

**CLINICAL AND THERAPEUTIC STUDIES OF HEAT STROKE  
IN CATTLE**

तापघात से ग्रसित गायों में नैदानिक और चिकित्सीय अध्ययन

**KIRAN**

B.V.Sc. & A.H.

**THESIS**

**MASTER OF VETERINARY SCIENCE**

**(VETERINARY CLINICAL MEDICINE)**



। पशुधनं नित्यं सर्वलोकोपकारकम् ।

**2016**

Department of Clinical Veterinary Medicine, Ethics & Jurisprudence  
College of Veterinary and Animal Science, Bikaner  
Rajasthan University of Veterinary and Animal Sciences  
Bikaner-334001

**CLINICAL AND THERAPEUTIC STUDIES OF HEAT STROKE  
IN CATTLE**

तापघात से ग्रसित गायों में नैदानिक और चिकित्सीय अध्ययन

**THESIS**

Submitted to the  
Rajasthan University of Veterinary and Animal Sciences, Bikaner  
In partial fulfillment of the requirements for  
the degree of

**Master of Veterinary Science**

**IN**

**Veterinary Clinical Medicine**

**FACULTY OF VETERINARY & ANIMAL SCIENCE**

**By**

**KIRAN**

**B.V.Sc .& A.H.**

**2016**

## **1. INTRODUCTION**

---

Cattle has been a cornerstone of Indian agriculture for centuries and has served as source of nutrition for farmer's families through milk and milk products, as well as providing draught animal power for both agricultural operations such as ploughing and tilling the land, as well as for transportation of goods. Nearly all basic necessities of life were woven around the cow, with contributions in all aspects of life, including farming and manure, food and nourishment, transport, fuel (burning of dried cow dung cakes) and medicinal usage of cow dung and cow urine (Dasgupta,1945).

According to 19<sup>th</sup> livestock census (2012) India has 299.98 million bovine populations. Cattle populations are 190.90 million among them; 151.17 million are indigenous while 39.73 million are exotic/cross

bred. Rajasthan has 13.32 million cattle among them 11.58 million are indigenous while 1.73 million are exotic/crossbred. Bikaner has 0.9 million cattle population. India is world's largest milk producer, producing 132.4 million tonnes in 2012, of which 45% was produced by cattle. Rajasthan contributes 10.50% of the total milk produced in India (BAHS, 2014).

Bikaner has a hot desert climate and extreme temperatures. In the summer season it is very hot when the temperatures lie in the range of 28–48.5 °C (82.4–119.3 °F). Bikaner has a subtropical desert, [arid](#) hot climate which leads to development of heat stroke in exotic and cross bred cattle as well as other livestock species ([www.BikanerClimatemps.com](http://www.BikanerClimatemps.com)).

Like all other mammals and birds, cattle are homoeothermic animals. Despite wide fluctuations in environmental temperature, they are normally capable of maintaining a relatively constant body temperature (i.e. between 38.4 and 39.0°C), which is essential to preserve the multitude of biochemical reactions and physiological processes that occur with normal metabolism. As environmental temperature increase, certain thermoregulatory responses (designed to stabilize body temperature) are initiated, including reduced feed intake, decreased activity and increased drinking, shade or wind seeking, increased peripheral blood flow, sweating and panting. However, these thermoregulatory activities may not be sufficient to maintain a normal body temperature during periods when ambient air temperature and humidity are particularly high (Vermunt and Tranter, 2010).

Heat stroke is a chief problem of high producing dairy exotic and cross breed cattle in summer season and especially when the environmental temperature and relative humidity are high and with prolonged exposure to direct sunlight (Grogan and Hopkins, 2002). Heat stress occurs when animals are exposed to environmental temperatures in excess of 25°C (the upper critical temperature), particularly in combination with high relative humidity or sunshine. High

humidity makes the sweating mechanism relatively ineffective, thereby making cattle unable to maintain their core body temperature (Vermunt and Tranter, 2010).

The temperature–humidity index (THI) can be used as an indicator of thermal climatic conditions. Temperature–humidity index values of 70 or less are considered comfortable, 75–78 stressful and values greater than 78 cause extreme distress with lactating cows being unable to maintain thermoregulatory mechanisms or normal body temperatures (Mc Dowell *et al.*, 1976).

Any exposure to an environmental temperature outside the thermally neutral zone induces in an animal a response which aims at maintaining deep body temperature within set limits. Death resulting from heat prostration occurs in most species when body temperature reaches 41.5–42.5 °C (Dougherty, 1971). If the body temperature rises at an increasing rate until it exceeds 42–43 °C there will be damage to the central nervous system and other structures (heat stroke) with possible fatal consequences (Bligh, 1985).

Effects of thermal stress vary among individuals according to breeds, production level, prior experience etc. (Kadzere *et al.*, 2002). *Bos indicus* (Zebu) cattle are more thermo tolerant than *Bos taurus* cattle due to possession of thermo tolerant gene (Hansen, 2004). Various studies under field conditions have demonstrated that body temperature increase with increasing environmental temperatures above 21 °C in European dairy breeds (Mc Dowell, 1958).

Heat stroke, the deadliest of heat illnesses, and is defined by a core body temperature above 40°C (104°F) (Lee-Chiong and Stitt, 1995). Heat stroke is a life-threatening condition characterized by hyperthermia, central nervous system abnormalities and varying degrees of organ dysfunction. It occurs when the normal thermoregulatory system fails (Tayeb and Marzouki, 1990; Drobatz and Macintire, 1996; Barrow and Clark, 1998; Bouchama and Knochel, 2002; Grogan and Hopkins, 2002). Affected cows attempt to reduce

heat load by reducing exercise, feed intake and lactation. They actively seek shade and wet areas (Wolfenson *et al.*, 2000).

Heat stroke affects almost all systems of the body include the CNS, gastrointestinal, cardiovascular, hepatobiliary, renal/ urologic, hematologic and muscular (Walters, 2002). Acute renal failure is common especially in the dehydrated animal; it can manifest as oliguric or polyuric renal failure and can be fatal. Central nervous system injury can occur as a result of direct thermal injury, respiratory alkalosis, thrombosis, or hypoperfusion from shock. Disseminated intravascular coagulation and hyperthermia lead to intraparenchymal hemorrhage that can result in seizures, thus sustaining hyperthermia (Roger, 2005). Direct thermal injury damages the gastrointestinal barrier. Hypo perfusion and hypoxemia results in necrosis, death, and sloughing of the mucosa, leading to bacterial translocation. Bacterial translocation can cause sepsis and systemic inflammatory syndrome. Direct thermal injury and hypoxemia from hypo perfusion also damage the liver. Heat stroke damages the muscles of the body, especially if it is the result of excessive exertion (Roger, 2005).

Rhabdomyolysis, due to muscle necrosis, is common and can exacerbate the acute tubular necrosis via dehydration, hypo perfusion, and pigment deposition (Walters, 2002). The thermal damage of the GI tract will predispose the patient to translocation of bacteria that can develop into systemic inflammatory response syndrome (SIRS) or septic shock (Davis, 2004).

The pulmonary system develops increased vascular resistance and cellular injury, leading to damage of the pulmonary endothelium. This causes non-cardiogenic pulmonary oedema and acute respiratory distress syndrome. The damage to the pulmonary endothelium, compounded with the decreased cardiac output and perfusion, creates a decrease in oxygen uptake by the tissues (Tabor, 2007).

Hyperthermia is an undesirable state because the metabolic rate may be increased up to 50% with a rapid depletion of liver glycogen

stores and increased metabolism of endogenous protein as a source of energy. The severity of metabolic disturbance is indicated by the degree of hypoglycemia and rise in blood non-protein nitrogen which occurs (Kelly, 1974).

In heat stroke as the body temperature rises animals become agitated and distressed, have labored open-mouth breathing and eventually collapse, convulse and die. Heat stroke that is not life-threatening leads to reduced milk production as much as 50 per cent and impaired reproductive performance, and may predispose to subclinical acidosis. It has been estimated that heat stroke causes severe economic loss in approximately 60% of the dairy farms around the world (Wolfenson *et al.*, 2000).

To reduce the effect of heat stroke feeding management, housing, facilities adjustments, and selection of tolerant breeds should be important. Rations should be adjusted to increase energy and protein intake while maintaining rumen environment and cow health. The purpose is to increase the quantity of grain fed and decreased the quantity of forage in the ration. Also, environmental modifications that help to alleviate heat stroke problems are structure orientation, structure ventilation, use of shades and use of cooling systems in different sections of the dairy farm. Evaporative cooling means a combination of wetting and forced ventilation drying the cow's coat to maximize the cooling effect. Milking parlors with adequate holding pens can employ the use of subsequent sprinkling and forced air in the pens. In dairies with adequate drainage and housing, evaporative cooling can be provided above the feed bunks in addition to or instead of in the holding pen (Armstrong, 1994).

Heat stroke is a medical emergency and treatment must begin immediately to ensure survival and decrease sequelae (Armstrong, 1996). The animal must be treated with total body cooling immediately, if a fatal outcome is to be avoided. In addition to total body cooling, treatment to control vascular collapse and shock should be instituted in

severe hyperthermia (greater than 107°F). Residual brain damage, especially the cerebellar syndrome and spinal cord lesions with motor neuron loss, may occur despite of prompt treatment in about 20% of cases and these are associated with higher morbidity (Mehta *et al.*, 1987; Grogan and Hopkins, 2002).

Keeping in view the huge economical losses from decreased production and mortality from heat stroke the present investigation entitled 'Clinical and therapeutic studies of heat stroke in cattle' was undertaken with the following objectives-

1. To study prevalence of heat stroke in cattle in and around Bikaner city.
2. To study clinical manifestations of heat stroke in cattle.
3. To investigate haemato-biochemical alterations in cattle with heat stroke.
4. To carry out the rationale therapeutic management of heat stroke in cattle.

## 2. REVIEW OF LITERATURE

---

The relevant literature related to proposed study is reviewed under the following headings-

### 2.1 Prevalence:

Randhawa *et al.* (2014) conducted a study in 85 clinical cases of summer hyperthermia in exotic and crossbred cows especially in South Western districts of Punjab *viz.* Mukatsar, Ferozpur, Bathinda, Faridkot, Sangrur and Mansa due to prolonged drought during summer of 2009 with highest prevalence during July to September and noted 90% morbidity and around 10% mortality. However, they didn't mention the overall prevalence. No previous literature related to prevalence of heat stroke in cattle was found.

### 2.2 The temperature-humidity index (THI)

Kibler (1964) reported that temperature-humidity index (THI) describes the effect of environment on animal's ability to dissipate heat. During drought, the ambient temperature and relative humidity frequently exceed the critical comfort level of temperature humidity index (72) resulting in elevated body temperature and panting.

Du Preez (2000) reported that temperature-humidity index (THI) was the best, simplest and most practical index (parameter) for measurement of environmental warmth which caused heat stress in dairy cattle.

Correa-Calderon *et al.* (2004) reported that heat stress is one of the most important stressors, and it could not be indicated only in terms of temperature. The THI combines temperature and humidity in a single value and has been widely used to quantify heat stress in dairy animals.

Vitali *et al.* (2009) observed that mortality of the animals was directly related with temperature humidity index (THI). Above certain break point, mortality rate in dairy cows was the highest in summer and the lowest in spring and reported that mortality in dairy cattle increases sharply when maximum and minimum temperature humidity index (THI) increased from 80 and 70, respectively. The same study specified 87 and 77 as upper maximum and minimum critical THI above which the mortality reached maximum.

Gantner *et al.* (2011) noted that the exotic Holstein crosses were uncomfortable above the temperature of 25°C depending upon the increase in temperature and increase in humidity. When the temperature humidity index was at 71 or below, cattle were in the thermal neutral zone or zone of comfort. Values ranging from 72 to 80 indicated mild stress whereas heat stress index values above 80 indicated severe stress.

### **2.3 Clinical signs:**

Mc Dowell (1958) carried out study under field conditions and demonstrated that body temperature increased with increasing environmental temperatures above 21<sup>0</sup>C in European dairy breeds.

Ingram and Legge (1969) noted in cattle that at high environmental temperatures when panting begin, respiratory rate increased but tidal volume decreased. In extremely hot environments when the body temperatures of cattle rise continuously, rapid shallow panting was suppressed by a second phase of slower deeper panting.

Mc Dowell (1972) observed in cattle that at high environmental temperatures, an increased respiratory rate was an important way of increasing heat loss by cattle and was usually the first visible sign of heat stress.

Kelly (1974) found that heat stroke caused depression of the nervous system, circulatory failure, the pulse becoming fast and irregular and laboured respiration in dogs.

Sharma *et al.* (1983) reported that reduction in milk production was one of the major economic impacts of thermal stress in dairy cattle. Decreased milk yield due to thermal stress was more prominent in Holstein than in Jersey cattle.

Conrad (1985) observed that voluntary feed intake was decreased by 10-35% when ambient temperature reaches 35°C and above. Mallonee *et al.* (1985) also reported that feed consumption by cows was reduced by 56% during the day time in outdoors as compared to cows kept indoors. Similar findings were observed by Beede and Collier (1986) who found decline in feed consumption by dairy cattle when average daily temperature reached 25 to 27°C.

Shafie (1985) reported that during a hot summer, the respiratory rate of Friesian and dairy Shorthorn cattle were increased.

Silanikove (2000) noted reduced feed intake, change in feeding patterns– more grazing at cooler times of the day, cattle stand rather than lie down, bunching or congregating in shade if it is available or around water when no shade is available, rapid shallow breathing; open mouth breathing with panting, sweating, increased saliva production, lack of coordination, trembling and increased water intake.

Mc Guban and Elizabeth (2001) revealed that heat stroke in human generally occurred with a hyperthermia of greater than 40.6 °C (105.1 °F) in combination with disorientation and a lack of sweating. However, in exertional heat stroke, the affected person could sweat excessively.

Walters (2002) reported excessive panting, brick red mucous membranes, vomiting, ataxia, hyper salivation, diarrhea and muscle tremors, with an episode of collapse or extreme lethargy in dogs affected with heat stroke. Elevated rectal temperature above 40.7°C, hyperemic mucous membranes, fast (due to vasodilation) or slow (cardiovascular failure) capillary refill time, increased respiratory effort, altered mentation, tachycardia with weak or irregular femoral pulse and possibly cutaneous or mucosa petechiae indicating disseminated intravascular coagulation were also noted.

Flournoy *et al.* (2003) reported in dogs that pulmonary system developed increased vascular resistance and cellular injury which lead to damage of the pulmonary endothelium. It caused non-cardiogenic pulmonary oedema and acute respiratory distress syndrome.

Srikanda and Johnson (2004) reported increased ( $p < 0.01$ ) rectal temperature and respiratory rate in all three breeds (Holstein, Jersey and Australian milking cows).

Roger (2005) reported Panting, hyper salivation, tachycardia, hyperemic mucous membranes with shortened capillary refill time, bounding pulses, and increased blood pressure in dogs suffered with heat stroke. The patient was excited and hyperactive, tachypnea and hyperventilation were noted. Severe dehydration, muscle tremors, seizures, collapse, and coma were also present. Further he also observed seizures, hypoventilation, coma, haemorrhage into the GIT, respiratory and cardiac arrest in dog suffering from heat stroke.

Beatty *et al.* (2006) reported that feed intake of *Bos taurus* was reduced whereas that of *Bos indicus* did not changed. Water intake for both *Bos taurus* and *Bos indicus* increased when wet bulb temperature (WBT) increased.

Abeni *et al.* (2007) reported that milk yield declined in cows that had rectal temperatures higher than 40°C.

Radostits *et al.* (2007) reported an increase in heart and respiratory rates with a weak pulse of large amplitude, sweating and salivation followed by a marked absence of sweating in cattle suffered with heat stroke. The animal became dull, stumbles while walking and tended to lie down.

Temizel *et al.* (2009) observed hyperthermia, ataxia, muscle tremor and depression in goats suffering from heat stroke.

Vitali *et al.* (2009) reported opened mouths and hard breathing, signs of lethargy with their heads down as well as increased salivation as early symptoms in cattle affected with heat stroke. Affected animals later on became too weak with flaccid muscles and eventually unable to rise.

Mohamed and Abdel-Hamid (2010) reported hyperthermia, panting, profuse salivation, lameness and reduced milk production in cattle suffering from heat stroke.

Vermunt and Tranter (2010) reported reduced exercise, feed intake and milk yield. Actively seek shade and wet areas, become agitated and distressed, have labored open-mouth breathing and eventually collapse, convulse and death in cattle affected with heat stroke.

The effect of heat stress on behaviour, some physiological parameters with nine goats of almost similar in age, sex and weight was studied by Alam *et al.* (2011). Further he observed that skin and rectal temperature had no significant differences among the treatment groups but respiration and pulse rate were increased with the increased of heat stress from T<sub>0</sub> to T<sub>8</sub> group (P<0.01). Significant

difference was found in standing time and lying time ( $P<0.01$ ) in experimental groups. There were significant changes ( $P<0.01$ ) in number of urination and defecation per hour but no significant changes was found in duration per urination in heat treated groups.

Chandrabhan *et al.* (2013) carried out a study in Karan fries cows and reported that forenoon and afternoon respiration rate and rectal temperature were increased by 4.67 and 3.67 breaths per min and  $0.87^{\circ}\text{C}$  and  $0.77^{\circ}\text{C}$ , respectively in growing Karan fries cows during summer over spring. The respective values increased in adult Karan fries cows to 2.83 and 6.67 breaths per min and  $0.79^{\circ}\text{C}$  and  $0.88^{\circ}\text{C}$  during forenoon and afternoon during summer over spring. Similar pattern of increase in skin temperature and pulse rate were observed during summer and hot humid season over the spring season.

Randhawa *et al.* (2014) reported panting, marked decrease in appetite and major loss of milk production as the common manifestations of heat stroke in cattle.

#### **2.4 Haematology:**

Gross and Siegel (1983) and Mc Farlane and Curtis (1989) reported that the neutrophil/lymphocyte (N/L) ratio was increased as a result of heat stress in cattle.

Walters (2002) reported elevations in packed cell volume due to dehydration. Disseminated intravascular coagulation was present (thrombocytopenia, increased fibrin degradation products, prolonged prothrombin time, and activated partial thromboplastin time) in dogs affected with heat stroke.

Al-Haidary (2004) observed significant increase in packed cell volume in Naimy breed of sheep exposed to heat stress. The higher

PCV values had been reported to be an adapted mechanism to provide water necessary for evaporative cooling process.

Srikanda and Johnson (2004) reported that total haemoglobin (g/dl) was elevated ( $p < 0.01$ ) in all three breeds (Holstein, Jersey and Australian Milking Zebu cows) suffering with heat stroke.

Abdalla *et al.* (2009) reported in goat suffering with heat stroke that highest values of erythrocyte count, packed cell volume and haemoglobin concentration were found during wet summer and the lowest values in dry summer. The total leukocyte count was not affected significantly by seasonal change in thermal environment. The seasonal changes in the ratios of neutrophils, lymphocytes and eosinophil's were not significant. The monocyte ratio was significantly higher during wet and dry summer, compared to winter value.

Alam *et al.* (2011) studied the effect of heat stress on blood parameters with nine goats of almost similar in age, sex and weight and found that amount of RBC, PCV, Hb and WBC were increased with the increased of heat stress ( $P < 0.01$ ). Neutrophil, eosinophil, lymphocyte and monocyte numbers increased significantly ( $P < 0.01$ ) in heat treated groups.

Chandrabhan *et al.* (2013) reported that the haematological parameters, *viz.* packed cell volume and haemoglobin were significantly higher during summer. The haematological parameters RBCs, WBCs, Hb and PCV were lower during afternoon compared to forenoon in both groups of cattle during different seasons.

Haque *et al.* (2013) studied the effect of acute heat stress on some haematological parameters in which young and adult Murrah buffaloes ( $n=6$ ) were exposed to 40°C, 42°C, 45°C for 4 h duration in climatic chamber and thermo neutral temperature (22°C). In packed cell volume and haemoglobin, there was an increasing trend with increase

of temperature, but there was no effect of age on these parameters. In case of total erythrocyte count there was no effect of temperature but adult animals had higher TEC. Total leukocyte count was significantly increased at 40°C and 42°C in young, but there was no effect of temperature on TLC in adult animals. Mean corpuscular volume and mean corpuscular haemoglobin values were similar in all the four temperatures groups in both young and adult animals. There was no effect of temperature and age on mean corpuscular haemoglobin concentration values.

Sreedhar *et al.* (2013) carried out study on the biochemical constituents of pure Sahiwal and Jersey × Sahiwal crossbred animals in tropical conditions. Six animals were selected from each category of Sahiwal heifers, Sahiwal cows and Jersey × Sahiwal crossbred cows. The haematocrit values of the above animals were recorded during the experimental period of 21 days and observed that haematocrit values (%) of Sahiwal heifers, Sahiwal cows and Jersey × Sahiwal cows ranged from  $29.17 \pm 1.22$  to  $68.00 \pm 1.06$ ,  $31.00 \pm 1.46$  to  $67.00 \pm 1.06$  and  $31.17 \pm 1.17$  to  $75.83 \pm 0.59$ , respectively. A non-significant difference was observed in haematocrit values of Sahiwal heifers and cows, whereas a significant ( $P < 0.01$ ) variation was recorded for Jersey × Sahiwal crossbred cows and concludes that haematocrit, levels of the Sahiwal cows were significantly ( $P < 0.01$ ) higher during the first three days of the experimental period and later declined to the normal range of the species. In the case of Jersey × Sahiwal crossbred cows, higher levels of haematocrit and the above serum constituents were observed for a period of six days, which later declined to the normal range of the species in tropical environment.

Rana *et al.* (2014) studied the effect of heat stress on blood parameters in indigenous sheep and found that the amount of red blood cell, hemoglobin and packed cell volume were increased significantly ( $p < 0.05$ ) with the increase of heat stress but the amount of

white blood cell had no significant difference ( $P>0.05$ ) among the treatment groups.

Abd-Alkareem *et al.* (2015) carried out a study in local Iraqi black female goats and noted that there was significant ( $P<0.05$ ) increase in red blood cell count, total white blood cell number and monocytes percentage in summer compared with spring and winter. Haemoglobin concentration and PCV percentage were significantly ( $P<0.05$ ) increased in summer compared with other seasons.

Chaudhary *et al.* (2015) studied the effect of temperature humidity index on lactating Surti buffaloes and concluded that it was sensitive indicator of heat stress in buffaloes. Higher THI was associated with significantly increased in rectal temperature, total erythrocyte count, haemoglobin, haematocrit and mean corpuscular haemoglobin concentration.

#### **4.5 Biochemistry:**

Johnson (1970) reported reduction in plasma potassium concentration during heat stress in cattle.

Davenport (1974) observed in cattle suffering from heat stroke that respiratory alkalosis is replaced later on by metabolic acidosis due to development of hyperlactaemia.

Kelly (1974) reported hypoglycaemia and rise in blood non-protein nitrogen in dog suffered with heat stroke.

El-Nouty *et al.* (1980) found that serum  $K^+$  concentrations were reduced in cows during prolonged heat stress. They suggested that reductions in serum  $K^+$  were due to loss of potassium through sweat. They also noted that during prolonged heat stress in cattle  $Na^+$  and  $K^+$  concentration were reduced in blood serum and urinary excretion of

$\text{Na}^+$  increased 80% in a hot environment, urinary excretion of  $\text{K}^+$  increased only 18%.

Mallonee *et al.* (1985) reported fivefold increase in potassium loss from the skin of cattle in unshaded areas compared to shaded areas during thermal stress under subtropical conditions.

Tucker *et al.* (1985) reported that hyponatremia, hypokalaemia, hypocalcaemia, hypomagnesaemia, hypophosphatemia, elevated levels of creatinine phosphokinase and glucose were found in cattle suffering with heat stroke.

