}F) of dogs in treatment group-I, treatment group-II and treatment group-III, were recorded 0.00, 102 and 101.2 respectively.

On 14th day mean values of rectal temperature (^{0}F) of dogs in treatment group-I, treatment group-II and treatment group-III, were recorded 00.0, 101.2 and 101.0, respectively.

Highly significant decrease in mean rectal temperature on 0 day was observed in treatment groups as compare to control group. On 7th day & 14th day the significance of the difference between groups was not feasible to test due to less number of observations because of death of dogs.

Respiration Rate *(RR)*

In present study mean±SE values of respiration rate (breath/minute) of dogs in control group, treatment group-I, treatment group-II and treatment group-III, were recorded 29.1±0.37, 30.2±0.32, 31±0.42 and 30±0.29, respectively on 0 day (Table No.2).

On 7th day mean values of respiration rate (breath/minute) of dogs in treatment group-I, treatment group-II and treatment

group-III, were recorded 0.00, 26.33 and 26, respectively.
On 14th day mean values of respiration rate (breath/minute) of dogs in treatment group-I, treatment group-II and treatment group-III, were recorded 00.0, 28 and 27, respectively.
Highly significant increase in mean respiration rate (breath/minute) on 0 day was observed in treatment groups as compare to control group. On 7th day & 14th day the significance of the difference between groups was not feasible to test due to less number of observations because of death of dogs.

Pulse Rate *(PR)*
In present study mean±SE values of pulse rate (beats/minute) of dogs in control group, treatment group-I, treatment group-II and treatment group-III, were recorded 84.2±0.48, 90±1.085, 91±0.53 and 90.5±0.45, respectively on 0 day (Table No.2).
On 7th day mean values of rectal pulse rate (beats/minute) of dogs in treatment group-I, treatment group-II and treatment group-III, were recorded 0.00, 83.66 and 71, respectively.
On 14th day mean values of pulse rate (beats/minute) of dogs in treatment group-I, treatment group-II and treatment group-III, were recorded 00.0, 86 and 72, respectively.
Highly significant increase in mean pulse rate (beats/minute) on 0 day was observed in treatment groups as compare to control group. On 7th day & 14th day the significance of the difference between groups was not feasible to test due to less number of observations because of death of dogs.

Mucous Membrane
In present study mucous membrane (colour) was observed as pale, congested and normal with frequency 76.67, 16.67 and 6.66, respectively.

Systolic Blood Pressure (SBP)
In present study mean±SE values of s***ystolic*** blood pressure (mmHg) of dogs in control group, treatment group-I, treatment group-II and treatment group-III, were recorded 130.3±0.42, 178.5±0.56, 182.9±0.50 and 178.8±0.55, respectively on 0 day (Table No.2).
On 7th day mean values of s***ystolic*** blood pressure (mmHg) of dogs in treatment group-I, treatment group-II and treatment group-III, were recorded 0.00, 180 and 178, respectively.
On 14th day mean values of s***ystolic*** blood pressure (mmHg) of dogs in treatment group-I, treatment group-II and treatment group-III, were recorded 0.00, 178 and 176, respectively.
Highly significant increase in mean s***ystolic*** blood pressure (mmHg) on 0 day was observed in treatment groups as compare to control group. On 7th day & 14th day the significance of the difference between groups was not feasible to test due to less number of observations because of death of dogs.

Discussion
Hypothermia was recorded in present study which is in harmony with Joshi *et al*., (1989) [21] and Cowgill and Francy (2005) [10], they reported hypothermia in 45.83 % dogs. Kumari, and Haque (2014) [25] also reported subnormal temperature in dogs with renal failure which was accordance to Kumar *et al*. (2011) [24], Ross *et al.*, (2006) [41], Mugford *et al.*, (2013) [29]. Rusenov *et al.*, (2014) [42] reported hypothermia in dogs with CKD.
Possible reason for hypothermia might be nephrosis, depression and severe uremia where body temperature may reach up to 96^0F or lower (Larry and Elliott, 1995) [26]. Fever was observed in few dogs with possible cause of secondary bacterial infection.
Highly significant increase in mean respiration rate was recorded in present study which is in harmony with Shapiro (1997) [43] who observed tachypnea in cases of renal failure. Kumari and Haque (2014) [25] also reported elevated respiration rate in dogs with renal failure which was accordance to Ross (2006) [41], Kumar *et al.*, (2011) [24] and Mugford *et al*. (2013) [29].
Possible reason for increase in mean respiration rate might be severe metabolic acidosis produces tachypnea and increased tidal respiration (Kussmaul's respiration) in cases of renal failure (Shapiro *et al*., 1997) [43].
Highly significant increase in mean pulse rate was recorded in present study which is in harmony with Cowgill and Francy (2005) [10] they reported tachycardia in dogs with azotemia. Kumari and Haque (2014) [25] also reported elevated pulse rate in dogs with renal failure which was accordance to Ross (2006) [41], Kumar *et al.*, (2011) [24] and Mugford *et al.*, (2013) [29].
In present study mucous membrane (colour) was observed as pale and congested which is in harmony with Mc grotty, (2014) who observed clinical sign like pallor in dogs with CKD. Dogs with renal disease exhibited clinical sign such as pale mucous membranes (Devipriya *et al.*, 2018) [12].
Possible reason for congested mucous membrane of dogs suffered with renal failure might be due to uremia and fever (Clement *et al*., 1993) [4].
The mean value of SBP revealed hypertension in dogs suffered from renal failure, which is in agreement with Cowgill and Kallet (1983) [11], Cowgill (1986) [7], Bodey and Michell (1996) [1], IRIS (1998) [19], Rapoport and Stepien (2001) [38], Cortadellas *et al*., (2006) [5] and Buranakarl *et al*., (2007) [2], they reported hypertension in 9 to 93 % of dogs with renal failure. O'Neill *et al*., (2013) [31] also observed significant clinical sign included hypertension in dogs with CKD. Koenhemsi and Gonul (2019) [22] reported blood pressure measurements were slightly increased in dogs suffering from CRF. Hezzell *et al*., (2020) [18] reported systolic blood pressure measurements >160 mmHg in dogs suffering from renal failure.
Current recommendations for dogs suggest that SBP >160 mmHg or diastolic blood pressure >95 mmHg measured by any method are reasonable values at which concern is warranted (Cowgill, 2001) [8] and blood pressure more than 180/120 always have high risk and require treatment first followed by diagnostic work-up for underlying disease (HCP, 2002) [17]. The major mechanisms thought to contribute hypertension in renal failure include fluid retention, activation of the renin- angiotensin system; large quantities of secretion of rennin lead to the formation of angiotensin II which cause decrease in the ability of the kidneys to excrete sodium and water results in hypertension (Guyton and Hall, 2006) [15], and increased activity of the sympathetic nervous system (Neumann *et al*., 2004; Rennke and Denker, 2007) [30]. It has also been reported that dogs with high SBP are more likely to develop a uremic crisis and to die, and the risks of developing a uremic crisis and of dying increase significantly as SBP increase (Jacob *et al.*, 2003) [20]. Although, small increases in blood pressure have been documented with increasing age in dogs (Bodey and Michell, 1996) [1], Stepien and Rapoport (1999) [45] found non-significant correlation between age and blood pressure. Male dogs tend to have slightly higher blood

pressure readings than females but the difference is unlikely to be clinically significant (Bodey and Michell, 1996) [1].

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