Shapiro and Cristol (1987) noted that the respiratory alkalosis was replaced later on by metabolic acidosis due to the development of hyperlactaemia in cattle suffering from heat stroke.

Anonymous (1989) reported that the serum creatinine kinase, transaminase enzyme greatly elevated and clotting factor deficiencies were found in cattle suffering with heat stroke. Serum calcium was reduced due to calcium binding to damaged cells.

West *et al.* (1991) reported that both sodium and potassium urinary excretion increased in hot environment in cattle.

In human Stoneham and Price (1992) found that initially there were a respiratory alkalosis due to hyperventilation, followed by a metabolic acidosis due to hyperlactataemia suffered with heat stroke.

Sanchez *et al.* (1994a) reported that respiratory alkalosis resulted in the renal excretion of  $\text{H}^+$  being depressed and the urine excretion of  $\text{HCO}_3^-$  must be accompanied by a cation and found sodium and potassium were possibilities with  $\text{Na}^+$  being more likely in cattle suffering with heat stroke.

[Alzeer et al. \(1997\)](#) reported serum CK, AST, ALT, and LD were higher in heat stroke victims than controls ( $P < 0.05$ ). The most useful indicator was LD, followed by CK and AST as useful prognostic factors in heat stroke suffered man.

Randell and Ohio (2002) reported in human affected with heat stroke that serum alanine transaminase, aspartate transaminase and alkaline phosphate were elevated due to hepatocellular damage. Patients can also have metabolic acidosis or a mixed acid base disturbance. A patient that is in septic shock might exhibit the low or high white blood cell count and signs of multiple organ involvement.

Walters (2002) reported elevations in total protein due to severe dehydration, elevation in blood urea nitrogen (BUN) and creatinine as seen in acute tubular necrosis and usually peaks within 24 to 48 hours of heat stroke in dog. Rhabdomyolysis could cause marked increases in creatinine. Blood glucose is often extremely low, although this is an inconsistent finding. As heat stress progresses respiratory alkalosis develops. Patients were usually hypophosphatemic and hypokalemic due to hypovolemia.

[Srikanda and Johnson \(2004\)](#) reported in cows suffering from [heat stroke that there was](#) no effect on blood pH in Holstein and AMZ cows but lowered ( $p < 0.01$ ) blood pH from 7.42 to 7.34 in Jersey cows. Lowered ( $p < 0.01$ ) blood  $p\text{CO}_2$  (kPa), bicarbonate (mmol/L), base excess (mmol/L) and plasma chloride (mmol/L) in all three breeds. Increased ( $p < 0.01$ ) oxygen saturation (%) in Jersey and Australian milking Zebu cows but lowered it ( $p < 0.01$ ) in Holstein cows. Increased ( $p < 0.01$ )  $p\text{O}_2$  (kPa) in Holstein and Jersey cows but lowered it ( $p < 0.01$ ) in AMZ cows. Increased ( $p < 0.01$ ) plasma potassium (mmol/L) and calcium (mmol/L) only in Holstein and Jersey cows but lowered them ( $p < 0.01$ ) in AMZ cows. The plasma glucose (mmol/L) increased ( $p < 0.01$ ) in Holstein and AMZ cows but decreased ( $p < 0.01$ ) in Jersey

cows. Increased ( $p < 0.01$ ) plasma creatinine (mol/L) but lowered ( $p < 0.01$ ) plasma creatinine phosphokinase (IU/L), aspartate aminotransferase (IU/L) and blood urea nitrogen (mmol/L) in all three breeds.

Roger (2005) reported in dog that oliguria and renal failure caused acidemia, hypokalemia, inactivation of heparin, and electrolyte loss. Protein loss accompanies polyuria renal failure if the glomerulus is damaged. Mucosal ulceration and loss of the gastrointestinal barrier leads to fluid and protein losses and electrolyte imbalances. Liver failure includes hypoalbuminemia, hyper ammonia and hypoglycaemia. Widespread necrosis of muscle cells results in hyperkalemia, hypocalcaemia, lactic acidosis.

Varghese *et al.* (2005) noted high levels of CPK (1000 IU/l), metabolic acidosis, and elevated liver enzymes in human suffering with heat stroke.

Beatty *et al.* (2006) reported decrease in the partial pressures of carbon dioxide and bicarbonate in venous blood in cows suffered from heat stroke. Blood pH declined from 7.44 to 7.36 for *Bos taurus* ( $P < 0.001$ ) and from 7.44 to 7.38 for *Bos indicus* ( $P < 0.001$ ).

Abeni *et al.* (2007) observed in cattle that there was decrease in plasma glucose and cholesterol. Reduction in plasma ALP activity confirmed the role of this enzyme as a quick and reliable heat-stress blood marker.

Temizel *et al.* (2009) reported increased serum urea, creatinine, potassium, plasma lactate, aspartate aminotransferase and lactate dehydrogenase activity in goats post-exposure to heat stroke.

Bras (2010) evaluated the effect of environmental temperature on ruminal fermentation and on mineral levels of growing ruminants.

The heat-stressed animals presented higher levels of ammonical nitrogen in the ruminal liquid and a decrease in the phosphorus, sodium, potassium and zinc serum levels.

Hashim (2010) studied biochemical findings in human suffering with heat stroke which included elevated urea, creatinine, cardiac and skeletal muscle enzymes, myoglobin and troponin. There was also biochemical evidence of metabolic acidosis, respiratory alkalosis, and hepatic injury with elevated enzyme levels as well as abnormal hematological and coagulation indices.

Mohamed and Abdel-Hamid (2010) reported that serum Na, Cl, Ca, Mg, Zn and Fe were significantly reduced but P was increased. The total protein, albumin, cholesterol and cortisol were significantly reduced but the glucose and malonaldehyde were significantly increased in cow affected with heat intolerable syndrome.

Singh *et al.* (2012) reported decrease in serum potassium concentration of buffalo heifers exposed to heat stress and referred this decrease to potassium loss by sweating through surface evaporation.

Chandrabhan *et al.* (2013) reported that plasma enzymes (ALT, AST, ALP, LDH) activity were significantly higher ( $P < 0.05$ ) during summer and hot humid season than spring and winter in both growing and adult group of Karan Fries cattle.

Sreedhar *et al.* (2013) conducted study on the biochemical constituents of pure Sahiwal and Jersey × Sahiwal crossbred animals in tropical conditions. Six animals were selected from each category of Sahiwal heifers, Sahiwal cows and Jersey × Sahiwal crossbred cows. The biochemical constituents of the above animals were recorded during the experimental period of 21 days and found the serum glucose, urea and creatinine levels (mg/dL) of Sahiwal heifers, Sahiwal cows and Jersey × Sahiwal cows ranged from  $61.90 \pm 1.34$  to  $97.32 \pm$

0.63,  $58.61 \pm 1.20$  to  $96.90 \pm 0.65$  and  $59.26 \pm 0.58$  to  $113.33 \pm 0.71$ ,  $11.72 \pm 0.94$  to  $47.21 \pm 0.64$ ,  $11.70 \pm 0.65$  to  $45.44 \pm 0.42$  and  $14.00 \pm 0.58$  to  $63.99 \pm 0.41$  and  $1.25 \pm 0.07$  to  $9.81 \pm 0.13$ ,  $1.29 \pm 0.09$  to  $9.90 \pm 0.17$  and  $1.43 \pm 0.10$  to  $16.18 \pm 0.15$ , respectively. The average serum glucose values were significantly ( $P < 0.01$ ) different among all the experimental animals during adaptability. The serum calcium and phosphorus levels (mg/dL) of Sahiwal heifers, Sahiwal cows and Jersey  $\times$  Sahiwal cows ranged from  $10.59 \pm 0.29$  to  $27.17 \pm 0.29$ ,  $10.84 \pm 0.27$  to  $26.61 \pm 0.46$  and  $10.45 \pm 0.33$  to  $36.76 \pm 0.71$ ,  $5.85 \pm 0.05$  to  $18.91 \pm 0.21$ ,  $5.94 \pm 0.08$  to  $18.87 \pm 0.12$  and  $5.88 \pm 0.10$  to  $20.80 \pm 0.13$ , respectively. A non-significant difference was observed in serum calcium and phosphorous levels for Sahiwal heifers and Sahiwal cows, whereas a significant ( $P < 0.01$ ) variation was recorded for Jersey  $\times$  Sahiwal crossbred cows in relation to adaptability and concludes that glucose, urea, creatinine, calcium, phosphorus levels of the Sahiwal cows were significantly ( $P < 0.01$ ) higher during the first three days of the experimental period and later declined to the normal range of the species.

Abdel-Fattah (2014) observed that plasma  $\text{Na}^+$  concentration increased significantly ( $P < 0.001$ ) in both goat breeds (Balady and Damascus) while plasma  $\text{K}^+$  concentration decreased significantly ( $P < 0.05$ ) in Damascus breed suffering with heat stroke.

Choudhary *et al.* (2015) studied the effect of temperature humidity index (THI) on ten lactating Surti buffaloes and concluded that THI is a sensitive indicator of heat stress in buffaloes. Higher THI was associated with significantly increased ALT, urea, sodium, creatinine, and with significant decreases in glucose.

Jain *et al.* (2015) reported in human suffering from heat stroke that the transaminase SGOT and SGPT were increased. Whereas, the increase in concentration of LDH and CPK in heat stress samples were

significant. In acclimatized plasma samples the concentration of these parameters was seen to decrease and reach close to control value. This study suggests that SGPT, LDH and CPK levels can be used as prognostic enzyme in heat stress related disease. Their validation in human samples will help them to be established as biomarkers of heat stress and acclimation.

## **2.6 Rationale therapeutic management of Heat stroke:**

Magazanik *et al.* (1980) carried out a studied on dogs affected with heat stroke were cooled by immersion in water at temperatures varying from 1-25°C. At all water temperatures, cooling occurred at a much slower rate in comatose dogs than in conscious ones. Dogs cooled in tap water (15-16°C) had the same cooling rate as those cooled in ice water (1-3°C). At water temperatures above 18°C, cooling rates were considerably decreased and tap water cooled as efficiently as ice water.

Mallonee *et al.* (1985) studied the effect of feeding sodium and potassium in cattle suffered with heat stress and observed higher milk yield with 0.7% sodium than 0.46% sodium when dietary potassium was 1.6% of the dry matter. Based on these studies, he recommended 1.5-1.6% potassium, 0.45-0.6% sodium and 0.35-0.4% magnesium should be fed to cattle during warm. Beede and Shearer (1991) also showed a 3-9% increase in milk yield with diets supplemented with potassium and sodium.

Mehta *et al.* (1987) observed that intravenous normal saline solution and cooling by conduction and evaporation immediately lowered core temperature to <39.0°C in human suffering from heat stroke.

Mehta *et al.* (1987) and Grogan and Hopkins (2002) reported in cattle suffering from heat stroke that animal must be treated with total

body cooling immediately if a fatal outcome is to be avoided. In addition to total body cooling, treatment for vascular collapse and shock should be instituted with severe hyperthermia (greater than 107<sup>0</sup>F). Intravenous crystalloid solutions given at shock doses and glucocorticoid were indicated in an attempt to prevent permanent organ damage and disseminated intravascular coagulopathy.

Strickland *et al.* (1989) studied the effect of fans and sprinklers by using 454 litres water per cow per day with a 30 second sprinkler period every 5 minutes for cooling of cow in hot and humid climates and observed reduced respiration rates and increased feed intake with a corresponding increase in milk production of cows. Turner *et al.* (1989) also studied the effect of fan and sprinkler by using 314 liters water per cow per day with a 2.5 minute sprinkler cycle every 7 minutes for cooling of cow in hot, humid climates and found similar findings that reduced rectal temperatures, reduced respiration rates and increased feed intake with a corresponding increase in milk production of cows.

Use of fans and sprinklers in humid areas was recommended by Bray (1992) and Turner *et al.* (1992). Furthermore Bray (1992) indicated that as little as 95 liters water per cow per day could be sufficient for cooling the heat stressed cow. Turner *et al.* (1992) also indicated that 23 liters per cow per day could be adequate.

Lin *et al.* (1998a) studied the effect of cooling by using 171, 411 and 302 liters water per cow per day for 1994, 1995 and 1996, respectively in hot, humid climates and noted increased feed intake with a corresponding increase in milk production of cows.

Lin *et al.* (1998 b) carried out study on heat stressed cows by using fan and water system, fans usually run continuously above a set temperature (80<sup>0</sup>F or 26.7<sup>0</sup>C). Water was applied with either a

continuous fine mist of water to cool the area and or cows and intermittent cycles *viz.*, 1-3 minutes every 15 minutes, applied with sprinklers. Both types of systems were used throughout the U.S. but misters or foggers do not give consistent result in humid areas.

Walters (2002) reported in dog suffering with heat stroke that judicious use of intravenous fluids was warranted with care given to avoid fluid overload.

Mehta and Jaswal (2003) carried out a study in human and reported that heat stroke was a relatively immunosuppressed state and chances of secondary infection existed. So he recommended use of antibiotics in all such patients.

Davis (2004) observed in dogs affected with heat stroke that treatment should be initiated by cooling with cool water (the use of ice water is contraindicated) that could lower body temperature of animal before transporting the animal to the veterinary hospital. The cooling process should continue with the temperature measured every five minutes to avoid rebound hypothermia. Ice packs can be placed over large superficial vessels in the inguinal and axillary areas. Oxygen supplementation should continue until it has been determined that the dog can maintain arterial oxygenation.

Drobatz (2004) reported in dog suffering with heat stroke that if cardiovascular disease is unlikely, then administration of crystalloid fluids intravenously was the initial fluid of choice, which can be given in doses up to 90 ml/kg b.wt./hrs for the first 1 to 2 hours. Continuously assess perfusion status and titrate the rate and volume of fluid to effect. If several boluses of crystalloid intravenous fluids does not improve tissue perfusion and blood pressure, then administration of colloids should be considered and/ or use of positive inotropes or pressure agents (such as dobutamine, dopamine, or adrenalin). Fluid

needs of the patient presented with heat stroke were individual and needed to be balanced with treatment response, central venous pressure, electrolyte balance, acid-base imbalances, blood pressure monitoring, lung resuscitation and urine output rather than a strict fluid volume end-point goal. Hypoglycaemia was not unusual in the severely compromised dog with heat stress, due to the increased metabolic activity which consumes vast amounts of glucose as well as the liver's inability to produce enough glucose. Therefore, the patient could be suffering with a hypoglycaemic coma. An intravenous bolus of 0.25 to 0.5 g/kg b.wt. of 25% dextrose should be administered as a bolus. Dextrose should be added to the intravenous fluids to a 2.5% to 5% concentration if hypoglycaemia is present. If the patient continues to show abnormal signs of mentation after the correction of the hypoglycaemia then cerebral oedema may be present. Mannitol (0.5 g/kg b.wt. slow IV, ensuring a syringe filter is used) should be considered. Therapy for disseminated intravascular coagulation is controversial. The removal of the underlying cause is essential therefore it is important to cool the patient, give cardiovascular support through fluid therapy and reduce any further complication. Symptomatic therapy might include fresh frozen plasma intravenously and low dose heparin therapy (100IU/kg b.wt. every 4-6 hours) subcutaneously. Alternatively, 20 IU/kg b.wt. of heparin incubated with fresh frozen plasma might be administered. Aggressive fluid therapy should improve the patient's cardiovascular parameters; if this is not the case then sympathomimetic such as dopamine and dobutamine could be considered.

Mazzaferro (2004) observed in dogs suffering with heat stroke that broad-spectrum antibiotics such as the second generation cephalosporin, cefoxitin, ampicillin with enrofloxacin, and sometimes metronidazole could be administered to decrease bacteraemia. Non-nephrotoxic antibiotics could be administered, as renal compromise is

a serious concern in patients with heat stroke. Sucralfate and H<sub>2</sub> blockers such as ranitidine and cimetidine would help to treat gastric ulceration; whereas an antiemetic such as metoclopramide could be added to the fluids at a rate of 1 to 2 mg/kg b. wt./day.

Macintire *et al.* (2005) reported in dogs affected with heat stroke that synthetic colloids could be considered if total proteins are below 35 g/l. Anti-inflammatory doses of dexamethasone could also be considered, though care must be taken to reduce the possibility of further immune suppression, gastric ulceration and ischemic injury to the kidneys. Oxygen supplementation improves tissue perfusion and decreases the risk of ischemia; therefore; it could be beneficial even if the patient shows no signs of requiring oxygen.

Roger (2005) reported that corticosteroids could be given in dog suffered heat stroke because they stabilize membranes, suppress inflammation and cytokine production, and decrease the severity of endotoxemia, as well as have antipyretic properties.

Chakravarti (2006) reported that dexamethasone could be given to prevent cerebral oedema and shock. Ruminants should be provided with better quality feeds (grains) during warmer parts of day and course roughages during cool night.

Johnson *et al.* (2006) studied in human affected with heat stroke and found that many patients suffered from hypovolemic shock; therefore it was important to begin aggressive cardiovascular support through volume expansion, which would decrease blood viscosity and improve blood flow. The use of antipyretic drugs such as aspirin and carprofen is contraindicated, as these drugs will alter the hypothalamic thermoregulation set point and heat stroke patients already have a normal hypothalamic set point.

Radostits *et al.* (2007) carried out a study in cattle suffered with heat stroke and recommended that affected animals should be immediately placed in the shade and hosed on the midline of the back with cold water so that their coats are saturated. Fans should be immediately placed in front of the animal to promote evaporative cooling, and cooled water, with and without added electrolytes, should be made available for the animal to drink. The rectal temperature should be monitored frequently during cooling, and water application should be stopped when the rectal temperature has returned to normal. The intravenous administration of 0.9% NaCl is indicated in animals that are weak, recumbent or dehydrated.

Powell (2008) reported broad-spectrum antibiotics could be given to prevent sepsis from bacterial translocation and may include a combination of antibiotics effective against gram-positive, gram-negative and anaerobic bacteria.

Adenkola *et al.* (2009) studied the effect of ascorbic acid on haematology and serum biochemical responses of pigs transported for short journey (4 h) during the hot summer season. Sixteen pigs administered with ascorbic acid at a dose of 250 mg/kg b.wt. *per os* and individually served as experimental animals and 13 others each administered orally with sterile water served as control animals. The leucocyte count ( $15830.25 \pm 1063.08 \times 10^3/\mu\text{l}$ ) dropped ( $P < 0.05$ ) in the experimental pigs after transportation and the value was significantly ( $P < 0.05$ ) lower than the value of  $22010.69 \pm 1722.00 \times 10^3/\mu\text{l}$  obtained in the control pigs after the journey. The neutrophils: lymphocyte ratio obtained in the control animals increased from  $0.61 \pm 0.04$  before transportation to  $0.79 \pm 0.17$  immediately after transportation. There was a significant ( $P < 0.05$ ) increase in total protein post- transportation in all experimental and control groups, but the increase was higher in the control than the experimental group. Alkaline phosphatase and aspartate amino transferase values in the

control pigs increased immediately after transportation. This study indicated the beneficial effect of ascorbic acid administration on haematology of pigs transported by road during the hot summer season for short journey of 4 hour and also found beneficial effect of ascorbic acid to reduce the risk of adverse effects of transportation stress in hot summer on health.

Upadhyay *et al.* (2009) and Sirohi (2010) reported in cattle and buffaloes that increased frequency of feeding and small quantities of the ration given during the day, improved the dry matter intake and reduced heat stroke during summer. Adequate supply of cool, fresh and clean water is minimizes the effects of heat stroke in lactating cows and buffaloes.

Cragoe (2010) reported that a natural solution for heat stress in cattle was the use of live yeast cultures. Yeast cultures had been shown to stimulate dry matter intake, helping maintain performance and rumen health during heat stress. Research on the use of yeast cultures had shown to lower rectal temperatures of dairy cows during heat-stressed situations.

Kumar *et al.* (2011) recommended the use of antioxidant nutrient supplementation especially vitamins C, A, E, zinc and chromium to attenuate the negative effects of environmental stress in cattle.

Samal (2013) reported that sprinkling cows with water and subsequently blowing air over the cow with a fan caused evaporative cooling. The evaporative cooling decreased body temperature in cattle suffering with heat stroke

Randhawa *et al.* (2014) found that subcutaneous administration of three doses (750 mg, 5ml) of iodized oil given at 24 h interval was effective in decreasing rectal temperature of hyper thermic cattle.

### **3. MATERIALS & METHODS**

---

---

The present study was conducted as per following headings:

3.1 Animals

3.2 Anamnesis

3.3 Physical examination

3.4 Clinico-physiological observations

3.5 Screening criteria for cattle to be included in study

3.6 Inclusive criteria for the selection of cattle for heat stroke

3.7 Laboratory diagnosis

3.7.1 Collection of blood samples

3.7.1.1 Blood smears examination: For identification of protozoa

### 3.7.1.2 Haematological examination

- (i) Haemoglobin estimation
- (ii) Determination of packed cell volume (PCV)
- (iii) Total leucocyte count (TLC)
- (iv) Total erythrocyte count (TEC)
- (v) Total platelet count (TPC)
- (vi) Differential leucocyte count (DLC)
- (vii) Mean corpuscular volume (MCV), Mean corpuscular haemoglobin (MCH), Mean corpuscular haemoglobin concentration (MCHC)

### 3.7.1.3 Biochemical estimation

- (i) Serum sodium
- (ii) Serum potassium
- (iii) Serum inorganic phosphorus
- (iv) Serum calcium
- (v) Serum blood urea nitrogen
- (vi) Serum creatinine
- (vii) Serum creatinine kinase
- (viii) Serum lactates
- (ix) Serum lactate dehydrogenase
- (x) Serum aspartate aminotransferase (SGOT)

(xi) Serum alanine aminotransferase (SGPT)

(xii) Alkaline phosphatase (ALKP)

(xiii) Blood glucose

3.8 Therapeutic management

3.9 Temperature humidity index

3.10 Statistical analysis

### **3.1 Animals:**

The present study entitled “Clinical and Therapeutic Studies of Heat Stroke in Cattle” was conducted from August to September, 2015 on total of 800 cattle which were brought for treatment to Medicine Clinic of Teaching Veterinary Clinical Complex CVAS, Bikaner (9 cattle) and visit to local dairy farms in and around Bikaner city (7 cattle). Among them 16 cattle were found to be suffered with heat stroke. All the 16 cattle found suffered with heat stroke were exotic and cross bred.

### **3.2 Anamnesis:**

A detailed history was obtained from each of the owner which includes:

(i) Animal’s details: Breed (exotic and cross bred dairy cattle), age, sex, duration of ownership, management practices.

(ii) History of exposure to high environmental temperature

(iii) History of other illness in recent past

(iv) Diet: Type, time and quantities of food and water consumed on daily basis.

(v) Owner's observation: Details of earliest signs and symptoms, progress and nature of the disease.

### **3.3 Physical examination:**

A complete physical examination was carried out and status of all body systems was evaluated and noted.

### **3.4 Clinico-physiological observations:**

Clinical signs, temperature, pulse and respiration rate were noted daily before start of treatment.

### **3.5 Screening criteria for cattle to be included in study:**

1. High body temperature (sudden rise of body temperature).
2. Panting, hyper salivation, severe depression, muscle tremor and ataxia.
3. History of exposure to high environmental temperature and humidity.

The cattle showing high body temperature along with any one or more of above mentioned signs were included for the study.

### **3.6 Inclusive criteria for the selection of cattle for heat stroke:**

Two ml of fresh blood was collected in EDTA vial from jugular vein for haematological analysis. Blood smear was prepared and stained with Geimsa stain to visualize the slide under oil immersion microscope to exclude the infection of protozoan parasites like Babesia species, Anaplasma species, Thieleria species etc., simultaneously total leucocyte (TLC) count were estimated and the animals showing normal or below normal TLC and negative for protozoan parasites were considered for further study.

### **3.7 Sampling procedure:**

### **3.7.1 Collection of blood sample:**

After clinical examination of the cattle, blood samples were collected from ear vein of all diseased cattle for preparation of blood smears for detection of piroplasms.

For hematological studies blood samples were collected from jugular vein with all aseptic precautions in sterilized test tubes from 16 cattle suffered with heat stroke and ten healthy cattle. Blood was collected in sterile tubes having disodium salt of ethylene diamine tetra acetic acid (EDTA) as an anticoagulant added at the rate of 1mg/ml of blood as recommended by Jain (1986).

For biochemical studies, blood was collected in another sterile tube having no anticoagulant. The blood slants were made and incubated for one hour at 37<sup>0</sup>C. Blood clots were broken and tubes were centrifuged at 2,500 rpm for 30 min. The serum was pipette out in small Pyrex tubes and was kept immediately in the deep freeze at - 20<sup>0</sup>C till analysis.

Blood samples were collected from all heat stroke suffered cattle at three days interval, taking first day as pre-treatment day and 3<sup>rd</sup> day as post- treatment day for haematological and biochemical estimation.

Ten apparently healthy animals were taken to serve as control group, and their clinical examination and haemato-biochemical analysis was done.

**Note:** - Blood samples from healthy control cattle were taken once only. Same values of haemato-biochemical parameters have been presented for the purpose of comparison with heat stroke suffered cattle.

#### **3.7.1.1 Haematological examination:**

The blood samples were subjected to estimation of haemoglobin, packed cell volume, total erythrocyte count, total leucocyte count, total platelet count, differential leucocyte count, mean

corpuscular volume, mean corpuscular hemoglobin, mean corpuscular haemoglobin concentration. These parameters were analyzed as per the methods described by Jain (1986).

**(i) Haemoglobin (Hb):**

Hemoglobin was determined by Sahli-Hellige haemoglobinometer. Blood was drawn in Sahli's pipette up to 20 cubic millimeter mark. It was then transferred to haemoglobinometer tube containing 4-5 drops of 0.1N hydrochloric acid and mixed well. The tube was then kept for 5 minutes for the haemoglobin to change into acid haematin. The fluid was diluted with distilled water drop by drop and mixing after each drop until it matched to the colour of the standard comparison tubes. The haemoglobinometer tube was read to give the amount of haemoglobin in g/dl of the blood.

**(ii) Packed cell volume (PCV):**

For determination of packed cell volume, microhaematocrit method was adopted. Non-heparinised capillary tubes were filled with blood up to three- fourth of total length. The blood adhered over the end of capillary tubes was wiped off with the help of a moist filter paper. The opposite ends of tubes were sealed over the spirit lamp by rotating between the thumb and the index finger for 2-3 seconds over the flame near its base. After perfect sealing of the end, the tubes were centrifuged for 5 minutes at 12,000 rpm in microhaematocrit centrifuge machine.

After centrifugation, packed cell volume was determined with the help of a special microhaematocrit reader scale. The bottom of the red column of capillary tube was adjusted with the zero line and the plasma level was matched with the hundred lines and top of red column excluding buffy layer was read in per cent.

**(iii) Total erythrocyte count (TEC):**

The RBC pipette was filled up to 0.5 marks with the blood. The diluting fluid (Hayem's fluid) was drawn up to 101 marks. After shaking the pipette for the three minutes, the fluid in its stem was discarded. The counting chambers of the haemocytometer were carefully charged with the diluted blood after placing cover slip. It was ensured that blood cells were evenly distributed over the counting chamber and overloading was avoided. The red blood corpuscles present in the four corner small squares and one small central square of the large central square were counted under high power of the microscope.

### **Calculations**

Numbers of red blood cells per cubic millimeter were calculated after multiplying the number of cells counted by 10,000 according to the following formula:

$$\text{Total erythrocytes} = \text{Cells counted} \times 200 \times 10 \times 5 \text{ per cubic mm}$$

Where:

200 stands for dilution

10 stands for depth in mm

5 stands for the 1/5<sup>th</sup> of square millimeter counted

### **(iv) Total leucocytes count (TLC):**

The WBC pipette was filled up to 0.5 marks with blood and the WBC diluting fluid was drawn up to 11 marks. After shaking the pipette for three minutes, the fluid in its stem was discarded. Counting chamber of the haemocytometer was carefully charged with diluted blood after placing cover slip. The cells were counted under low power objective of the microscope in the large four corner squares of the haemocytometer.

### **Calculations**

The numbers of leucocytes in one cubic millimeter of blood were calculated by multiplying the total leucocytes counted by factor 50, according to the following formula:

Total leucocytes per cubic mm = 4 cells counted x 20 x 10

Where:

20 stands for dilution.

10 stands for depth in mm.

4 stands for the number of square millimeters counted.

**(v) Differential leucocytes count (DLC):**

1. The dried blood film was flooded with methyl alcohol for 10 min.
2. The slides were washed gently with tap water to remove alcohol.
3. Fixed slide was stained in working dilution of Geimsa's stain (1:10) for 30 minutes.
4. The slide was washed with tap water and dried in air.
5. A drop of cedar wood oil was placed on the smear and slide was examined under oil immersion lens of microscope for the differential leucocyte count. Neutrophils, eosinophils, basophils, lymphocytes and monocytes were differentiated and expressed into percentage.

**(vi) Mean corpuscular volume (MCV):**

This index expresses in fl (fentolitre, i.e.  $10^{-5}$  fl) the average volume of a population of erythrocytes as follow:

$$\text{Mean corpuscular volume (fl)} = \frac{\text{Packed cell volume (ml/dl)} \times 10}{\text{Red blood cells (million per } \mu\text{l)}}$$

**(vii) Mean corpuscular haemoglobin (MCH):**

This index expresses in pictograms ( $10^{-12}$  g) the weight of haemoglobin in an average erythrocyte of population of cells:

$$\text{Mean corpuscular haemoglobin (pg)} = \frac{\text{Haemoglobin (g/dl)} \times 10}{\text{Red blood cells (million per } \mu\text{l)}}$$

**(viii) Mean corpuscular haemoglobin concentration (MCHC):**

This index expresses the ratio of weight of the haemoglobin to the volume of the erythrocytes and unit is percentage or gram per deciliter of red cells. It can be computed as follows:

$$\text{MCHC} = \frac{\text{Haemoglobin (g/dl)} \times 100}{\text{Packed cell volume (percent)}}$$

**3.7.1.2 Biochemical estimation:**

Biochemical analysis of serum samples was done to estimate serum sodium, potassium, inorganic phosphorus, calcium, blood urea nitrogen, creatinine, creatinine kinase, lactate, lactate dehydrogenase, serum aspartate aminotransferase (AST), serum alanine aminotransferase (ALT), Alkaline phosphatase (ALKP) and blood glucose.

Blood urea nitrogen, creatinine, creatinine kinase, lactate, lactate dehydrogenase, serum aspartate aminotransferase (AST), serum alanine aminotransferase (ALT), Alkaline phosphatase (ALKP), inorganic phosphorus, calcium and blood glucose were determined by the Vet Test Chemistry Analyzer using kit supplied by Idexx laboratories, as per the manufacturer's subscribed procedure.

**Procedure of vet test chemistry analyzer using Idexx test kits.**

First of all power was connected and the analyzer was turned on, after 30 minutes power switching, the on screen instructions-species information, patient number etc. were entered. Selected vet test chemistry slides were inserted when instructed by analyzer. Samples were kept at room temperature before feeding and were inserted as per manufacturer's manual in vet test analyzer using automatic pipettor, connected with the analyzer. All the necessary instructions given by analyzer were followed. The results were displayed on screen as well as in the form of printed slip after

automatic analysis process of about 5 to 6 minutes, depending on the chemistries.

**(i) Estimation of serum sodium:**

Sodium was estimated colorimetrically by the modified method of Trinder (1951) with kits from Span Diagnostic Pvt. Ltd.

**Principle**

Sodium from the specimen was quantitatively precipitated as the triple salt of uranyl magnesium sodium acetate and the excess of uranyl salt reacted with potassium ferrocyanide to produce brownish colour. The intensity of brownish colour was inversely proportional to the sodium concentration in the specimen measurable colorimetrically. Reagents required, procedure and precaution were used as per the directives of the manufacturer of the kit.

**Calculation**

$$\text{Sodium (mmol/lit)} = \frac{\text{Absorbance (Blank)} - \text{Absorbance (Test)} \times 150}{\text{Absorbance (Blank)} - \text{Absorbance (std)}}$$

**(ii) Estimation of serum potassium:**

Serum potassium was estimated colorimetrically by the modified method of Trinder (1951) with kits from Span Diagnostic Pvt. Ltd.

**Principle**

Potassium ions from the specimen reacted with sodium tetra phenyl boron of the boron reagent to produce a colloidal suspension, which was stabilized in a special matrix. The extent of turbidity was measured in terms of absorbance and was proportional to potassium concentration. Reagents required, procedure and precaution were used as per the directives of the manufacturer of the kit.

Reagents required, procedure and precaution were used as per the directives of the manufacturer of the kit.

**Calculation**

$$\text{Potassium (mmol/lit)} = \frac{\text{Absorbance (Test)} \times 5}{\text{Absorbance (std)}}$$

### **3.8 Therapeutic management:**

All the confirmed cases of heat stroke were managed by following common therapy as per requirement:

#### **1. To manage the temperature:**

I. All affected animals were immediately brought indoors in a cool environment to avoid high humidity and the adverse effects of sunlight.

II. Immediately hydration of animal was made by isotonic saline solution (0.9% NaCl), Ringers lactate @ 20–50 ml/kg b.wt, IV based on degree of dehydration (Silanikove, 2000).

III. Afterwards they were sprayed with a water hose on the midline of the back with tap water (15-16°C) (Silanikove, 2000). Fans were immediately placed in front of the animal to promote evaporative cooling; this was done continuously until their body temperature reached 101.5°F (Radostits, 2007).

#### **2. To manage the shock:**

Dexamethasone @ 0.1mg/kg b.wt. IV was given immediately, repeated after six hour or followed as per the recovery of the animal (Roger, 2005).

#### **3. To manage the stress:**

Ascorbic acid @ 25 mg/kg b.wt. IV was given for 3 days to reduce the stress (Silanikove, 2000).

#### **4. To prevent secondary bacterial infection:**

Broad spectrum antimicrobial, Oxytetracycline @ of 10 mg/kg b.wt. IV was given for 3 days.

## **5. Supportive treatment:**

I. When temperature comes down to normal Vit B complex- Tribivet (Intas Pvt. Ltd.) containing Vit B<sub>1</sub> 50 mg, B<sub>6</sub> 50 mg and B<sub>12</sub> 500 mcg per ml, 10ml i/m was given for 3 days.

II. Antioxidant supplementation- Liq. Intavita-NH (Intas Pvt. Ltd.) containing Vit A 1200 IU, Vit D<sub>3</sub> 6000 IU, Vit E 48 mg, Biotin 50 mcg and Nicotinamide 3 mg per ml, 10ml orally daily for 10 days.

III. To boost up rumen microorganism- Probiotic bolus Triboost (Biotech Vety. Pharma. Pvt. Ltd.) containing Live yeast culture 3 gram, Live lactobacillus sporogenes culture 20 million CFU, Amino acid 2 gram, Liver extract 5 mg and Fungal diastase 50 mg per bolus, 2 bid for 3 days.

**6. Management of other associated clinical conditions.**

### **3.9 Temperature humidity index (THI):**

Ambient temperature and relative humidity were recorded by the standard thermometer and hygrometer, respectively. The THI was calculated as proposed by utilizing maximum temperature (°C) and average relative humidity (%) using the following equation as per Du Preez (2000).

$$\text{THI} = (0.8 \times T_{\text{max}}) + (\% \text{ average RH}/100) \times (T_{\text{max}} - 14.4) + 46.4$$

Where  $T_{\text{max}}$  was environmental temperature in °C

### **3.10 Statistical analysis:**

The data obtained in research work were statistically analyzed and compared as per the standard procedures suggested by Sendecor and Cochran (1994).

## 4. RESULTS & DISCUSSION

---

The present study entitled “Clinical and therapeutic studies of heat stroke in cattle” was conducted on sixteen clinical cases of cattle suffered from heat stroke brought to Medicine Clinic of Teaching Veterinary Clinical Complex, CVAS, Bikaner (9 cases) and visit to local dairy farms in and around Bikaner city (7 cases) during the period of August 2015 to September 2015. The presented cattle were investigated for absence of protozoan parasite, clinical signs such as hyperthermia, hyper salivation, panting, tachycardia, hyperemic mucous membrane and changes in the haemato-biochemical parameters. The investigation generated data along with the cattle detail were used for confirmation of heat stroke which provide basis for rational therapeutic management. The evident amelioration in the clinical signs as well as the complications were meticulously monitored, identified and recorded. The details of temperature humidity index, prevalence, clinico-haemato-biochemical parameters as well as the therapeutic evaluation were discussed in this chapter.

### 4.1 Temperature humidity index (THI):

Bikaner is situated in the middle of the Thar Desert with very little rainfall and extreme temperatures. The environmental temperature, average relative humidity and temperature humidity index of the study period are depicted in table-1.

Mortality of the animal is reported to be directly related with temperature humidity index (THI) above certain break point, mortality rate in dairy cows was the highest in summer and the lowest in spring and reported that mortality in dairy cattle increases sharply when maximum and minimum temperature humidity index (THI) increased from 80 and 70, respectively. The same study specified 87 and 77 as upper maximum and minimum critical THI above which the mortality reached maximum (Vitali *et al.*, 2009).

**Table 1: Environmental temperature, relative humidity and temperature humidity index.**

<b>Month</b>	<b>Environmental temperature (°C)</b>	<b>Relative humidity (per cent)</b>	<b>THI</b>
	Mean (Range)	Mean (Range)	Mean (Range)
August	39 (37-41)	61 (59-65)	92.60 (89.78-95.42)
September	40 (36-42)	52 (48-56)	91.71 (86.43-94.35)

#### **4.2 Prevalence:**

Eight hundred cattle were screened during study period on the basis of history, clinical sign and thorough clinical examination. Out of these 800 cattle, 16 were found to be suffered with heat stroke on the basis of history, clinical examination, blood smear and haemato-biochemical examination.

The results have been presented in table-2. Prevalence of heat stroke in 800 cattle during August 2015 to September 2015 was studied. The overall prevalence of heat stroke amongst cattle was found to be 2.00 per cent. The finding of present study was in agreement with findings of Randhawa *et al.* (2014) who observed highest prevalence of summer hyperthermia from July to September. The incidence can vary depending upon the breed, age, climatic conditions and management practices.

**Table 2: Prevalence of heat stroke in cattle.**

S.No.	Time period	Total no. of diseased cattle	No. of cattle suffered with heat stroke	Per cent Prevalence
1.	August to September	800	16	2.00

#### **4.3 Clinical manifestations:**

All the sixteen cattle suffered with heat stroke were reported to be exposed to high environmental temperature from last few days with additional history of marked decrease in appetite, reducing exercise, actively seek shade and wet areas, labored open-mouth breathing and major loss of milk production and depression were found.

The clinical signs of heat stroke suffered cattle revealed hyperthermia, panting, tachycardia, hyper salivation, congested mucous membranes, ataxia, muscle tremor and severe dehydration in cows. Clinical manifestations recorded in the present study are in agreement with those reported by Kelly (1974), Srikanda and Johnson (2004), Roger (2005), Beatty *et al.* (2006), Abeni *et al.* (2007), Radostits *et al.* (2007), Temizel *et al.* (2009), Mohamed and Abdel-Hamid (2010), Vermunt and Tranter (2010), Chandrabhan *et al.* (2013) and Randhawa *et al.* (2014)

**Table 3: Major clinical manifestations of heat stroke suffered cattle.**

<b>S.No.</b>	<b>Clinical manifestations</b>	<b>Suffered cattle (n=16)</b>	<b>Frequency (%)</b>
1.	Hyperthermia	16	100
2.	Panting	16	100
3.	Tachycardia	16	100
4.	Congested mucous membrane	10	62.50
5.	Hyper salivation	16	100
6.	Ataxia	6	37.50
7.	Muscle tremor	5	31.25
8.	Severe dehydration	10	62.50
9.	Milk loss	16	100
10.	Decreased appetite	16	100

**4.3.1 Rectal temperature, pulse rate and respiration rate (<sup>0</sup>F, per minute and per minute, respectively):**

The mean±SE value of temperature, pulse rate and respiration rate in heat stroke suffered cattle are presented in table-4, figure-1, 2 and 3, respectively and relative data are presented in appendix-3. The mean±SE values of temperature, pulse rate and respiration rate in heat stroke suffered cattle were 107.52±0.1450<sup>0</sup>F, 113.69±2.9892 per minute and 115.50±2.4859 per minute, respectively. Corresponding mean±SE values for healthy control group are given in table-4 and data are presented in appendix-2. The mean±SE values of temperature, pulse rate and respiration rate in healthy control group were 101.94±0.1950 <sup>0</sup>F, 62.20±1.2792 per minute and 24.50±0.9618 per minute, respectively.

There was highly significant increase ( $P<0.01$ ) in temperature, pulse rate and respiration rate (Table-3, Figure-1, 2, 3 and Appendix-3) in heat stroke suffered cattle as compared to healthy control cattle. Similar findings were recorded by Kelly (1974), Srikanda and Johnson (2004), Roger (2005), Radostits *et al.* (2007), Temizel *et al.* (2009), Vermunt and Tranter (2010), Chandrabhan *et al.* (2013) and Randhawa *et al.* (2014).

The mean $\pm$ SE value of temperature, pulse rate and respiration rate in heat stroke suffered cattle after treatment are presented in figure-1, 2, 3 and appendix-4. It was  $102.07\pm 0.0794$  °F,  $65.12\pm 0.8564$  per minute and  $35.19\pm 0.9066$  per minute, respectively. There was highly significant decrease ( $p<0.01$ ) in temperature, pulse rate and respiration rate as compared to its pre-treatment level  $107.52\pm 0.1450$  °F,  $113.69\pm 2.9892$  per minute and  $115.50\pm 2.4859$  per minute, respectively.

The mean $\pm$ SE value of temperature and pulse rate on 3<sup>rd</sup> day of treatment had no significant difference ( $p<0.01$ ) as compared to healthy control while respiration rate on 3<sup>rd</sup> day of treatment had significant difference ( $p<0.05$ ) as compared to mean $\pm$ SE value of pulse rate of healthy control. Respiration rate was significant higher as compared to healthy cattle.

In the present study temperature, pulse rate and respiration rate were significantly increased in heat stroke suffered cattle as compared to with healthy control cattle.

The high rise in temperature may be attributed to cattle exposed to thermal environment, especially in hot conditions. Low humidity and air movements are important to allow evaporation of sweat and convection of heat (Jessen, 1977; Damanhoury and Tayeb 1992; Bricknell, 1995; Bruchim *et al.*, 1999-2004 and Al-Tamimi, 2007). Dehydration and increase of electrolyte concentration in the body fluid

of cattle exposed to heat reduced their thermoregulatory evaporation and allow the body temperature to rise. This readjustment in thermoregulation has been observed in both panting and sweating and appears to be a regulated response that allows the dehydrated animal to save water. In species that both pant and sweat, such as the cattle, progressive dehydration leads to suppressed sweating and increased panting (Silanikove, 1987). At high environmental temperatures, an increased respiratory rate is an important way of increasing heat loss by cattle and is usually the first visible sign of heat stress (Mc Dowell, 1972). Tachycardia may be associated with translocation of blood from the central circulation to the periphery in an attempt to get rid of heat, or it might have resulted from the increased production of nitric oxide (Howorth, 1995 and Alzeer *et al.*, 1999).

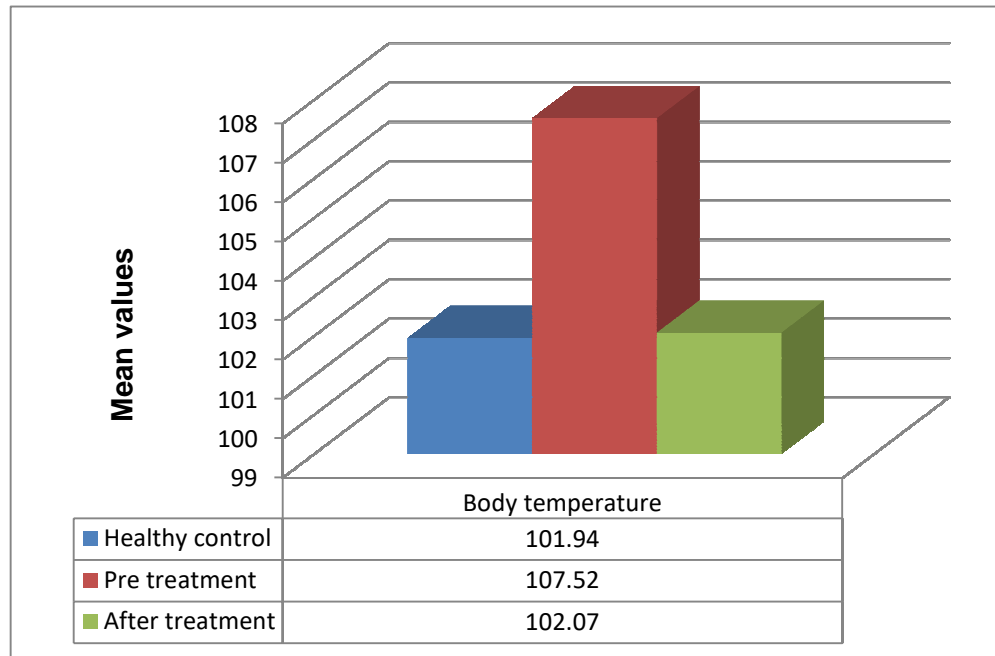
**Table 4: Mean±SE values of body temperature (°F), Pulse rate (per minute) and Respiration rate (per minute) in healthy control and heat stroke suffered cattle.**

S.No.	Parameters	Healthy control (n=10)	Before Treatment (n=16)	After treatment (n=16)
1	Body Temperature** (°F)	101.94±0.1950 <sup>a</sup>	107.52±0.1450 <sup>b</sup>	102.07±0.0794 <sup>a</sup>
2	Pulse (Rate/min)**	62.20±1.2792 <sup>a</sup>	113.69±2.9892 <sup>b</sup>	65.12±0.8564 <sup>a</sup>
3	Respiration** (Rate/min)	24.50±0.9618 <sup>a</sup>	115.50±2.4859 <sup>c</sup>	35.19±0.9066 <sup>b</sup>

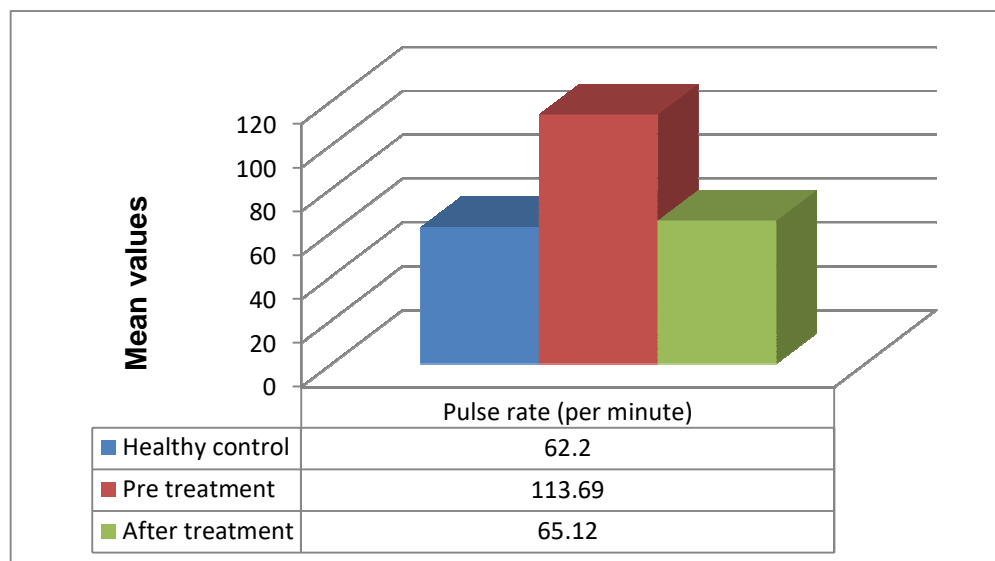
\* (P<0.05) \*\* (P<0.01)

Means with different superscripted letters in the same row differ significantly.

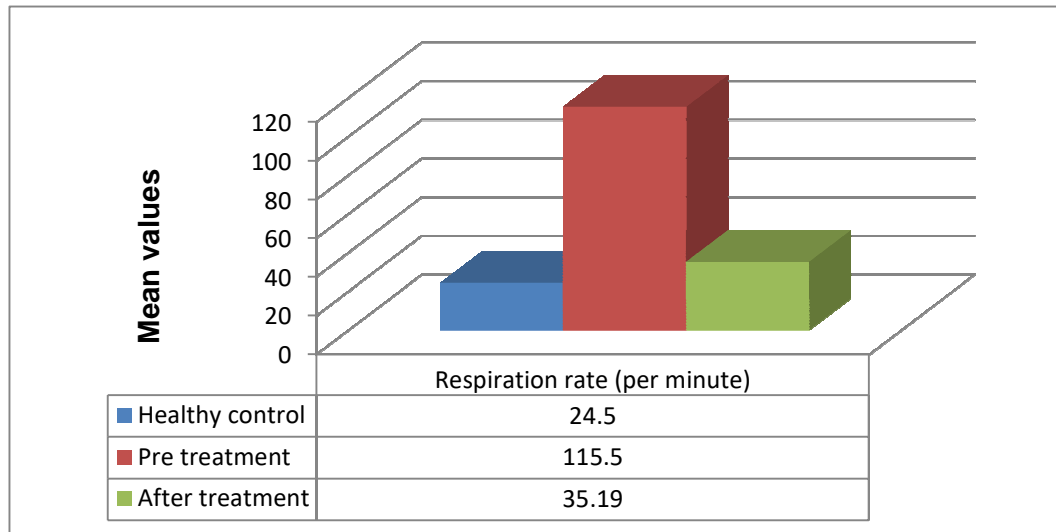
**Figure 1: Mean values of body temperature (<sup>0</sup>F) in healthy and heat stroke suffered cattle.**



**Figure 2: Mean values of pulse rate (per minute) in healthy and heat stroke suffered cattle.**



**Figure 3: Mean values of respiration rate (per minute) in healthy and heat stroke suffered cattle.**



#### 4.4 Haematological parameters:

##### 4.4.1 Haemoglobin (g/dl):

There was significant increase in Mean $\pm$ SE pre-treatment value ( $p < 0.05$ ) of haemoglobin (Table-5, Figure-4 and Appendix-6) in heat stroke suffered cattle ( $11.54 \pm 0.1466$  g/dl) as compared to healthy cattle ( $10.68 \pm 0.4168$  g/dl). Similar findings were recorded by several workers including Srikanda and Johnson (2004), Abdalla *et al.* (2009), Alam *et al.* (2011), Chandrabhan *et al.* (2013), Haque *et al.* (2013), Rana *et al.* (2014), Abd-Alkareem *et al.* (2015) and Chaudhary *et al.* (2015).

The Mean $\pm$ SE values of haemoglobin after treatment (Table-5, Figure-4 and Appendix-7) were  $11.17 \pm 0.1478$  g/dl. There was significant decrease ( $p < 0.05$ ) in haemoglobin level as compared to its pre-treatment level ( $11.54 \pm 0.1466$  g/dl).

There was no significant difference between Mean $\pm$ SE values of haemoglobin on 3<sup>rd</sup> day of treatment as compared to healthy cattle.

#### **4.4.2 Packed cell volume (per cent):**

There was non-significant increase in Mean $\pm$ SE pre-treatment value of PCV (Table-5, Figure-5 and Appendix-6) in heat stroke suffered cattle (35.31 $\pm$ 0.4899 per cent) as compared to healthy cattle (33.62 $\pm$ 1.2323 per cent). Similar findings were recorded by several workers including, Walters (2002), Abdalla *et al.* (2009), Alam *et al.* (2011), Chandrabhan *et al.* (2013), Haque *et al.* (2013), Rana *et al.* (2014), Abd-Alkareem *et al.* (2015) and Chaudhary *et al.* (2015).

The Mean $\pm$ SE values of packed cell volume after treatment (33.56 $\pm$ 0.4997 per cent) are presented in table-5, figure-5 and appendix-7. There was non-significant lower in PCV level as compared to its pre-treatment level (35.31 $\pm$ 0.4899 per cent).

The Mean $\pm$ SE value of packed cell volume on 3<sup>rd</sup> day of treatment was non- significantly lower as compared to healthy cattle.

#### **4.5.3 Total erythrocyte count (10<sup>12</sup>/lt):**

There was non-significant increase in Mean $\pm$ SE pre-treatment value of total erythrocyte count (Table-5, Figure-6 and Appendix-6) in heat stroke suffered cattle (7.07 $\pm$ 0.1962 $\times$ 10<sup>12</sup>/lt) as compared to healthy cattle (6.94 $\pm$ 0.2420 $\times$ 10<sup>12</sup>/lt). Similar findings were recorded by several workers including Abdalla *et al.* (2009), Alam *et al.* (2011), Chandrabhan *et al.* (2013), Haque *et al.* (2013), Rana *et al.* (2014), Abd-Alkareem *et al.* (2015) and Chaudhary *et al.* (2015).

The Mean $\pm$ SE values of total erythrocyte count (6.98 $\pm$ 0.1269 $\times$ 10<sup>12</sup>/lt) after treatment are presented in table-5, figure-6 and appendix-7. There was non-significant decrease in total erythrocyte count level as compared to its pre-treatment level (7.07 $\pm$ 0.1962  $\times$ 10<sup>12</sup>/lt).

The Mean±SE values of total erythrocyte count on 3<sup>rd</sup> day of treatment were non-significantly higher as compared to healthy cattle.

#### **4.5.4 MCH (pg), MCV (fl), MCHC (per cent):**

There was increase in Mean±SE pre-treatment value of mean corpuscular haemoglobin ( $p < 0.05$ ), mean corpuscular volume (non-significant) and mean corpuscular haemoglobin concentration ( $p < 0.05$ ) (Table-5, Figure- 7, and Appendix-6) in heat stroke suffered cattle ( $16.50 \pm 0.4615$  pg,  $50.52 \pm 1.5295$  fl,  $32.72 \pm 0.3469$  per cent) as compared to healthy cattle ( $15.38 \pm 0.3725$  pg,  $48.51 \pm 1.3380$  fl and  $31.74 \pm 0.2171$  per cent). Similar findings were recorded by several workers including Srikanda and Johnson (2004), Abdalla *et al.* (2009), Chandrabhan *et al.* (2013) and Abd-Alkareem *et al.* (2015).

The Mean±SE values of mean corpuscular haemoglobin ( $16.06 \pm 0.3260$  pg), mean corpuscular volume ( $48.29 \pm 1.0802$  fl) and mean corpuscular haemoglobin concentration ( $33.29 \pm 0.1858$  per cent) after treatment are presented in table-5, figure-7 and appendix-7.

There were significant ( $p < 0.05$ ) decrease in mean corpuscular haemoglobin, non-significant decrease in mean corpuscular volume and significant ( $p < 0.05$ ) increase in mean corpuscular haemoglobin concentration as compared to its pre-treatment level  $16.50 \pm 0.4615$  pg,  $50.52 \pm 1.5295$  fl,  $32.72 \pm 0.3469$  per cent, respectively.

The Mean±SE values of mean corpuscular haemoglobin were non-significantly higher, mean corpuscular volume were non-significantly lower and mean corpuscular haemoglobin concentration on 3<sup>rd</sup> day of treatment were significantly higher ( $p < 0.01$ ) as compared to healthy cattle.

In present study total erythrocyte count, packed cell volume percentage, haemoglobin concentration, mean corpuscular

haemoglobin, and mean corpuscular haemoglobin concentration were significantly higher in heat stroke suffered cattle as compared with healthy control cattle.

In the present study the increase in haemoglobin, PCV, total erythrocyte, MCH, MCV and MCHC values may be attributed to moderate to severe dehydration (Silanikove, 1987; Knochel, 1996 and Dematte *et al.*, 1998). The higher PCV values had been reported to be an adapted mechanism to provide water necessary for evaporative cooling process (Al-Haidary, 2004).

#### **4.5.6 Total leucocyte count ( $10^9$ /lt):**

There was non-significantly increase in Mean $\pm$ SE pre-treatment value of total leucocyte count (Table-5, Figure-8 and Appendix-6) in heat stroke suffered cattle ( $7.87\pm 0.2930\times 10^9$ /lt) as compared to healthy cattle ( $7.66\pm 0.0.2544\times 10^9$ /lt). Similar findings were recorded by Abdalla *et al.* (2009), Chandrabhan *et al.* (2013), Haque *et al.* (2013) and Rana *et al.* (2014).

The Mean $\pm$ SE values of total leucocyte count ( $7.25\pm 0.0.1503\times 10^9$ /lt) after treatment are presented in table-5, figure-8 and appendix-7. There were non-significant decrease in total leucocyte counts as compared to its pre-treatment level ( $7.87\pm 0.2930 \times 10^9$ /lt).

The Mean $\pm$ SE values of total leucocyte counts on 3<sup>rd</sup> day of treatment were non-significantly decreased as compared to healthy cattle.

In present study total leucocyte counts were non-significantly changed in heat stroke suffered cattle as compared with healthy control cattle which may be due to seasonal changes in thermal environment was not affect the total leucocyte counts (Abdalla *et al.*, 2009).

#### **4.5.7 Total Platelet count ( $10^9$ /lt):**

There was significant decrease in ( $p < 0.01$ ) Mean $\pm$ SE pre-treatment value of total platelet count (Table-5, Figure-9 and Appendix-6) in heat stroke suffered cattle ( $315.62 \pm 12.3424 \times 10^9$ /lt) as compared to healthy cattle ( $467.40 \pm 28.6909 \times 10^9$ /lt). Similar findings were recorded by several workers including Anonymous (1989) and Hashim (2010).

The Mean $\pm$ SE values of total platelet count ( $395.80 \pm 9.9754 \times 10^9$ /lt) after treatment are presented in table-5, figure-9 and appendix-7. There was significant increase ( $p < 0.01$ ) in total platelet count as compared to its pre-treatment level ( $315.62 \pm 12.3424 \times 10^9$ /lt).

The Mean $\pm$ SE values of total platelet count on 3<sup>rd</sup> day of treatment were significantly lower ( $p < 0.01$ ) as compared to healthy cattle.

In the present study decrease in total platelets count (thrombocytopenia) were observed which may be explained as hyperthermia enhances vascular permeability, and increases the ability of cells to adhere to the vascular walls causing alterations in blood flow. The activation of the coagulation cascade is exaggerated and platelets are consumed in large quantities, leading to thrombocytopenia (Knochel and Reed 1994; Bouchama and Knochel 2002).

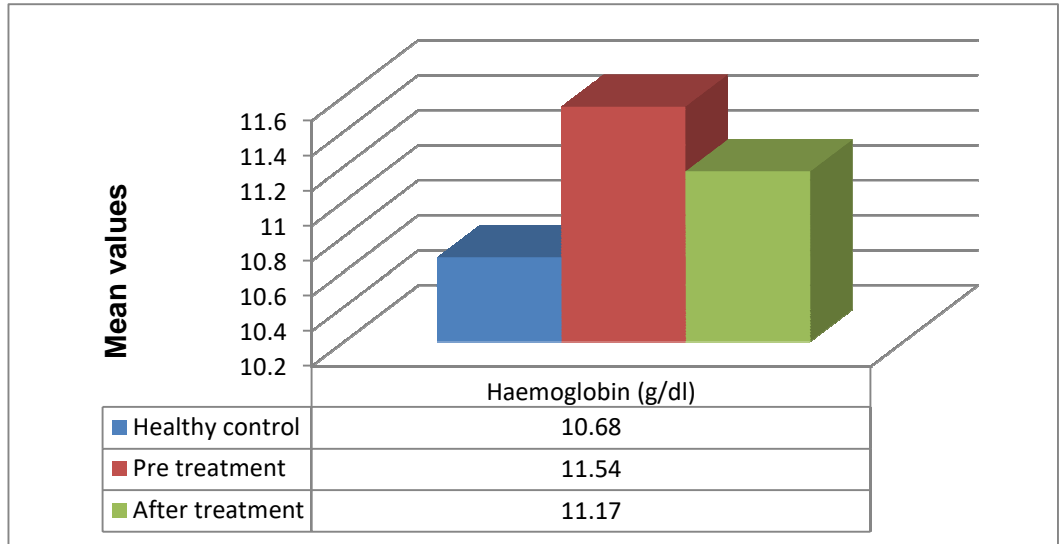
**Table 5: Mean±SE values of haematological parameters in apparently healthy and heat stroke suffered cattle.**

S.No.	Parameters	Healthy Cattle (n=10)	Before Treatment (n=16)	After treatment (n=16)
1	Hb (g/dl)*	10.68±0.4168 <sup>a</sup>	11.54±0.1466 <sup>b</sup>	11.17± 0.1478 <sup>a</sup>
2	PCV	33.62±1.2323	35.31±0.4899	33.56 ± 0.4997
3	TEC (10 <sup>12</sup> /lt)	6.94±0.2420	7.07 ±0.1962	6.98 ± 0.1269
4	TLC (10 <sup>9</sup> /lt)	7.66±0.2544	7.87 ±0.2930	7.25 ± 0.1503
5	TPC (10 <sup>9</sup> /lt)**	467.40 ± 28.6909 <sup>c</sup>	315.62±12.3424 <sup>a</sup>	395.80±9.9754 <sup>b</sup>
5	MCH (pg)*	15.38±0.3725 <sup>a</sup>	16.50±0.4615 <sup>b</sup>	16.06±0.3260 <sup>a</sup>
6	MCV (fl)	48.51±1.338	50.52±1.5295	48.29±1.0802
7	MCHC (per cent)*	31.74±0.2171 <sup>a</sup>	32.72±0.3469 <sup>b</sup>	33.29±0.1858 <sup>c</sup>

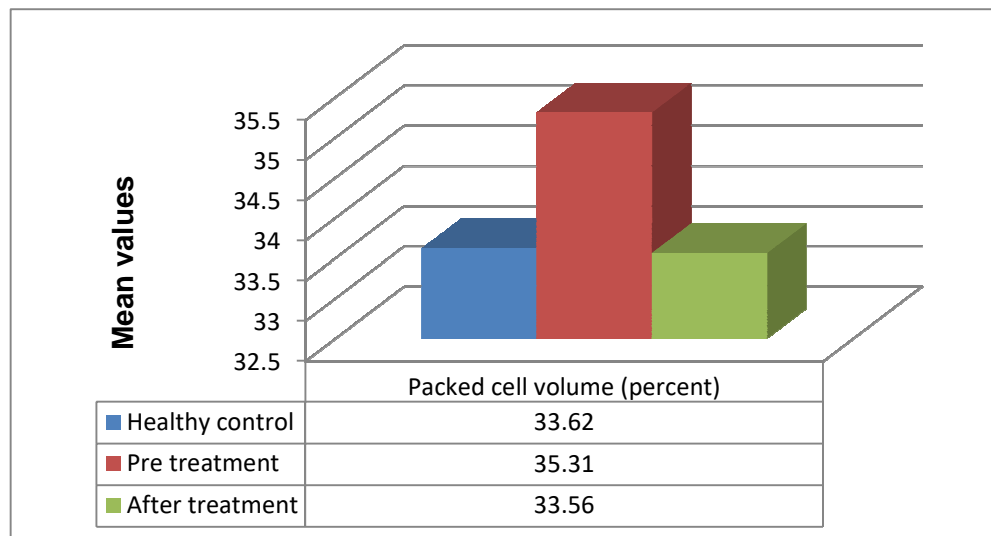
\* (P<0.05) \*\* (P<0.01)

Means with different superscripted letters in the same row differ significantly.

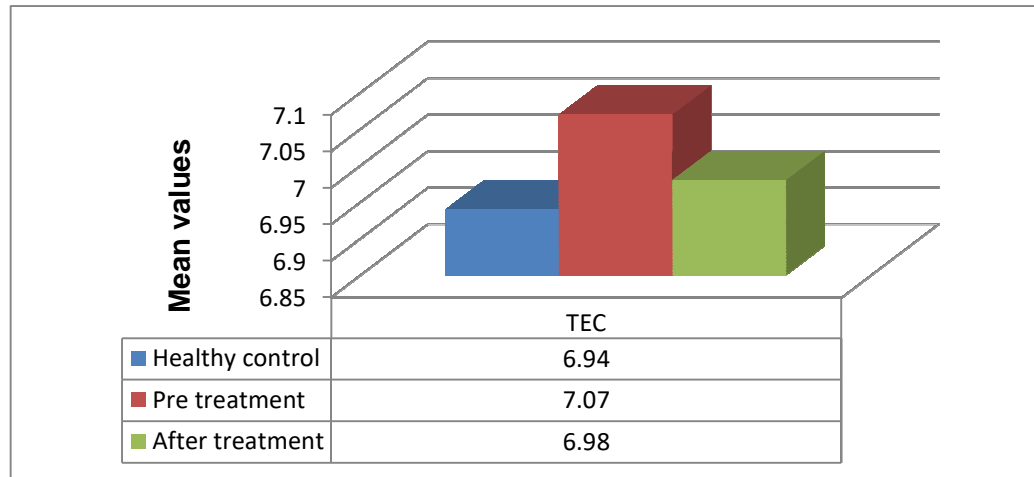
**Figure 4: Mean values of Haemoglobin (g/dl) in healthy and heat stroke suffered cattle.**



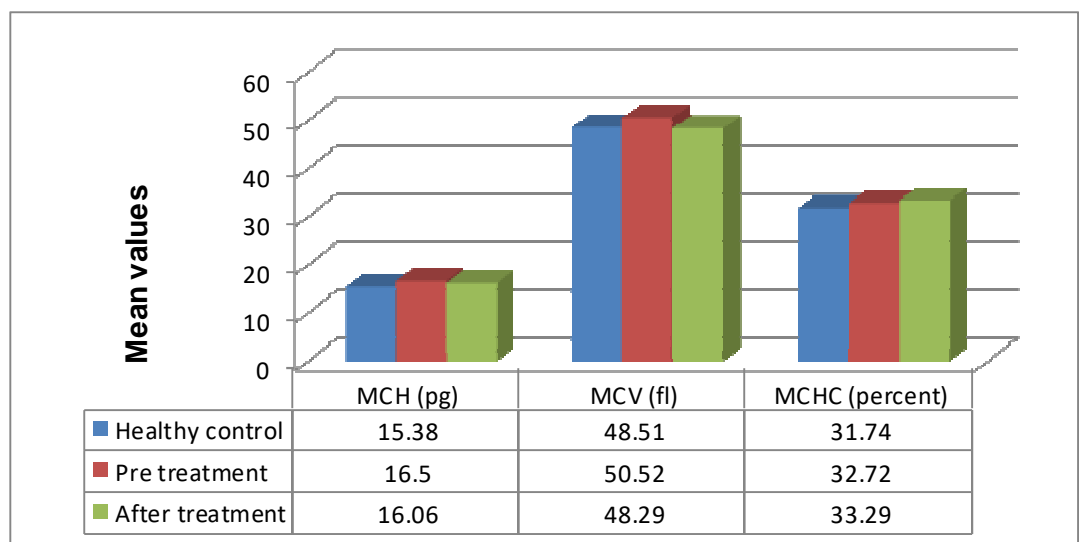
**Figure 5: Mean values of packed cell volume (per cent) in healthy and heat stroke suffered cattle.**



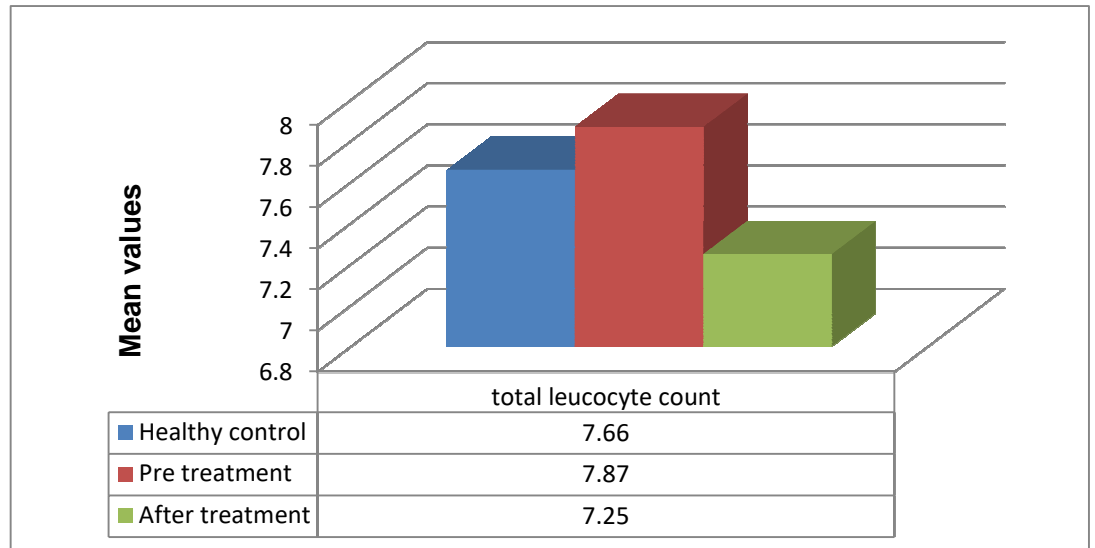
**Figure 6: Mean values of total erythrocyte count ( $10^{12}/\text{lt}$ ) in healthy and heat stroke suffered cattle.**



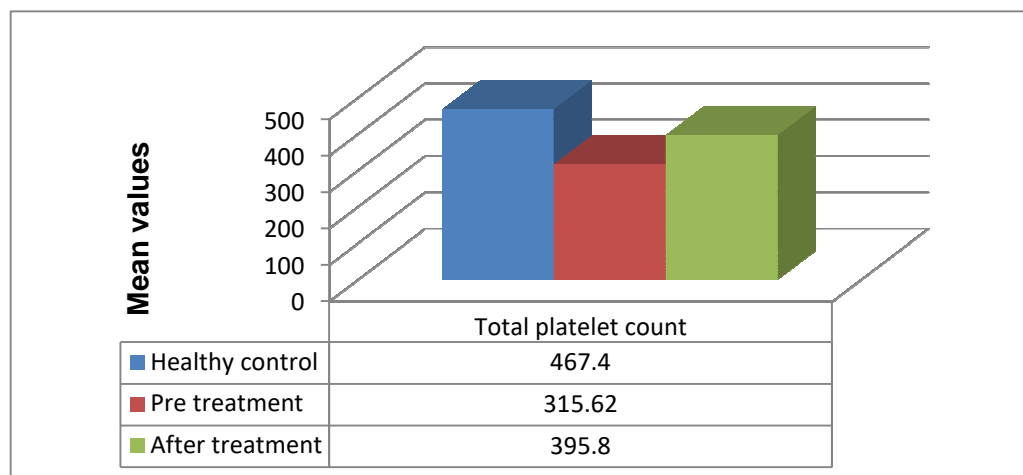
**Figure 7: Mean values of MCH (pg), MCV (fl) and MCHC (per cent) in healthy and heat stroke suffered cattle.**



**Figure 8: Mean values of total leucocyte count ( $10^6/l$ ) in healthy and heat stroke suffered cattle.**



**Figure 9: Mean values of total platelet count ( $10^6 /l$ ) in healthy and heat stroke suffered cattle.**



#### 4.5.8 Differential leucocyte count (%):

The Mean $\pm$ SE values of differential leucocyte count in heat stroke suffered cattle are presented in table-6, figure-10 and appendix-9. The Mean $\pm$ SE value of lymphocyte, monocyte, neutrophils, eosinophil and basophils in heat stroke suffered cattle were 50.31 $\pm$ 1.2491, 2.75 $\pm$ 0.2997, 44.56 $\pm$ 1.1318, 2.37 $\pm$ 0.2633 and 0 $\pm$ 0 %, respectively. Corresponding Mean $\pm$ SE values for normal healthy cattle are given in table-6 and data are presented in appendix-8.

The Mean $\pm$ SE values of lymphocyte, monocyte, neutrophil, eosinophil and basophils in normal healthy cattle were 64.60 $\pm$ 0.9092, 2.60 $\pm$ 0.3711, 29.70 $\pm$ 1.0651, 3.10 $\pm$ 0.4582 and 0 $\pm$ 0 %, respectively.

There was highly significant increase ( $p < 0.01$ ) in neutrophils are presented in table-6, figure-10 and appendix-9 in heat stroke suffered cattle as compared to healthy cattle. Similar findings were recorded by Gross and Siegel (1983), Mc Farlane and Curtis (1989) and Alam *et al.* (2011).

There was highly significant decrease ( $p < 0.01$ ) in lymphocyte (Table-6, Figure-10 and Appendix-9) in heat stroke suffered cattle as compared to healthy cattle. Similar findings were recorded by Gross and Siegel (1983), Blecha *et al.* (1984) and Mc Farlane and Curtis (1989).

The non-significant increase in monocyte, non-significant decrease in eosinophil and non-significant changes in basophil counts were observed in heat stroke suffered cattle as compared to healthy cattle. Similar findings were recorded by Abdalla *et al.* (2009) and Alam *et al.* (2011).

The Mean $\pm$ SE values of lymphocyte, monocyte, neutrophils, eosinophil and basophils after treatment are presented in table-6,

figure-10 and appendix-10. It was  $60.12 \pm 0.4319$ ,  $2.69 \pm 0.1920$ ,  $34.62 \pm 0.5144$ ,  $2.56 \pm 0.2495$ ,  $0 \pm 0$  %, respectively. There was highly significant increase ( $p < 0.01$ ) in lymphocyte and highly significant decrease in neutrophils as compared to its pre-treatment level ( $50.31 \pm 1.2491\%$ ), ( $44.56 \pm 1.1318\%$ ), respectively. There was non-significant decrease in monocyte and non-significant increase in eosinophil as compared to its pre-treatment level.

The Mean  $\pm$  SE value of lymphocyte and neutrophils had significant difference while monocyte, eosinophil and basophils on 3<sup>rd</sup> day of treatment had no significant difference as compared to healthy cattle.

In the present study significant increase in the number of neutrophil was observed and is may be explained by inflammation associated with heat stroke. Moderate heat stroke resulted in mild to moderate inflammatory responses which were self-limited and subsided after 36 hours with recovery of the animals (Bouchama and Knochel, 2002 and Bouchama *et al.*, 2004). A significant decrease in the number of lymphocyte was observed. This may be due to stress associated with high ambient temperature and high temperature humidity index. These findings are consistent to that (Gross and Siegel, 1983; Blecha *et al.*, 1984; Mc Farlane and Curtis, 1989). No significant difference was observed in other leucocyte count.

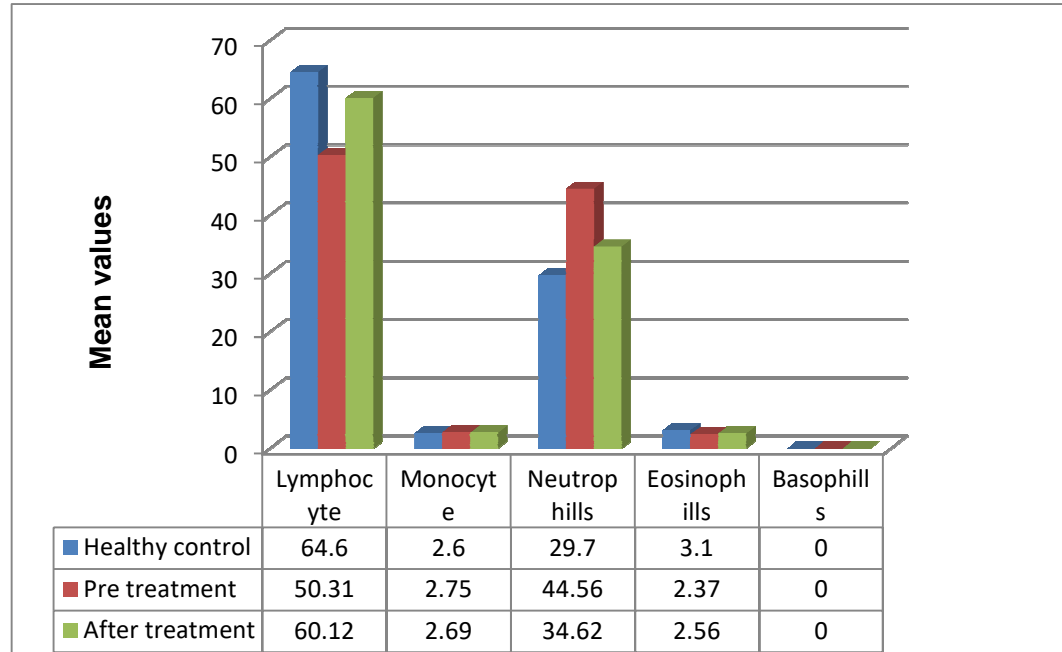
**Table 6: Mean±SE values of Differential leucocyte count in apparently healthy and heat stroke suffered cattle.**

S.No.	Parameters	Healthy Cattle (n=10)	Before Treatment (n=16)	After treatment (n=16)
1	Lymphocyte (%) **	64.60±0.9092 <sup>c</sup>	50.31±1.2491 <sup>a</sup>	60.12±0.4319 <sup>b</sup>
2	Monocyte (%)	2.60±0.3711	2.75 ± 0.2997	2.69 ± 0.1920
3	Neutrophils (%) **	29.70±1.0651 <sup>a</sup>	44.56±1.1318 <sup>c</sup>	34.62±0.5144 <sup>b</sup>
4	Eosinophils (%)	3.10±0.4582	2.37±0.2633	2.56±0.2495
5	Basophils (%)	0	0	0

\* (P<0.05) \*\* (P<0.01)

Means with different superscripted letters in the same row differ significantly.

**Figure 10: Mean values of differential leucocyte count (Per cent) in healthy and heat stroke suffered cattle.**



#### 4.6 Biochemical parameters:

##### 4.6.1 Serum alanine aminotransferase (ALT U/lt):

There was highly significant increase in Mean $\pm$ SE pre-treatment value ( $P < 0.01$ ) of serum alanine aminotransferase (Table-7, Figure-11 and Appendix-12) in heat stroke suffered cattle ( $85.12 \pm 5.9605$  U/lt) as compared to that of healthy cattle ( $25.22 \pm 2.3679$  U/lt). The observations recorded in the present study are in agreement with findings of Anonymous (1989), [Alzeer et al.](#) (1997), Varghese *et al.* (2005), Hashim (2010), Chandrabhan *et al.* (2013), Choudhary *et al.* (2015) and Jain *et al.* (2015).

The Mean $\pm$ SE values of ALT after treatment ( $38.68 \pm 1.9359$  U/lt) are presented in table-7, figure-11 and appendix-13. There was highly significant decrease ( $p < 0.01$ ) in ALT as compared to its pre-treatment level ( $25.22 \pm 2.3679$  U/lt) in heat stroke suffered cattle.

The Mean±SE value of ALT on 3<sup>rd</sup> day of treatment were significantly higher ( $p<0.01$ ) as compared to healthy cattle.

#### **4.6.2 Serum aspartate aminotransferase (AST U/l):**

There was highly significant increase in Mean±SE pre-treatment value ( $P<0.01$ ) of serum aspartate aminotransferase (Table-7, Figure-12 and Appendix-12) in heat stroke suffered cattle ( $359.75\pm 10.7726$  U/l) as compared to that in healthy cattle ( $92.49\pm 4.7832$  U/l). The observations recorded in the present study are in agreement with findings of Anonymous (1989), [Alzeer et al.](#) (1997), Srikanda and Johnson (2004), Varghese *et al.* (2005), Temizel *et al.* (2009), Hashim (2010), Chandrabhan *et al.* (2013), Choudhary *et al.* (2015) and Jain *et al.* (2015).

The Mean±SE values of serum aspartate aminotransferase after treatment ( $139\pm 5.4754$ ) are presented in table-7, figure-12 and appendix-13. There was highly significant decrease ( $p<0.01$ ) in AST as compared to its pre-treatment level ( $359.75\pm 10.7726$  U/l) in heat stroke suffered cattle.

The Mean±SE value of AST on 3<sup>rd</sup> day of treatment were significantly higher ( $p<0.01$ ) as compared to healthy cattle.

#### **4.6.2 Serum alkaline phosphatase (ALKP U/l):**

There was significant increase in Mean±SE pre-treatment value ( $P<0.05$ ) of serum alkaline phosphatase (Table-7, Figure-13 and Appendix-12) in heat stroke suffered cattle ( $99.87\pm 4.5568$  U/l) as compared to that in healthy cattle ( $76.50\pm 9.4130$  U/l). The observations recorded in the present study are in agreement with findings of Varghese *et al.* (2005), Hashim (2010) and Chandrabhan *et al.* (2013).

The Mean $\pm$ SE values of serum alkaline phosphatase after treatment (84.75 $\pm$ 17.8728 U/lit) are presented in table-7, figure-13 and appendix-13. There was significant decrease ( $p<0.05$ ) in ALKP as compared to its pre-treatment level (99.87 $\pm$ 4.5568 U/lit) in heat stroke suffered cattle.

The Mean  $\pm$  SE value of ALKP on 3<sup>rd</sup> day of treatment was non-significantly higher as compared to healthy cattle.

These increases in AST, ALT and ALKP values may be associated with liver damage due to direct thermal injury and hypoxia secondary to splenic re-distribution (Kasahara *et al.*, 1979; Van Der Linde *et al.*, 1992; Alzeer *et al.*, 1997 and Giercksky *et al.* 1999).

#### **4.6.3 Serum Lactate dehydrogenase (LDH U/lit):**

There was highly significant increase in Mean $\pm$ SE pre-treatment value ( $P<0.01$ ) of serum LDH (Table-7, Figure-14 and Appendix-12) in heat stroke suffered cattle (1355.25 $\pm$ 27.2066 U/lit) as compared to that in healthy cattle (1019.2 $\pm$ 77.0168 U/lit). The observations recorded in the present study are in agreement with findings of [Alzeer \*et al.\* \(1997\)](#), Temizel *et al.* (2009), Chandrabhan *et al.* (2013) and Jain *et al.* (2015).

The Mean $\pm$ SE values of serum LDH after treatment (1256.62 $\pm$ 32.2229 U/lit) are presented in table-7, figure-14 and appendix-13. There was highly significant decrease ( $p<0.01$ ) in LDH as compared to its pre-treatment level (1355.25 $\pm$ 27.2066 U/lit) in heat stroke suffered cattle.

The Mean $\pm$ SE value of LDH on 3<sup>rd</sup> day of treatment were significantly higher ( $p<0.01$ ) as compared to healthy cattle.

#### **4.6.4 Serum Creatinine kinase (CK U/lit):**

There was highly significant increase in Mean $\pm$ SE pre-treatment value ( $P<0.01$ ) of serum CK (Table-7, Figure-15 and Appendix-12) in heat stroke suffered cattle ( $297.37\pm 2.4951$  U/lit) as compared to that in healthy cattle ( $123.51\pm 26.5901$  U/lit). The observations recorded in the present study are in agreement with findings of Tucker *et al.* (1985), Anonymous (1989), [Alzeer et al.](#) (1997), Varghese *et al.* (2005) and Jain *et al.* (2015).

The Mean $\pm$ SE values of serum CK after treatment ( $286.57\pm 5.6218$  U/lit) are presented in table-7, figure-15 and appendix-13. There was significant decrease ( $p<0.01$ ) in CK as compared to its pre-treatment level ( $297.37\pm 2.4951$  U/lit) in heat stroke suffered cattle.

The Mean $\pm$ SE value of CK on 3<sup>rd</sup> day of treatment were significantly higher ( $p<0.01$ ) as compared to healthy cattle.

In the present study LDH and CK level were increased significantly in heat stroke suffered cattle which may be associated with liver damage due to direct thermal injury and hypoxia secondary to splenic re-distribution and may be associated with muscle damage (Kasahara *et al.*, 1979; Van Der Linde *et al.*, 1992; Alzeer *et al.*, 1997; Giercksky *et al.*, 1999).

#### **4.6.5 Serum Blood urea nitrogen (BUN mg/dl):**

There was highly significant increase in Mean $\pm$ SE pre-treatment value ( $P<0.01$ ) of serum BUN (Table-7, Figure-16 and Appendix-12) in heat stroke suffered cattle ( $39.5\pm 2.2998$  mg/dl) as compared to that in healthy cattle ( $14.30 \pm 0.9515$  mg/dl). The observations recorded in the present study are in agreement with findings of Kelly (1974), Walters (2002), Roger (2005), Temizel *et al.*

(2009), Bras (2010), Hashim (2010), Sreedhar *et al.* (2013) and Choudhary *et al.* (2015).

The Mean $\pm$ SE values of serum BUN after treatment ( $29.5 \pm 1.8307$  mg/dl) are presented in table-7, figure-16 and appendix-13. There was significant decrease ( $p < 0.01$ ) in BUN as compared to its pre-treatment level ( $39.5 \pm 2.2998$  mg/dl) in heat stroke suffered cattle.

The Mean $\pm$ SE value of BUN on 3<sup>rd</sup> day of treatment were significantly higher ( $p < 0.01$ ) as compared to healthy cattle.

#### **4.6.6 Serum Creatinine (Cr. mg/dl):**

There was highly significant increase in Mean $\pm$ SE pre-treatment value ( $P < 0.01$ ) of serum creatinine (Table-7, Figure-17 and Appendix-12) in heat stroke suffered cattle ( $3.08 \pm 0.3199$  mg/dl) as compared to that in healthy cattle ( $0.77 \pm 0.0557$  mg/dl). The observations recorded in the present study are in agreement with findings of Walters (2002), [Srikanda](#) and [Johnson \(2004\)](#), Temizel *et al.* (2009), Hashim (2010), Sreedhar *et al.* (2013) and Choudhary *et al.* (2015). The Mean $\pm$ SE values of serum creatinine after treatment ( $1.34 \pm 0.0957$ ) are presented in table-7, figure-17 and appendix-13. There was highly significant decrease ( $p < 0.01$ ) in creatinine as compared to its pre-treatment level ( $3.08 \pm 0.3199$  mg/dl) in heat stroke suffered cattle.

The Mean $\pm$ SE values of creatinine after treatment were significantly higher ( $p < 0.01$ ) as compared to healthy cattle.

The increase in serum BUN and creatinine level may be associated with severe heat stroke displayed an oliguric renal failure with an increase in serum blood urea nitrogen and creatinine levels (Bouchama *et al.*, 2004). These increases may be associated with renal dysfunction or with pre-renal azotemia that might develop due to

shock. Also, the high haematocrit would support at least a degree of dehydration as a contributing factor to azotemia (Dematte *et al.*, 1998).

#### **4.6.7 Serum Lactate (mmol/l):**

There was highly significant increase in Mean $\pm$ SE pre-treatment value ( $P<0.01$ ) of serum lactate (Table-7, Figure-18 and Appendix-12) in heat stroke suffered cattle ( $7.29\pm 0.6150$  mmol/l) as compared to that in healthy cattle ( $1.44\pm 0.1598$  mmol/l). The observations recorded in the present study are in agreement with findings of Tucker *et al.* (1985), Shapiro and Cristol (1987), Stoneham and Price (1992), Roger (2005), Varghese *et al.* (2005) and Temizel *et al.* (2009). The Mean $\pm$ SE values of serum lactate after treatment ( $2.89\pm 0.1434$  mmol/l) are presented in table-7, figure-18 and appendix-13. There was highly significant decrease ( $p<0.01$ ) in lactate as compared to its pre-treatment level ( $7.29\pm 0.6150$  mmol/l) in heat stroke suffered cattle.

The Mean $\pm$ SE values of lactate on 3<sup>rd</sup> day of treatment were significantly higher ( $p<0.01$ ) as compared to healthy cattle.

In the present study serum lactate level was increased significantly in heat stroke suffered cattle which may be attributed to formation of L-lactate from anaerobic glycolysis following tissue hypo perfusion (Knochel, 1996). High plasma L-lactate level was the most important biochemical change has long been considered to be a cause of high anion gap acidosis in cattle with heat stroke. A negative correlation between increased plasma lactate and decreased survival rate was reported by Katamoto *et al.* (1998) and Grogan and Hopkins (2002).

#### **4.6.8 Serum glucose (mg/dl):**

There was significant decrease in Mean $\pm$ SE pre-treatment value ( $P<0.01$ ) of serum glucose (Table-7, Figure-19 and Appendix-12)

in heat stroke suffered cattle ( $51.50 \pm 1.6677$  mg/dl) as compared to that in healthy cattle ( $63.80 \pm 2.6038$  mg/dl). The observations recorded in the present study are in agreement with findings of Kelly (1974), Walters (2002), Srikanda and Johnson (2004), Roger (2005), Abeni *et al.* (2007) and Choudhary *et al.* (2015).

The Mean $\pm$ SE values of serum glucose after treatment ( $62.12 \pm 1.0893$  mg/dl) are presented in table-7, figure-19 and appendix-13. There was significant increase ( $p < 0.01$ ) in glucose as compared to its pre-treatment level ( $51.50 \pm 1.6677$  mg/dl) in heat stroke suffered cattle.

The Mean $\pm$ SE value of glucose on 3<sup>rd</sup> day of treatment were no significantly changes as compared to healthy cattle.

In the present study decrease in glucose was observed which may be explained as in heat stroke, due to the increased metabolic activity which consumes vast amounts of glucose while glycogen store rapidly depleted in injured liver due to thermal injury and hypo-perfusion to liver in heat stroke (Drobatz, 2004).

#### **4.6.9 Serum sodium and potassium (mEq/l):**

There was significant decrease in Mean $\pm$ SE pre-treatment value ( $P < 0.01$ ) of serum sodium and potassium (Table-7, Figure-20, 21 and Appendix-15) in heat stroke suffered cattle ( $130.59 \pm 0.2682$  and  $4.28 \pm 0.0813$  mEq/l), respectively as compared to that in healthy cattle ( $138.76 \pm 1.0646$  and  $5.07 \pm 0.1566$  mEq/l), respectively. The observations recorded in the present study are in agreement with findings of Johnson (1970), El-Nouty *et al.* (1980), Mallonee *et al.* (1985), Tucker *et al.* (1985), West *et al.* (1991), Sanchez *et al.* (1994a), Walters (2002), [Srikanda](#) and [Johnson \(2004\)](#), Roger (2005), Bras

(2010), Mohamed and Abdel-Hamid *et al.* (2010), Singh *et al.* (2012) and Abdel-Fattah (2014).

The Mean $\pm$ SE values of serum sodium and potassium after treatment were 137.62 $\pm$ 2.0951, 4.51 $\pm$ 0.1397, respectively (Table-7, Figure-20, 21 and Appendix-16). There was significant increase ( $p < 0.01$ ) in sodium and potassium as compared to its pre-treatment level (130.59 $\pm$ 0.2682, 4.28 $\pm$ 0.0813 mEq/l), respectively in heat stroke suffered cattle.

The Mean $\pm$ SE value of sodium and potassium on 3<sup>rd</sup> day of treatment were no significantly changes as compared to healthy cattle.

In the present study hypokalemia and hyponatremia were observed which may be explained as hypokalemia is commonly seen in the early period. This can either be due to direct effects of catecholamine or it can occur secondary to heat induced hyperventilation, leading to respiratory alkalosis (Grogan and Hopkins, 2002). Respiratory alkalosis results in the renal excretion of H<sup>+</sup> being depressed and the urine excretion of HCO<sub>3</sub><sup>-</sup> must be accompanied by a cation and found sodium and potassium are possibilities with Na<sup>+</sup> being more likely (Sanchez *et al.*, 1994a). Reductions in serum K<sup>+</sup> may be due to loss of potassium in sweat during prolonged heat stress in cattle (El-Nouty *et al.*, 1980). Oliguria and renal failure caused hypokalemia (Roger, 2005). Heat stroke occurs in two types, water and sodium depleted, although in reality they often overlap (Grogan and Hopkins, 2002; Lee-Chiong, 1995 and Mellion and Shelton, 1998). In humans heat exhaustion from sodium depletion occurs most often in un-acclimated people who fail to replace sodium lost in sweat (Backer *et al.*, 1999).

#### 4.6.10 Serum Calcium and Phosphorus (mg/dl):

There was significant decrease in Mean $\pm$ SE pre-treatment value ( $P < 0.01$ ) of serum calcium and serum phosphorus (Table-7, Figure-22, 23 and Appendix-15) in heat stroke suffered cattle ( $8.29 \pm 0.1403$ ,  $4.96 \pm 0.1140$  mg/dl), respectively as compared to that in healthy cattle ( $9.63 \pm 0.2631$ ,  $6.35 \pm 0.3931$  mg/dl), respectively. The observations recorded in the present study are in agreement with findings of Tucker *et al.* (1985), Srikanda and Johnson (2004), Roger (2005), Bras (2010) and Mohamed and Abdel-Hamid (2010).

The Mean $\pm$ SE values of serum calcium and phosphorus after treatment ( $9.59 \pm 0.1536$  and  $5.79 \pm 0.0999$  mg/dl) are presented in table-7, figure-22, 23 and appendix-16. There was significant increase ( $p < 0.01$ ) in calcium and phosphorus as compared to its pre-treatment level ( $8.29 \pm 0.1403$ ,  $4.96 \pm 0.1140$  mg/dl), respectively in heat stroke suffered cattle.

The Mean $\pm$ SE value of calcium and phosphorus on 3<sup>rd</sup> day of treatment were not significantly changed as compared to healthy cattle.

In the present study decrease in calcium and phosphorus was observed which may be explained as acute hypophosphatemia and hypocalcaemia are observed in classical heat stroke (Bouchama *et al.*, 1991; Tucker *et al.*, 1985) and are probably related to the increased glucose phosphorylation seen in alkalotic conditions. Also, in some cases of heat stroke rhabdomyolysis may develop and muscle cells may be injured. The injured cells leak phosphorus, which reacts with extracellular calcium. This process may lead to hypocalcaemia and hyperphosphataemia (Grogan and Hopkins, 2002).

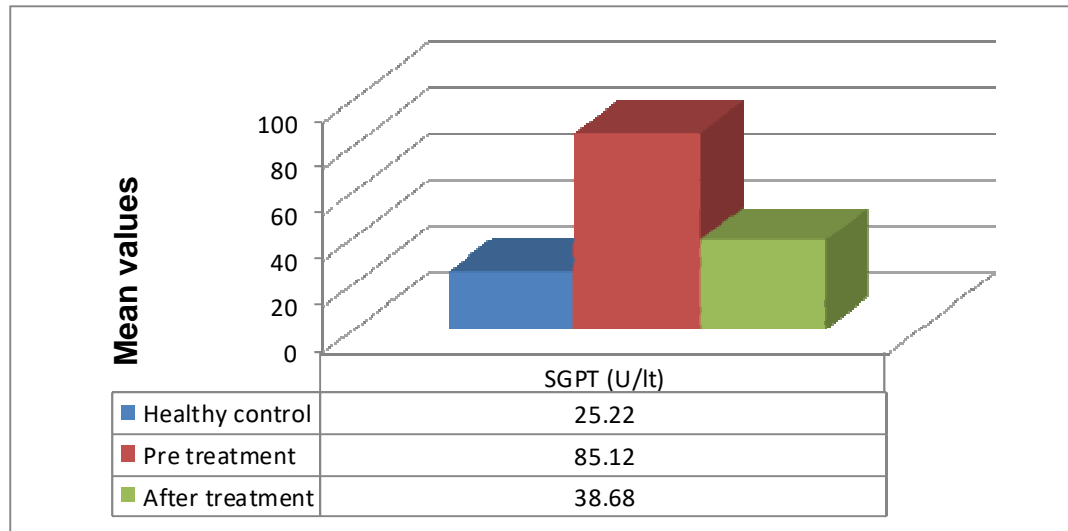
**Table 7: Mean ± SE values of Biochemical parameters in apparently healthy and heat stroke suffered cattle.**

S.No.	Parameters	Healthy Cattle (n=10)	Before Treatment (n=16)	After treatment (n=16)
1	Sodium (mmol/lit)**	138.76±1.0646 <sup>b</sup>	130.59 ± 0.2682 <sup>a</sup>	137.62 ± 2.0951 <sup>b</sup>
2	Potassium(mmol/lit) **	5.07± 0.1566 <sup>b</sup>	4.28 ± 0.0813 <sup>a</sup>	4.51 ± 0.1397 <sup>b</sup>
3	Calcium (mg/dl) **	9.63 ± 0.2631 <sup>b</sup>	8.29 ± 0.1403 <sup>a</sup>	9.59 ± 0.1536 <sup>b</sup>
4	Phosphorus ((mg/dl) **	6.35 ± 0.3931 <sup>b</sup>	4.96 ± 0.1140 <sup>a</sup>	5.79 ± 0.0999 <sup>b</sup>
5	BUN (mg/dl) **	14.30 ± 0.9515 <sup>a</sup>	39.5±2.2998 <sup>c</sup>	29.5 ± 1.8307 <sup>b</sup>
6	Creatinine(mg/dl) **	0.77 ± 0.0557 <sup>a</sup>	3.08 ± 0.3199 <sup>c</sup>	1.34 ± 0.0957 <sup>b</sup>
7	CK (U/lit) **	123.51±26.5901 <sup>a</sup>	297.37 ± 2.4951 <sup>c</sup>	286.75 ±5.6218 <sup>b</sup>
8	Lactate (mmol/lit) **	1.44 ± 0.1598 <sup>a</sup>	7.29 ± 0.6150 <sup>c</sup>	2.89 ± 0.1434 <sup>b</sup>
9	LDH (U/lit) **	1019.2±77.0168 <sup>a</sup>	1644.62±24.3325 <sup>c</sup>	1419.12 ±34.01 <sup>b</sup>
10	SGOT (U/lit) **	92.49 ±4.7832 <sup>a</sup>	359.75±10.7726 <sup>c</sup>	139 ± 5.4754 <sup>b</sup>
11	SGPT(U/lit) **	25.22 ± 2.3679 <sup>a</sup>	85.12 ± 5.9605 <sup>c</sup>	38.68 ± 1.9359 <sup>b</sup>
12	ALKP(U/lit) *	76.50 ± 9.4130 <sup>b</sup>	99.87 ± 4.5568 <sup>c</sup>	84.75 ± 17.8728 <sup>b</sup>
13	Glucose (mg/dl)**	63.80±2.6038 <sup>b</sup>	51.50±1.6677 <sup>a</sup>	62.12±1.0893 <sup>b</sup>

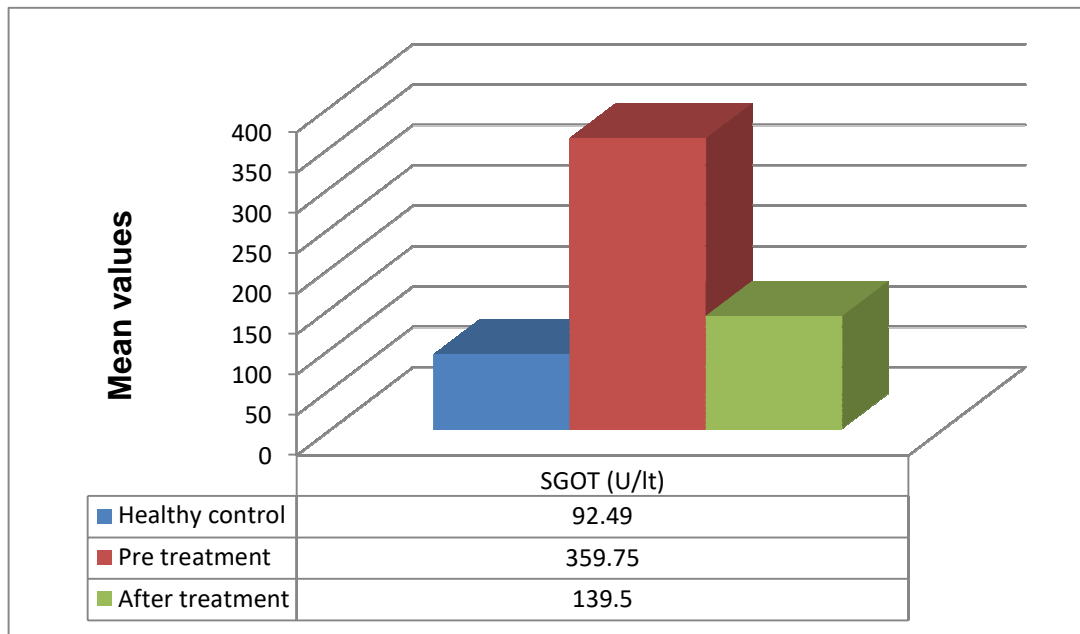
\* (P<0.05) \*\* (P<0.01)

Means with different superscripted letters in the same row differ significantly.

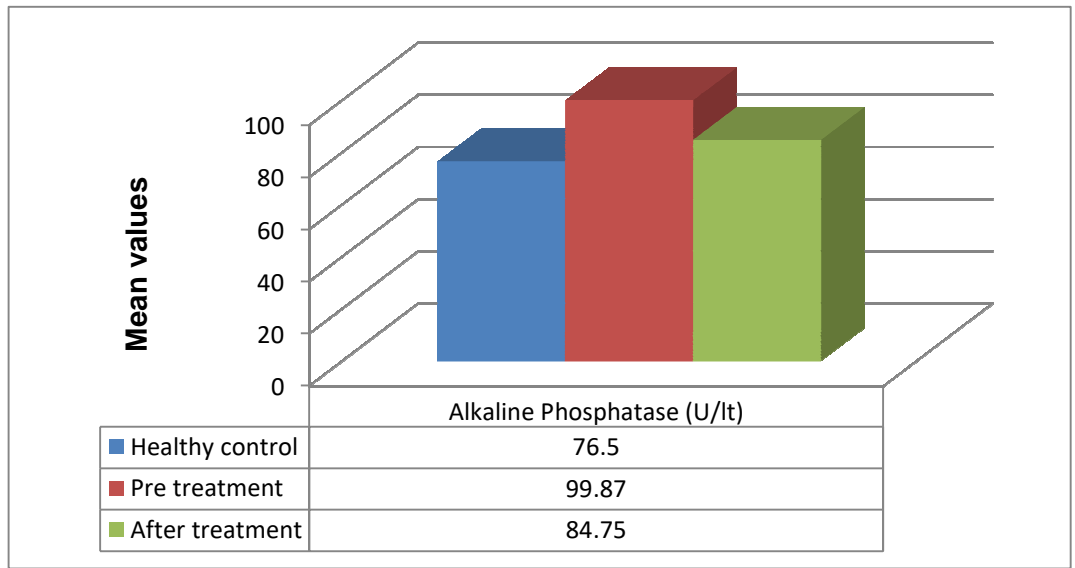
**Figure 11: Mean values of SGPT (U/l) in healthy and heat stroke suffered cattle.**



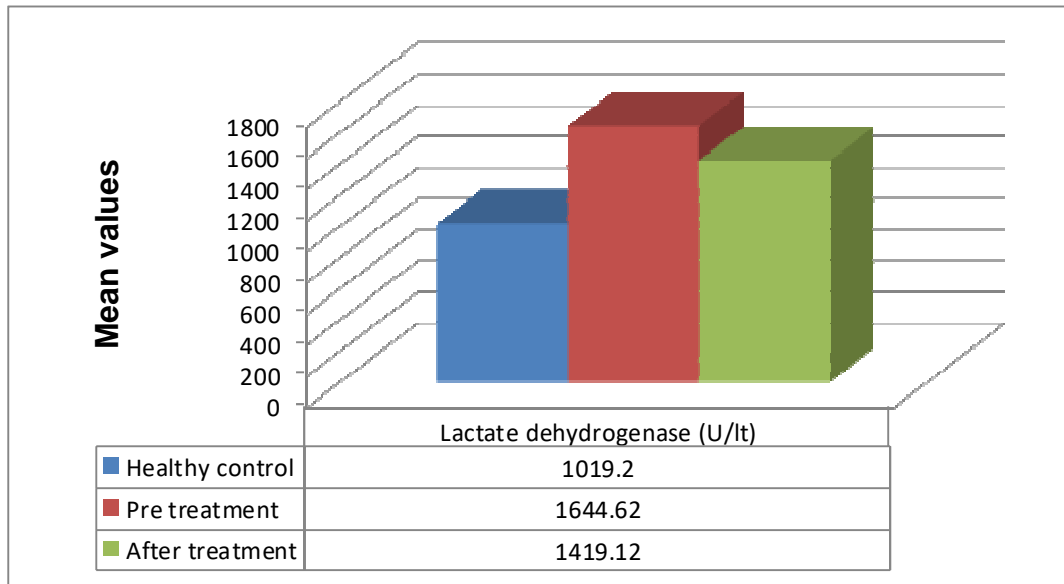
**Figure 12: Mean values of SGOT (U/l) in healthy and heat stroke suffered cattle.**



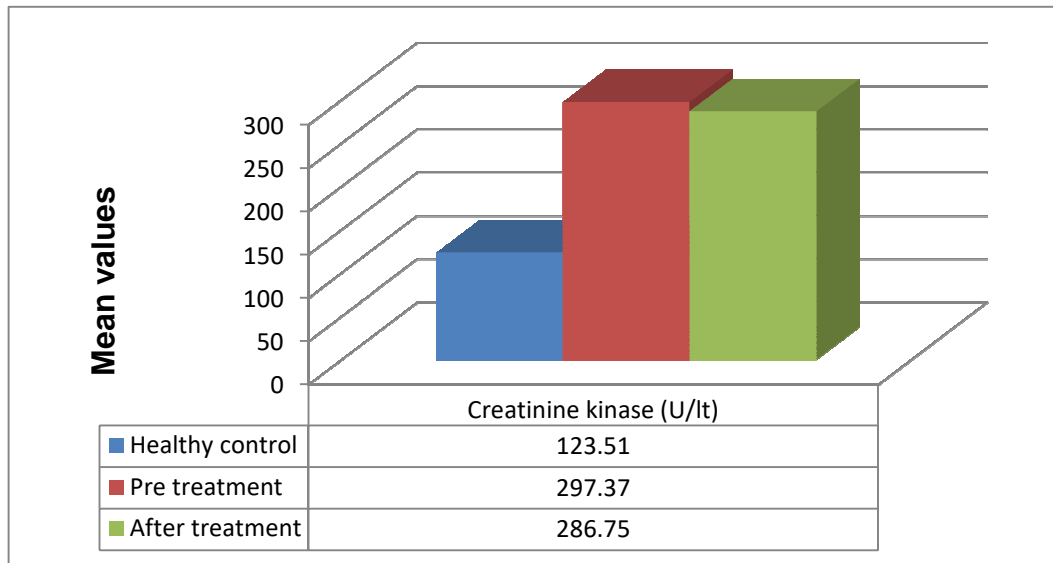
**Figure 13: Mean values of alkaline phosphatase (U/l) in healthy and heat stroke suffered cattle.**



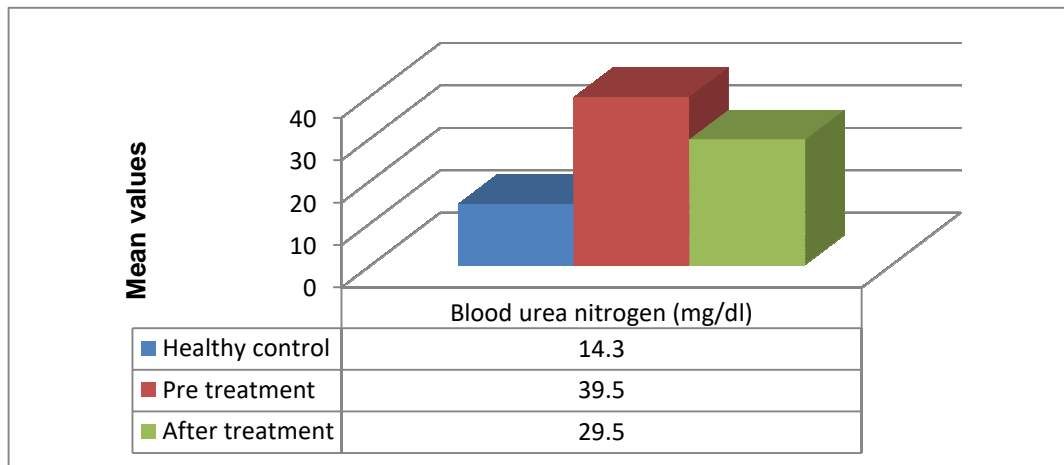
**Figure 14: Mean values of lactate dehydrogenase (U/l) in healthy and heat stroke suffered cattle.**



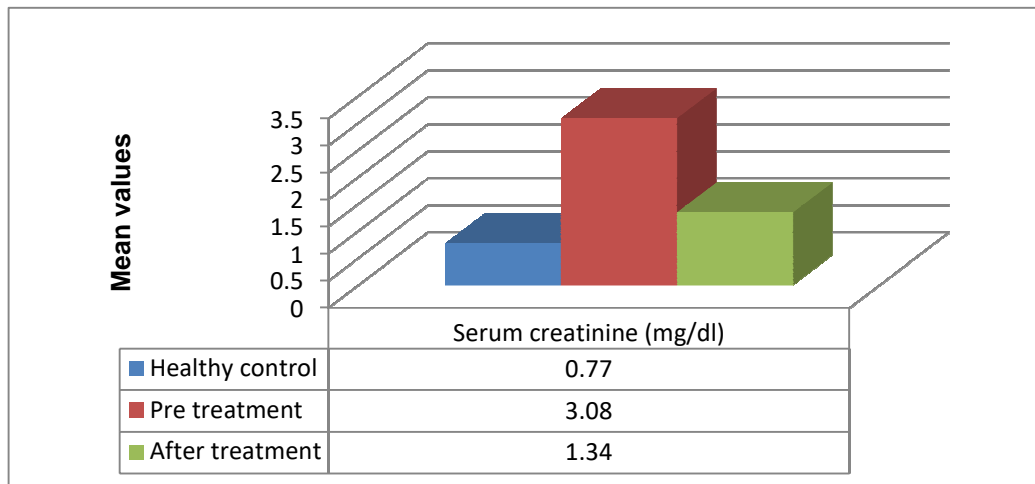
**Figure 15: Mean values of creatinine kinase (U/l) in healthy and heat stroke suffered cattle.**



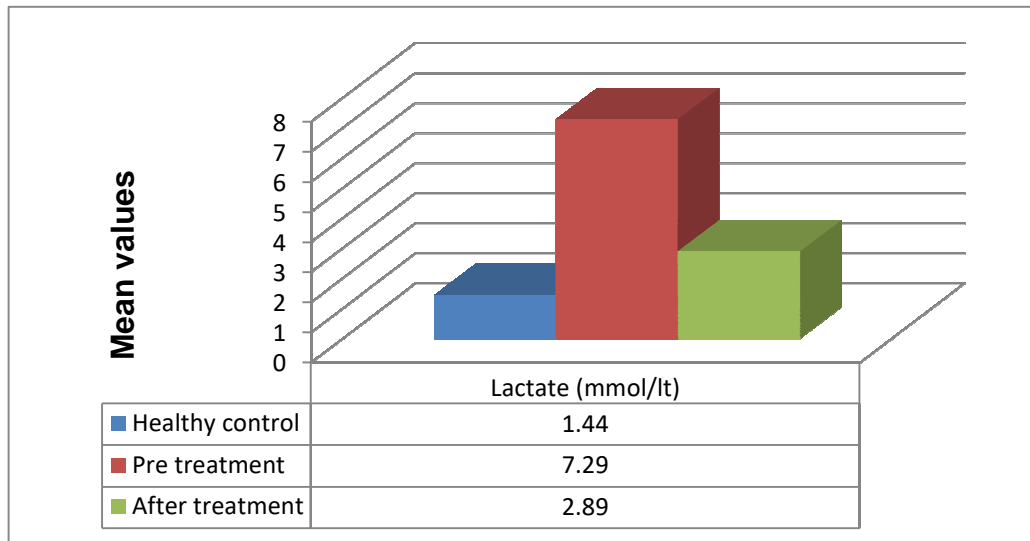
**Figure 16: Mean values of blood urea nitrogen (mg/dl) in healthy and heat stroke suffered cattle.**



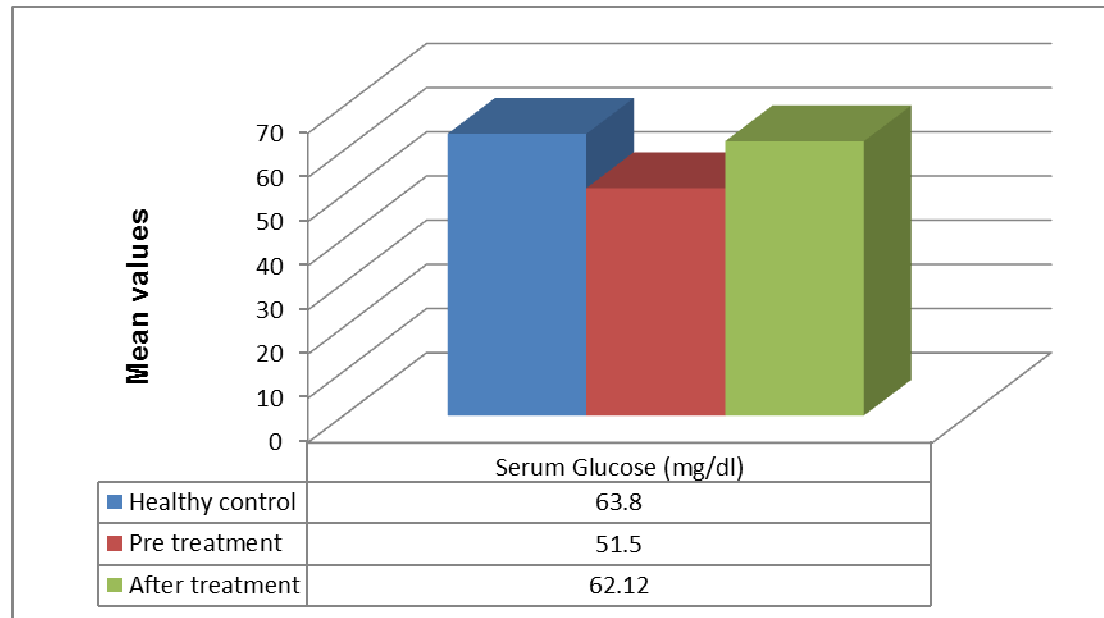
**Figure 17: Mean values of serum creatinine (mg/dl) in healthy and heat stroke suffered cattle.**



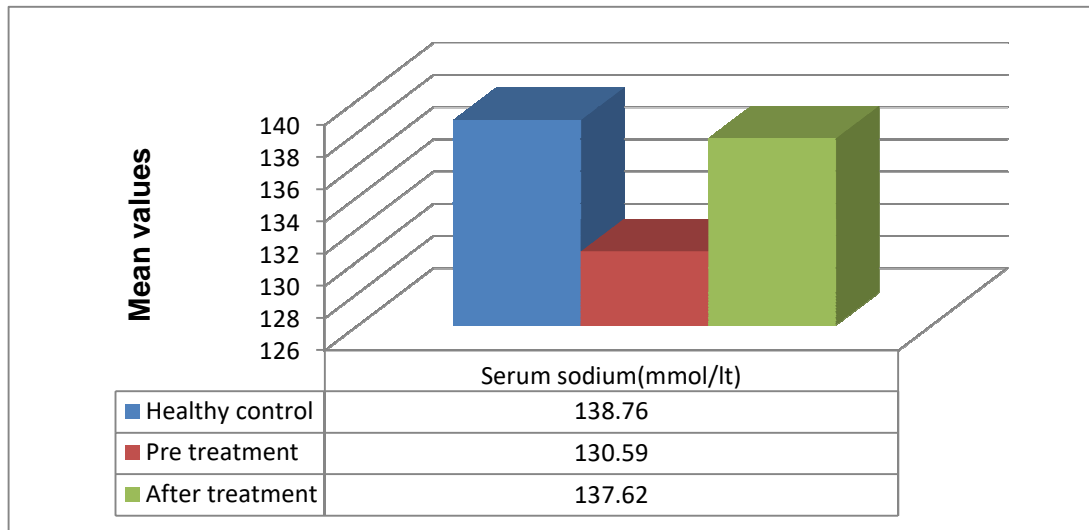
**Figure 18: Mean values of serum lactate (mmol/l) in healthy and heat stroke suffered cattle.**



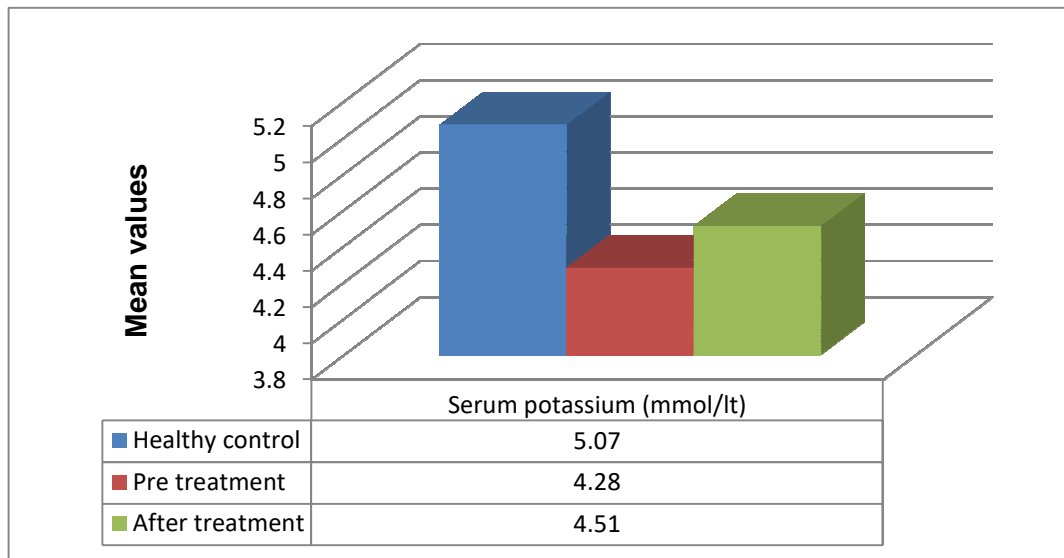
**Figure 19: Mean values of serum glucose (mg/dl) in healthy and heat stroke suffered cattle.**



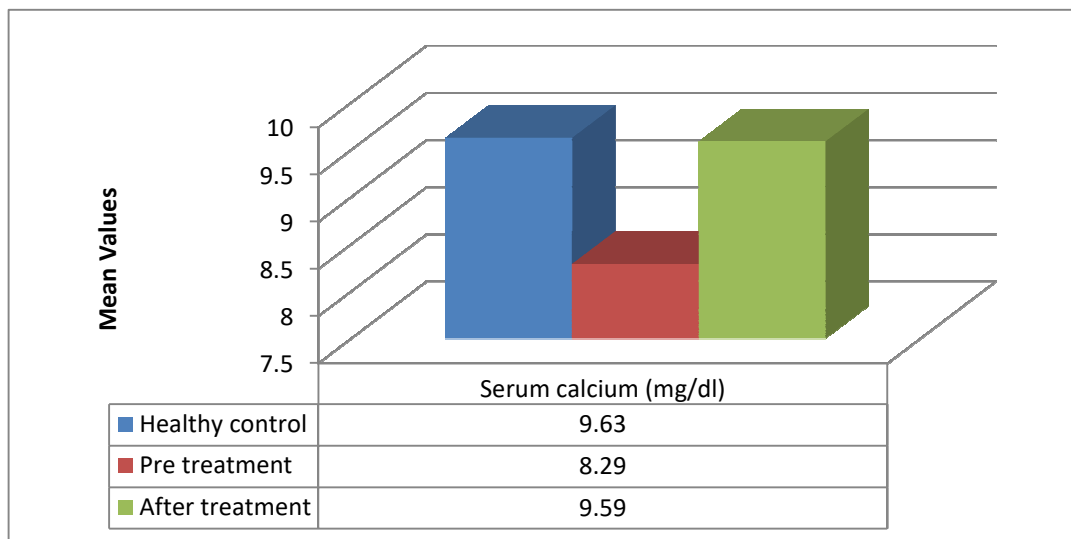
**Figure 20: Mean values of serum sodium (mmol/l) in healthy and heat stroke suffered cattle.**



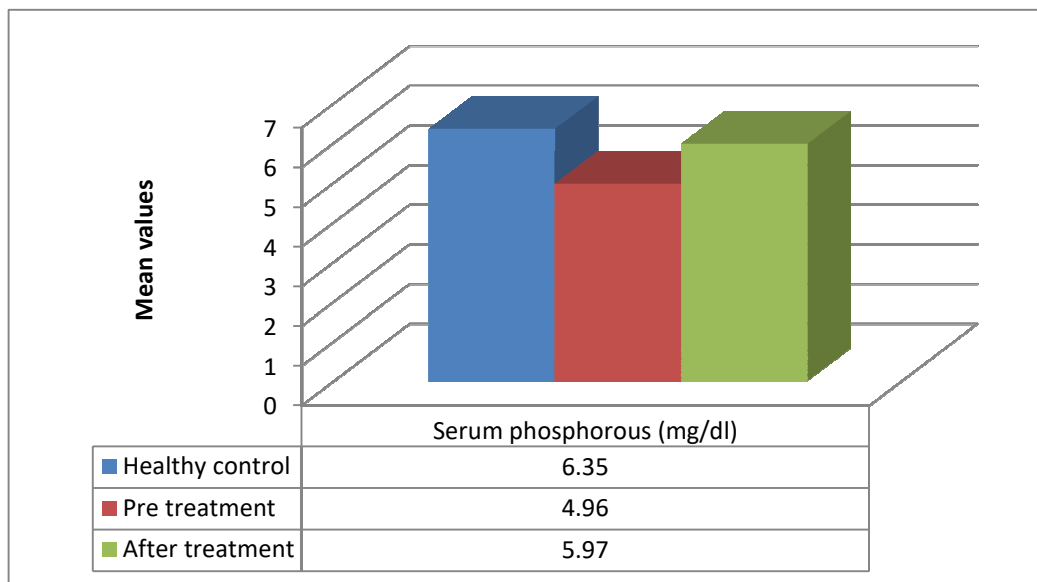
**Figure 21: Mean values of serum potassium (mmol/L) in healthy and heat stroke suffered cattle.**



**Figure 22: Mean values of serum calcium (mg/dl) in healthy and heat stroke suffered cattle.**



**Figure 23: Mean values of serum phosphorous (mg/dl) in healthy and heat stroke suffered cattle.**



#### 4.7 Treatment of heat stroke affected cattle:

In the present investigation entitled “Clinical and therapeutic studies of heat stroke in cattle” a total of sixteen cattle found positive for heat stroke were subjected to treatment.

All the cases received isotonic saline solution (0.9% NaCl) and Ringers lactate @ 20–50 ml/kg b.wt, IV based on degree of dehydration. Afterwards they were sprayed with a water hose on the midline of the back with tap water (15-16°C). Fans were immediately placed in front of the animal to promote evaporative cooling; this was done continuously until their body temperature reached 101.5°F. Dexamethasone @ 0.1mg/kg b.wt. IV was given immediately and Ascorbic acid @ 25 mg/kg b.wt. IV was given for 3 days to reduce the stress. Oxytetracycline @ of 10 mg/kg b.wt. IV was given for 3 days. When temperature comes down to normal Vit B complex- Tribivet (Intas Pvt. Ltd.) containing Vit B<sub>1</sub> 50 mg, B<sub>6</sub> 50 mg and B<sub>12</sub> 500 mcg per ml, 10ml i/m was given for 3 days. Antioxidant supplementation was done with Liq. Intavita-NH (Intas Pvt. Ltd.) containing Vit A 1200 IU, Vit D<sub>3</sub> 6000 IU, Vit E 48 mg, Biotin 50 mcg and Nicotinamide 3 mg per ml, 10ml orally daily for 10 days. Bolus Triboost (Biotech Vety. Pharma. Pvt. Ltd.) containing Live yeast culture 3 gram, Live lactobacillus sporogenes culture 20 million CFU, Amino acid 2 gram, Liver extract 5 mg and Fungal diastase 50 mg per bolus, 2 bid for 3 days were given to boost up rumen microorganisms.

Out of sixteen heat stroke suffered cases, thirteen responded to the treatment. These animals on 3<sup>rd</sup> day of treatment regained their appetite, the temperature and respiration rate comes within the normal range while three animals got paralyzed with hind limbs. The paralyzed cattle were further managed by putting the cattle on slings for one hour at stretched for 4-5 instances and nervine tonic, Neuroxin-M (Zydus AHL) containing Methyl Cobalamin 500 mcg, Pyridoxine 50 mg and Nicotinamide 50 mg per ml was given @ 15 ml i/m once in day for 5 days. The paralyzed animals showed some improvement in term of sensation and movement of hind limbs. These animals were discharged on 7<sup>th</sup> day on the request of owner and were advised to putting the cattle

on slings and carryout physiotherapy *viz.*, upward massage of hind limbs for improvement the blood circulation and extending and flexing the legs for 15-20 times to maintain the tonicity of muscle till complete recovery.

Conclusively, the evaporative cooling therapy, as well as simultaneous fluid therapy (Normal saline solution and Ringer's lactate) along with dexamethasone, broad spectrum antibiotics- Oxytetracycline, Ascorbic acid, antioxidant liquid Intavita-NH (Intas Pvt. Ltd.) containing Vit A 1200 IU, Vit D<sub>3</sub> 6000 IU, Vit E 48 mg, Biotin 50 mcg and Nicotinamide 3 mg per ml, Vit B complex- Tribivet (Intas Pvt. Ltd.) containing Vit B<sub>1</sub> 50 mg, B<sub>6</sub> 50 mg and B<sub>12</sub> 500 mcg per ml and probiotic bolus Triboost (Biotech Vety. Pharma. Pvt. Ltd.) containing Live yeast culture 3 gram, Live lactobacillus sporogenes culture 20 million CFU, Amino acid 2 gram, Liver extract 5 mg and Fungal diastase 50 mg per bolus, were quite effective in treating the clinical cases of heat stroke if the heat stroke suffered animals could be detected and treated as early as possible without any complication.

## 5. SUMMARY

---

The present study was carried out from August 2015 to September 2015. Total 800 cattle were brought for treatment to Medicine Clinic of Teaching Veterinary Clinical Complex, CVAS, Bikaner (9 cases) and visit to local dairy farms in and around Bikaner city (7 cases). Among them 16 cattle were found to be affected with heat stroke. These cattle were adult dairy cattle. The overall prevalence of heat stroke amongst cattle was found to be 2.00 per cent.

The major clinical manifestations were hyperthermia, ataxia, muscle tremor, panting, tachycardia, hyper salivation, hyperemic mucous membranes, severe dehydration, marked decrease in appetite, major loss of milk production and depression.

Among hematological parameter haemoglobin, mean corpuscular hemoglobin, mean corpuscular haemoglobin concentration were significantly increased ( $P<0.05$ ). Total erythrocyte count, packed cell volume, total leucocyte count and mean corpuscular volume were non-significantly increased while total platelet count had significantly ( $P<0.01$ ) decreased. Differential leucocyte count including neutrophil were significantly ( $P<0.01$ ) increased, lymphocyte had significantly decreased ( $P<0.01$ ) while in monocyte, eosinophil and basophil had non-significant changes occurred in heat stroke suffered cattle as compared to healthy cattle.

Among biochemical parameters serum aspartate aminotransferase (AST), serum alanine aminotransferase (ALT), serum alkaline phosphatase (ALP), blood urea nitrogen, creatinine, creatinine kinase, lactate and lactate dehydrogenase had increased

significantly ( $P < 0.01$ ) while blood glucose, serum sodium, potassium, inorganic phosphorus and calcium had decreased significantly ( $P < 0.01$ ) in heat stroke suffered cattle as compared to healthy cattle.

All the sixteen cases received isotonic saline solution (0.9% NaCl) and Ringers lactate @ 20–50 ml/kg b.wt, IV based on degree of dehydration. Afterwards they were sprayed with a water hose on the midline of the back with tap water (15-16°C). Fans were immediately placed in front of the animal to promote evaporative cooling; this was done continuously until their body temperature reached 101.5°F. Dexamethasone @ 0.1mg/kg b.wt. IV was given immediately and Ascorbic acid @ 25 mg/kg b.wt. IV was given for 3 days to reduce the stress. Oxytetracycline @ of 10 mg/kg b.wt. IV was given for 3 days. When temperature comes down to normal Vit B complex- Tribivet (Intas Pvt. Ltd.) containing Vit B<sub>1</sub> 50 mg, B<sub>6</sub> 50 mg and B<sub>12</sub> 500 mcg per ml, 10ml i/m was given for 3 days. Antioxidant supplementation was done with Liq. Intavita-NH (Intas Pvt. Ltd.) containing Vit A 1200 IU, Vit D<sub>3</sub> 6000 IU, Vit E 48 mg, Biotin 50 mcg and Nicotinamide 3 mg per ml, 10ml orally daily for 10 days. Probiotic bolus Triboost (Biotech Vety. Pharma. Pvt. Ltd.) containing Live yeast culture 3 gram, Live lactobacillus sporogenes culture 20 million CFU, Amino acid 2 gram, Liver extract 5 mg and Fungal diastase 50 mg per bolus, 2 bid for 3 days were given to boost up rumen microorganisms.

Efficacy of the treatment was seen with regard to clinical, haematological and biochemical parameters before and after treatment. The three day post treatment clinico-haemato-biochemical study revealed that animals recovered clinically but ALT, AST, LDH, Lactate, creatinine, creatinine kinase and BUN did not attain their normalacy however three animals got paralyzed.

The paralyzed cattle were further managed by putting the cattle on slings for one hour at stretched for 4-5 instances and nervine tonic, Neuroxin-M (Zydus AHL) containing Methyl Cobalamin 500 mcg, Pyridoxine 50 mg and Nicotinamide 50 mg per ml.

The present study concluded that evaporative cooling therapy, as well as simultaneous fluid therapy, dexamethasone, ascorbic acid, broad spectrum antibiotics, Vit B complex- Tribivet (Intas Pvt. Ltd.) containing Vit B<sub>1</sub> 50 mg, B<sub>6</sub> 50 mg and B<sub>12</sub> 500 mcg per ml, antioxidant Liq. Intavita-NH (Intas Pvt. Ltd.) containing Vit A 1200 IU, Vit D<sub>3</sub> 6000 IU, Vit E 48 mg, Biotin 50 mcg and Nicotinamide 3 mg per ml, probiotic bolus Triboost (Biotech Vety. Pharma. Pvt. Ltd.) containing Live yeast culture 3 gram, Live lactobacillus sporogenes culture 20 million CFU, Amino acid 2 gram, Liver extract 5 mg and Fungal diastase 50 mg per bolus, were effective if the heat stroke affected animals could be detected and treated as early as possible without any complication.

## 6. LITERATURE CITED

---

---

- Abd-Alkareem, A. B., Waleed, Y. K. and AL-Hellou, M.F. (2015). Effect of season on some hematological, biochemical and some hormone of local Iraqi black female goats. *Bas. J. Vet. Res.*, 14 (1): 52-61.
- Abdalla, M. A., Ibrahim, M. Y. and Hassan, Y. Y. (2009). Seasonal variations in erythrocyte and leukocyte indices and serum proteins of female Nubian goats. *Middle-East Journal of Scientific Research*, 4 (3): 168-174.
- Abdel-Fattah, M.S. (2014). Changes in body fluids and plasma electrolytes ( $\text{Na}^+$  and  $\text{K}^+$ ) concentrations of Balady and Damascus goats exposed to heat stress in Desert of Sinai, Egypt. *World Applied Sciences Journal*, 30 (5): 534-542.
- Abeni, F., Calamari, L. and Stefanini, L. (2007). Metabolic conditions of lactating Friesian cows during the hot season in the Po Valley. Blood indicators of heat stress. *Int. J. Bio meterol.*, 52:87-96.
- Adenkola, A.Y., Ayo, J.O., Sackey, A.K.B. and Adelaiye, A. B. (2009). Haematological and serum biochemical changes in pigs administered with ascorbic acid and transported by road for four hours during the harmattan season. *Journal of Cell and Animal Biology*, 3 (2): 21-28.

- Alam, M. M., Hashem, M. A., Rahman, M. M., Hossain, M. M., Haque, M. R., Sobhan, Z. and Islam, M. S. (2011). Effect of heat stress on behavior, physiological and blood parameters of goat. *Progress. Agric.*, 22(1 & 2): 37–45.
- Al-Haidary, A. (2004). Physiological responses of naimey sheep to heat stress challenge under semi-arid environments. *Int. J. Agri. Biol.*, 6 (2): 2004.
- Al-Tamimi, H.J. (2007). Thermoregulatory response of goat kids subjected to heat stress. *Small Rumin Res.*, 71: 280–285.
- [Alzeer, A.H.](#), [El-Hazmi, M.A.](#), [Warsy, A.S.](#), [Ansari, Z.A.](#) and [Yrkendi, M.S.](#) (1997). Serum enzymes in heatstroke: prognostic implication. [Clin Chem.](#), 43 (7):1182-7.
- Alzeer, A.H., Al-Arifi, A., Warsy, A.S., Ansari, Z., Zhang, H. and Vincent, J.L. (1999). Nitric oxide production is enhanced in patients with heat stroke. *Intensive Care Med.*, 25: 58–62.
- Anonymous. (1989). Endotoxins in heatstroke. *Lancet*, 2: 1137-8.
- Armstrong, D. V. (1994). Heat stress interactions with shade and cooling. *J. Dairy Sci.*, 77: 2044-2050.

- Armstrong, L.E., Crago, A.E., Adams, R., Roberts, W.O. and Maresh, C.M. (1996). Whole-body cooling of hyperthermic runners. Comparison of two field therapies. *Am J Emerg Med*, 14:355-358.
- Backer, H.D., Shopes, E., Collins, S.L. and Barkan, H. (1999). Exertional heat illness and hyponatremia in hifers. *Am J Emerg Med*, 1: 532–539.
- BAHS, (2014). Basic Animal Husbandry & Fisheries Statistics. Ministry Of Agriculture, Department Of Animal Husbandry, Dairying and Fisheries Krishi Bhawan, New Delhi.
- Barrow, M.W. and Clark, K.A. (1998). Heat-Related illnesses. *Am Fam Physician*, 58: 749–759.
- [Beatty, D.T.](#), [Taylor, B.A.](#), [Pethick, D.](#), [McCarthy, M.](#) and [Maloney, S.K.](#) (2006). Physiological responses of *Bos Taurus* and *Bos indicus* cattle to prolonged, continuous heat and humidity. *J Anim Sci.*, 84 (4): 972-85.
- Beede, D. and Collier, R. (1986). Potential nutritional strategies for intensively managed cattle during thermal stress. *Journal of Animal Science*, 62: 543.
- Blecha, F., Boyles, S.L. and Riley, J.G. (1984). Shipping suppresses lymphocyte blastogenic responses in Angus and Brahmin Cross Angus feeder calves. *J. Anim. Sci.*, 59: 576.

- Bligh, J. (1985). Temperature regulation, in *Stress physiology in livestock. Basic principles*, 1, edited by M.K. Yousef. Boca Raton, Florida: CRC Press, pp. 76-96.
- Bouchama, A., Cafege, A., Robertson, W., Al-Dossary, S. and El-Yazigi, A. (1991). Mechanisms of hypophosphatemia in humans with heatstroke. *J Appl Physiol*, 71: 328–332.
- Bouchama, A. and Knochel, JP. (2002). Heat stroke. *N Engl J Med.*, 346: 1978–1988.
- Bouchama, A., Roberts, G. and Al Mohanna, F. (2004). Inflammatory, hemostatic, and clinical changes in a baboon experimental model for heatstroke. *J Appl Physiol.*, 98: 697–705.
- Bras, R. (2010). Changes in ruminal fermentation and mineral serum level in animals kept in high temperature environment. vol. 39 (4) <http://dx.doi.org/10.1590/S1516>.
- [Bray, D. \(1992\). Providing a comfortable environment for dairy cows. Southeast Dairy Herd Management Conference. Cooperative Extension Service, University of Georgia, Athens, G.A.](#)
- Bricknell, M.C.M. (1995). Heat illness – a review of military experience. *J R Army Med Corps.*, 141 (1): 157–166.

- Bruchim, Y., Klement, E., Saragusty, J., Finkeilstein, E., Kass, P. and Aroch, I. (1999–2004). Heat stroke in dogs: A retrospective study of 54 cases and analysis of risk factors for death. *J Vet Intern Med.*, 20: 38–46.
- Chakrabarti, A. (2006). Text Book of Clinical Veterinary Medicine. 3<sup>rd</sup> Ed. Kalyani Publisher, New Delhi, pp. 78-79.
- Chandrabhan, Singh, S. V., Hooda, O. K., Upadhyay, R. C. and Beenam (2013). Influence of temperature variability on physiological, hematological and biochemical profiles of growing and adult Karan Fries cattle. *Indian Journal of Animal Sciences*, 83 (10): 1090–1096.
- Chaudhary, S. S., Singh, V. K., Upadhyay, R.C., Puri, G., Odedara, A. B. and Patel, P. A. (2015). Evaluation of physiological and biochemical responses in different seasons in Surti buffaloes - *Veterinary World*, 8 (6): 727-731.
- Conrad, J. (1985). Feeding of farm animals in hot and cold environments. In: YOUSEF, M.K. (ed.) *Stress Physiology in Livestock, Vol. 2: Ungulates*. CRC Press, Boca Raton, FL, U.S.A., pp. 78-82.
- Correa-Calderon, A., Armstrong, D., Ray, D., De Nise, S., Enns, M. and Howison, C. (2004) Thermoregulatory responses of Holstein and brown Swiss heat-stressed dairy cows to two different cooling systems. *Int. J. Bio-meteorol.*, 48 (3): 142-148.
- Cragoe, R. (2010). Nutritionist Consulting Service. <http://dx.doi.org/10.16.90/S14169>

- Damanhour, Z.A. and Tayeb, O.S. (1992). Animal models for heat stroke studies. *J Pharmacol Toxicol.*, 28: 119–127.
- Dasgupta, S. C. (1945). Cow in India, 1: 115-117.
- Davenport, H.W. (1974). The ABC of Acid-Base Chemistry, 6<sup>th</sup> ed. Chicago: The University of Chicago press, pp. 134-145.
- Davis, H. (2004): Nursing management of the heatstroke patient, ACVIM conference proceedings, pp. 26-30.
- Dematte, J.E., O'Mara, K. and Buescher, J. (1998). Near-fatal heat stroke during the 1995 heat wave in Chicago. *Ann Intern Med.*, 129: 173–81.
- Dougherty, R.W. (1971). Environment and physiopathology, a guide to environmental research on animals. Washington, DC: National Academy of Science, pp. 78-80.
- Drobatz, K.J. and Macintire, D.K. (1996). Heat-induced illness in dogs: 42 cases (1976–1993). *J Am Vet Med Assoc.*, 209: 1894–1899.
- Drobatz, K.J. (2004), Heatstroke: Assessment and stabilization, Atlantic Coast Veterinary Conference Proceedings, pp. 35-67.
- DU Preez, J.H. (2000). Parameters for the determination and evaluation of heat stress in dairy cattle in South Africa. *Onderstepoort Journal of Veterinary Research*, 67: 263-271

- El-Nouty, F.D., Elbanna, I.M., Davis, T.P. and Johnson, H.D. (1980). Aldosterone and ADH response to heat and dehydration in cattle. *J. Appl. Physiol.*, 48: 249–255.
- Flournoy, S., Macintire, D.K. and Wohl, J. (2003). Heatstroke in dogs: Clinical signs, treatment, prognosis, and prevention. *Compendium*, 25: 422–431.
- Gantner, V., Mijic, P., Kuteroyal, K., Solic, D. and Gantner, R. (2011) Thermal humidity index values and their significance on daily production of dairy cattle. *Mljekarstvo*, 61 (1): 56-63.
- Giercksky, T., Boberg, K.M., Farstad, I.N., Halvorsen, S. and Schrupf, E. (1999). Severe liver failure in exertional heat stroke. *Scand J Gastroenterol*, 34: 24–27.
- Grogan, H. and Hopkins, P.M. (2002). Heat stroke: Implications for critical care and anaesthesia. *Br J Anaesth.*, 88: 700–707.
- Gross, W.B. and Siegel, H.S.C. (1983). Evaluation of the Heterophil/Lymphocyte ratio as a measure of stress in chickens. *Avi. Diseases*, 27: 972-979.
- Hansen, P. (2004). Physiological and cellular adaptations of Zebu cattle to thermal stress. *Animal Reproduction Science*, 82: 349-360.

Hashim, I.A. (2010). Clinical biochemistry of hyperthermia, 47 (6): 516-23.

Haque, N., Ludri, A., Hossain, S.A. and Ashutosh, M. (2013). Impact on hematological parameters in young and adult Murrah buffaloes exposed to acute heat stress. *Buffalo Bulletin*, 32 (4): 321-326.

Howorth, P.J.N. (1995). The biochemistry of heat illness. *J R Army Med. Corps.*, 141: 40–41.

Ingram, D.L. and Legge, K.F. (1969). The effect of environmental temperature on respiratory ventilation in the pig. *Respiratory Physiology*, 8:1.

Jain, N.C. (1986). Haematologic techniques. In: Schalm's Veterinary Haematology. Lea and Febiger, Philadelphia, pp: 20-86.

Jain, K., Sharma, J., Bhardawaj, M., Sharma, P. G., Madhyani, S., Rohtagi, D., Verma, A., Akanksha, Roopashi, Kaur, H., Shama, I. J., Vanshika, Loomba, P., Aarunita, Isha, Jain, P., Chauhan, N. R., Prabha, L., Kapoor, M., Goel, A., Taneja, J., Jain, A., Baburaj, T. P., Singh, B. S. and Nanda, S. (2015). *DU Journal of Undergraduate Research and Innovation*, 1 (3): 49-56.

Jessen, C. (1977). Interaction of air temperature and core temperatures in thermoregulation of the goat. *J Physiol.*, 264: 585–606.

- Johnson, K.G. (1970). Sweating rate and electrolyte content of skin secretions of *Bos taurus* and *Bos indicus* cross-bred cows. *J. Agric. Sci. Camb.*, 75: 397-402.
- Johnson, S.I., Mc Michael, M. and White, G. (2006): Heatstroke in small animal medicine: a clinical practice review. *J Vet Emerg Crit Care*, 16:112-119.
- Kadzere, C. T., Murphy, M. R., Silanikove, N. and Maltz, E. (2002). Heat stress in lactating dairy cows: a review. *Livestock Production Science*, 77: 59-91.
- Kasahara, T., Ozawa, T., Tamura, K., Shibata, A. and Muto, N. (1979). A case of sunstroke with elevation of multiple serum enzymes. *Nihon Naika Gakkai Zasshi*, 10:1126–1132.
- Katamoto, H., Fukuda, H., Oshima, I., Ishikawa, N. and Kanai, Y. (1998). Nitroblue tetrazolium reduction of neutrophils in heat stressed goats is not influenced by selenium and vitamin E injection. *J Vet Med Sci.*, 60: 1243–1249.
- Kelly, W.R. (1974). Hyperthermia in: *Veterinary Clinical Diagnosis*. 2<sup>nd</sup> Ed. London: Bailliere Tindal, pp. 23-24.
- Kibler, H.H. (1964). Environmental physiology and shelter engineering. With special reference to domestic animals. LXVII. Thermal effects of various temperature humidity combinations on Holstein cattle as measured by eight physiological responses. *Res Bull Mo Agric Exp Sta.*, 862: 40.

- Knochel, J.P. and Reed, G. (1994). Disorders of heat regulation. In: Narins RG, ed. Maxwell and Kleeman's clinical disorders of fluid and electrolyte metabolism. 5<sup>th</sup> Ed. New York: Mc Graw-Hill, pp. 1549-90.
- Knochel, JP. (1996). Exertional heat stroke-pathophysiology of heat stroke. In: Hyperthermic and Hypermetabolic Disorders, 2nd ed. Hopkins PM, Ellis FR, eds. Cambridge: Cambridge University Press, pp. 42–62.
- Kumar, S. B.V., Kumar, A. and Kataria, M. (2011). Effect of heat stress in tropical livestock and different strategies for its amelioration, *Journal of Stress Physiology & Biochemistry*, 7 (1): 45-54.
- Lee-Chiong, T.L. and Stitt, J.T. (1995). Heatstroke and other heat-related illnesses. The maladies of summer. *Post grad Med.*, 98: 26-36.
- Lin, J.C., Moss, B.R., Koon, J.L., Flood, C.A., Rowe, S., Martin, J.R., Brady, B., De Graves, F. and Smith, R.C. (1998a). Effect of sprinkling over the feed area and misting free stalls on milk production. *The professional Animal Scientist*, 14:102.
- Lin, J.C., Moss, B.R., Koon, J.L., Flood, C.A., Smith, R.C., Cummins, K.A. and Coleman, D.A. (1998b). Comparison of various fan, sprinkler and mister systems in reducing heat stress in dairy cows. *Appl. Eng. Agric.*, 14: 177.
- Macintire, D.K., Drobatz, K.J., Haskins, S.C. and Saxon, W.D. (2005). Miscellaneous emergencies, in *Manual of Small Animal Emergency and Critical Care Medicine*. Philadelphia, Williams & Wilkins, pp. 402-404.

- [Magazanik, A.](#), [Epstein, Y.](#), [Udassin, R.](#), [Shapiro, Y.](#) and [Sohar, E.](#) (1980). Tap water, an efficient method for cooling heat stroke victims-a model in dogs. *[A Viat Space Environ Med.](#)*, 51(9 Pt.1): 864-6.
- Mallonee, P.G., Beede, D. K., Collier, R. J. and Wilcox, C. J. (1985). Production and physiological responses of dairy cows to varying dietary potassium during heat stress. *J. Dairy Sci.*, 68: 1479-1487.
- Mazzaferro, E. M. (2004). Critical care for heat-induced illness, International Veterinary Emergency and Critical Care Symposium, pp. 37-43
- Mc Dowell, R.E. (1958). Physiological approaches to animal climatology. *Journal of Heredity*, 49:52-61.
- Mc Dowell, R.E. (1972). Improvement of livestock production in warm climates. San Francisco: W.H. Freeman & Co.
- Mc Dowell, R.E., Hooven, N.E. and Comoers, J.K. (1976). Effect of climate on performance of Holstein in first lactation. *Journal of Dairy Science*, 59: 965-973.
- Mc Farlane, J.M. and Curtis, S.E. (1989). Multiple concurrent stressors in chicks. Three effects on plasma corticosterone and the H/L ratio. *Poult. Sci.*, 68: 522-527.
- Mc Gugan and Elizabeth, A. (2001). "Hyperpyrexia in the emergency department". *Emer. Medi. Australasia*, 13 (1):116-120.

Mehta, S.R., Narayan, S. and Swamy, A.S. (1987). Heat stroke. *J Assoc Phy India*, 35: 822-5.

Mehta, S.R. and Jaswal, D.S. (2003). Heat Stroke. *Armed Forces Med J India*, 59: 140–143.

Mellion, M. and Shelton, G. (1998). Thermoregulation, heat illness, and safe exercise in heat. In: Office Sports Medicine. Mellion M, ed. Philadelphia: Mosby, pp. 45–57.

Mohamed, M.G. and Abdel-Hamid, O.M. (2010). Clinical, hematological and biochemical alterations in heat intolerance (panting) syndrome in Egyptian cattle following natural foot-and-mouth disease (FMD). *Trop Anim Health Prod.*, 42:1167-1173.

Powell, L. L. (2008). Canine Heatstroke. Complications NAVC clinician's brief .Critical care, pp. 13-16.

Radostits, O. M., Gay, C.C., Hinchcliff, K.W. and Constable, P. D. (2007). General systemic states. Hyperthermia. In: Veterinary Medicine. 10<sup>th</sup> edition. Philadelphia: Elsevier, pp. 47-48.

Rana, M. S., Hashem, M. A., Sakib, M. N. and Kumar, A. (2014). Effect of heat stress on blood parameters in indigenous sheep. *J. Bangladesh Agril. Univ.*, 12 (1): 91–94.

Randell, K. W. (2002). Evaluation and treatment of heat-related illnesses. *Am Fam Physician*, 65 (11): 2307-2314.

- Randell, K. W. and Ohio, M.D. (2002). State University College of Medicine and Public Health, Columbus, Ohio *Am Fam Physician*, 65 (11): 2307-2315.
- Randhawa, S.S., Chhabra, S., Randhawa, C.S., Zahid, U. and Dhaliwal, P.S. (2014). *Asian Pac J Trop Biomed.*, 4 (1): 272-274.
- Roger, G. (2005). Heat stroke In: Ettinger S.J., Feldman, E.C. Eds. Textbook of Veterinary Internal Medicine. 6th ed. Philadelphia: Saunders WB, pp. 437-440.
- Samal, L. (2013). Heat stress in dairy cows- reproductive problems and control measures. *International Journal of Livestock Research*, 3 (3): 14-22.
- Sanchez, W.K., Beede, D.K. and Cornell, J.A. (1994a). Interactions of sodium, potassium, and chloride on lactation, acid-base status, and mineral concentrations. *Dairy Sci.*, 77: 1661-1675.
- Shafie, M.M. (1985). Physiological responses and adaptation of water buffalo, in Stress physiology in livestock. Ungulates, 2<sup>nd</sup> edition, Youssef M.K., Boca Raton, Florida: CRC Press, pp. 67-80.
- Shapiro, Y. and Cristal, N. (1987). Hyperthermia and Heat stroke: Effects on acid-base balance, blood electrolytes and hepato-renal function. In Exespta media International Congress series, pp. 289-296.

- Sharma, A., Rodriguez, L., Mekonnen, G., Wilcox, C., Bachman, K. and Collier, R. (1983). Climatological and genetic effects on milk composition and yield. *Journal of Dairy Science*, 66: 119-126.
- Silanikove, N. (1987). Impact of shelter in hot Mediterranean climate on feed intake, feed utilization and body fluid distribution in sheep. *Appetite*, 9: 207–215
- Silanikove, N. (2000). Effects of heat stress on the welfare of extensively managed domestic ruminants. *Livest. Prod. Sci.*, 67: 1-18.
- Singh, S.P., Hooda, O.K., Kundu, S. S. and Singh, S.V. (2012). Biochemical changes in heat exposed buffalo heifers supplemented with yeast. *Trop. Anim. Heal. Prod.*, 44: 1383- 1387.
- Sirohi, S. (2010). Vulnerability of milk producers to climate change: technological and policy options for livelihood security. Published in the compendium of *National Symposium on Climate Change and Livestock Productivity in India*. NDRI, Karnal.
- Snedecor, G.W. and Cochran, W.G. (1994). Statistical methods. 8<sup>th</sup> Edn. Iowa state Univ. Press, Ames. Iowa, USA, pp.146-165.
- Sreedhar, S., Rao, K.S., Jeepalyam, S.P., Moorthy, R. S. and Reddy, V.P. (2013). Changes in hematocrit and some serum biochemical profile of Sahiwal and Jersey × Sahiwal cows in tropical environments. *Veterinarski arhiv.*, 83 (2): 171-187.

- [Srikandakumar, A.](#) and [Johnson, E.H.](#) (2004). Effect of heat stress on milk production, rectal temperature, respiratory rate and blood chemistry in Holstein, Jersey and Australian Milking Zebu Cows. [Trop Anim Health Prod.](#), 36 (7): 685-92.
- Strickland, J.T., Bucklin, R.A., Nordstedt, R., Beede, D.K. and Bray, D.R. (1989). Sprinkler and fan cooling system for dairy cows in hot, humid climates. *Appl. Eng. Agri.*, 5 (2): 231.
- Stoneham, M.D. and Price, D.I.A. (1992). Acid-base disturbances and heat-stress in the armed forces. *Lancet*, 339: 870-1.
- Tabor, B. (2007). Heat stroke in dogs. *Veterinary Technician*, pp. 228-241.
- Tayeb, O.S. and Marzouki, Z.M. (1990). Effect of dantrolene pre-treatment on heatstroke in Sheep. *Pharmacol Res.*, 22: 565–572
- Temizel, E. M., Senturk, S. and Kasap, S. (2009). Clinical, hematological and biochemical findings in Saanen goat kids with naturally occurring heat stroke. 37 (G): 236–241.
- Trinder, P. (1951). *Analyst* 56: 596 (cited in M.V.Sc. Thesis by Tinna, N.K. (1999). Studies on ketosis in goats (*Capra hircus*). RAU, Bikaner.
- [Tucker, L.E.](#), [Stanford, J.](#), [Graves, B.](#), [Swetnam, J.](#), [Hamburger, S.](#) and [Anwar, A.](#) (1985). Classical heatstroke: clinical and laboratory assessment. [South Med J.](#), 78 (1): 20-5.

- Turner, L.W., Chastin, J.P., Hemken, R.W., Gates, R.S. and Crist, W.L. (1989). Reducing heat stress in dairy cows through sprinkler and fan cooling. ASAE paper no. 89-4025. St. Joseph, MIASAE.
- Turner, L.W., Warner, R.C. and Chastin, J.P. (1992). Micro-sprinkler and fan cooling for dairy cows: Practical design considerations. Coop. Ext. Service AEN-75. University of Kentucky, Lexington, pp. 36-38.
- Upadhyay, R. C., Ashutosh, Raina, V. S. and Singh, S.V. (2009). Impact of climate change on reproduction functions of cattle and buffaloes. *Global climate change and Indian Agriculture*, pp.107–10.
- Van Der Linde, A., Kielblock, A.J., Rex, D.A. and Terblanche, S.E. (1992). Diagnostic and prognostic criteria for heatstroke with special reference to plasma enzyme and isoenzyme release patterns. *Int J Biochem.*, 24: 477–485.
- Varghese, G M., John G., Thomas, K., Abraham, O. C. and Mathai, D. (2005). Predictors of multi-organ dysfunction in heatstroke. *Emergency Med J.*, 22: 185–187.
- Vermunt, J. J. and Tranter, B. P. (2010). Heat stress in dairy cattle. A review and some of the potential risks associated with the nutritional management of this condition, pp. 212-221.

- Vitali, A., Segnalini, M., Bertocchi, L., Bernabucci, U., Nardone, A. and Lacetera, N. (2009). Seasonal pattern of mortality and relationships between mortality and temperature-global climate change humidity index in dairy cows. *Journal of Dairy Science*, 92: 3781-3790.
- Walters, M.J. (2002): Hyperthermia, in Wing field WE, Raffe MR (Eds): The Veterinary ICU Book, Jackson, WY, Teton New Media, pp.1130-1135.
- West, J. W., Yullinix, B. O. and Sandifer, G. (1991). Changing dietary electrolyte balance for dairy in cool and hot environments. *J. Dairy Sci.*, 74: 1662–1674.
- Wolfenson, D., Roth, Z. and Meidan, R. (2000). Impaired reproduction in heat-stressed cattle: basic and applied aspects. *Anim Reprod Sci.*, 60 (61): 535-547.
- www.Bikaner.Climatemps.com (2009-2015).11.06.15/1345

**Rajasthan University of Veterinary and Animal Sciences**

**College of Veterinary and Animal Science, Bikaner**

*CERTIFICATE - I*

Date.....

This is to certify that Mrs. **KIRAN** has successfully completed the comprehensive examination held on 31-10-2015 and 02-11-2015 as required under the regulations for the degree of MASTER OF VETERINARY SCIENCE.

**(ANIL AHUJA)**

Head

Department of Clinical Veterinary Medicine, Ethics and Jurisprudence

College of Veterinary and Animal Science

Bikaner

**Rajasthan University of Veterinary and Animal Sciences**

**College of Veterinary and Animal Science, Bikaner**

***CERTIFICATE - II***

Date.....

This is to certify that the thesis entitled “**Clinical and therapeutic studies of heat stroke in cattle**” submitted for the degree of **Master of Veterinary Science** in the subject of **Veterinary Clinical Medicine** of the Rajasthan University of Veterinary and Animal Sciences, Bikaner, embodies bonafide research work carried out by Mrs. **KIRAN** under my guidance and supervision and that no part of this thesis has been submitted for any other degree. The assistance and help received during this work have been fully acknowledged. The draft of the thesis was also approved by the advisory committee on 28-01-2016.

**(Anil Ahuja)**

Major Advisor

**(Anil Ahuja)**

Head

Department of Clinical Veterinary Medicine, Ethics and Jurisprudence

College of Veterinary and Animal Science Bikaner

**(Tribhuvan Sharma)**

Dean

College of Veterinary and Animal Science

Bikaner

**Rajasthan University of Veterinary and Animal Sciences**

**College of Veterinary and Animal Science, Bikaner**

*CERTIFICATE – III*

Date.....

This is to certify that the thesis entitled “**Clinical and therapeutic studies of heat stroke in cattle**” submitted by Mrs. **KIRAN**, to the Rajasthan University of Veterinary and Animal Sciences, Bikaner, in partial fulfillment for the degree of **Master of Veterinary Science** in the subject of **Veterinary Clinical Medicine** after recommendation by the external examiner was defended by the candidate before the following members of the examination committee. The performance

of the candidate in the oral examination on his thesis has been found satisfactory; we therefore recommend that the thesis be approved.

**(Anil Ahuja)**

Major Advisor

**(Deepika Goklani)**

Advisor

**(Anju Chahar)**

Advisor

**(T.K.Gahlot)**

Dean PG Nominee

**(Anil Ahuja)**

**APPROVED**

Head

Department of Clinical Veterinary Medicine, Ethics and Jurisprudence

College of Veterinary and

Animal Science, Bikaner

DEAN

Post Graduate Studies

RAJUVAS, Bikaner

**Rajasthan University of Veterinary and Animal Sciences**

**College of Veterinary and Animal Science, Bikaner**

CERTIFICATE – IV

Date.....

This is to certify that Mrs. **Kiran**, of the Department of Clinical Veterinary Medicine, Ethics and Jurisprudence, College of Veterinary and Animal Science, Bikaner has made all corrections /modifications in the thesis entitled “**Clinical and therapeutic studies of heat stroke in cattle**”, which were suggested by the external examiner and the advisory committee in the oral examination held on..... The final copies of the thesis duly bound and corrected were submitted on.....are enclosed herewith for approval.

(**Anil Ahuja**)  
Major Advisor

Enclosed: One original and two copies of bound thesis.

Forwarded to the Dean, Post Graduate Studies, RAJUVAS, Bikaner through the Dean, CVAS, Bikaner.

**(Tribhuwan Sharma)**

Dean

CVAS, Bikaner

**(Anil Ahuja)**

Head

Department of Clinical Veterinary Medicine, Ethics and Jurisprudence

CVAS, Bikaner

**APPROVED**

Dean

Post Graduate Studies

RAJUVAS, Bikaner

## ACKNOWLEDGEMENT

*Writing of this page has come in this educational pilgrimage with the blessings of my parents.*

*I owe my sincere gratitude and heartiest appreciation to all those helping hands and minds that has helped me in the preparation of this manuscript.*

*I am profoundly thankful to my worthy and reverend major advisor Dr. Anil Ahuja, Professor and Head, Department of Clinical Veterinary Medicine, Ethics and Jurisprudence, College of Veterinary and Animal Science, Bikaner cum Director Clinics, RAJUVAS for his guidance, cooperation and persistent motivation during entire period of study.*

*I am in extreme drought of words to express my heartiest regards to express my indebtedness and heartfelt thanks to Prof. A.K. Gahlot, Honorable Vice-Chancellor, Rajasthan University of Veterinary and Animal Sciences , Prof. Tribhuwan Sharma, Dean, College of Veterinary and Animal Sciences, Prof. Rakesh Rao, Director of Research, (Veterinary and Animal Sciences) Rajasthan University of Veterinary and Animal Sciences, Bikaner.*

***It is my pleasant duty to express the profound sense of gratitude to***

***members of advisory committee, Dr. Anju Chahar, Professor & Head, Dept. of Epidemiology and Veterinary Preventive Medicine, Dr. Deepika Goklani, Assistant Professor, Dept. of Clinical Veterinary Medicine, Ethics and Jurisprudence, and Dean PGS Nominee Dr. T.K. Gahlot, Professor (Reemployed) Dept. of Veterinary Surgery and Radiology for their valuable suggestions. My sincere thanks are due to Dr. D.K. Bihani, Dr. A.P. Singh, Professors, Dr. Sunita Choudhary, Dr. J.P. Kachhawa, Dr. S.K. Vyas, (Reemployed) Assistant Professors Dr.***

***Ankita Sharma, Teaching Associate, Dept. of Clinical Veterinary Medicine, Ethics and Jurisprudence and Dr. Ram Hari Meena, Teaching Associate, T.V.C.C. for their moral support, amicable help, kind cooperation and encouragement in conducting the research.***

*My sincere thanks are due to Dr. R.K. Tanwar and Dr. Fakhruddin, Professors (Reemployed), Dept. of Epidemiology and Veterinary Preventive Medicine, for their moral support, amicable help, kind cooperation and encouragement in conducting the research. The financial assistance provided by the Rajasthan University of Veterinary and Animal Sciences, Bikaner in the form of stipend during the period of studies is thankfully acknowledged.*

*I am also thankful to my colleagues, Rakesh Bhojani, Jyoti Yadav, Aprajita Johari, Vikas kumar Meena, Amandeep Singh, Parmal, Mukesh Meena, Diksha Sharma and Nareshi Meena for their mental support during my work.*

*My deepest gratitude goes to my family for their unflagging love and support throughout my life; this dissertation is simply impossible without them. I am indebted to my father, Mr. Ram Kumar Beniwal & my mother Mrs. Birma Devi, my sisters Dr. Pushpa, Anita and Ravina, my brother Vishal, my father in law Mr. Narayan Ram and mother in law Mrs. Tulsi Devi, my husband Dr. Jaiprakash Khichar and brother in law Dr. Shrawan Khichar for their care and support.*

*I take this opportunity to express my gratefulness to Miss Monika and Sh. Laxman Ram, T.A., Sh. Ram Ratan Meel, Riding Instructor, Sh. Chandra Shekhar, T.A., Sh. Gopal, Sh. Rajesh Bhadani, Sh. Bhuralal and Mrs. Sua Bai, Fourth class, Department of Clinical Veterinary Medicine, Ethics and Jurisprudence, CVAS, Bikaner for providing assistance and friendly atmosphere. I take this opportunity to extend my deep sense of gratitude and words of appreciation towards those, who directly or indirectly helped me during the pursuit of my present study.*

*Above all I am thankful to Almighty God for his mercy on me, without which nothing could have been possible for me.*

**Place:**

**Date:**

**KIRAN**

**LIST OF CONTENTS**

<b>S.No.</b>	<b>Titles</b>	<b>Page No.</b>
<b>1.</b>	<b>INTRODUCTION</b>	<b>1-5</b>
<b>2</b>	<b>REVIEW OF LITERATURE</b>	<b>6-26</b>
<b>3.</b>	<b>MATERIALS AND METHODS</b>	<b>27-37</b>

<b>4.</b>	<b>RESULTS AND DISCUSSION</b>	<b>38-75</b>
<b>5.</b>	<b>SUMMARY</b>	<b>76-78</b>
<b>6.</b>	<b>LITERATURE CITED</b>	<b>79-93</b>
	<b>ABSTRACT (English &amp; Hindi)</b>	
	<b>APPENDIX</b>	<b>i-xvii</b>

---

LIST OF TABLES

<b>Table No.</b>	<b>Title</b>	<b>Page No.</b>
1	Environmental temperature, relative humidity and temperature humidity index	39
2	Prevalence of heat stroke in cattle	40
3	Major clinical manifestations of heat stroke suffered cattle	41
4	Mean $\pm$ SE values of body temperature, pulse rate and respiration rate in healthy control and heat	

<b>Table No.</b>	<b>Title</b>	<b>Page No.</b>
	stroke suffered cattle	43
5	Mean±SE values of haematological parameters in apparently healthy and heat stroke suffered cattle	50
6	Mean±SE values of differential leucocyte count in apparently healthy and heat stroke suffered cattle	56
7	Mean±SE values of biochemical parameters in apparently healthy and heat stroke suffered cattle	66

LIST OF FIGURES

<u>Fig. No.</u>	<u>Titles</u>	<u>Page No.</u>
<u>1</u>	Mean values of body temperature ( <sup>0</sup> F) in healthy and heat stroke suffered cattle	44
<u>2</u>	Mean values of pulse rate (per minute) in healthy and heat stroke suffered cattle	44
<u>3</u>	Mean values of respiration rate (per minute) in healthy and heat stroke suffered cattle	45
<u>4</u>	Mean values of haemoglobin (g/dl) in healthy and heat stroke suffered cattle	51
<u>5</u>	Mean values of packed cell volume (per cent) in healthy and heat stroke suffered cattle.	51
<u>6</u>	Mean values of total erythrocyte count (10 <sup>12</sup> /lt) in healthy and heat stroke suffered cattle	52
<u>7</u>	Mean values of MCH (pg), MCV (fl) and MCHC (per cent) in healthy and heat stroke suffered cattle	52
<u>8</u>	Mean values of total leucocyte count (10 <sup>6</sup> /lt) in healthy and heat stroke suffered cattle	53
<u>9</u>	Mean values of total platelet count (10 <sup>6</sup> /lt) in healthy and heat stroke suffered cattle	53
<u>10</u>	Mean values of differential leucocyte count (per cent) in healthy and heat stroke suffered cattle	57
<u>11</u>	Mean values of SGPT (U/lt) in healthy and heat stroke suffered cattle	67
<u>12</u>	Mean values of SGOT (U/lt) in healthy and heat stroke suffered cattle	67

<u>Fig. No.</u>	<u>Titles</u>	<u>Page No.</u>
<b><u>13</u></b>	Mean values of alkaline phosphatase (U/lt) in healthy and heat stroke suffered cattle	68
<b><u>14</u></b>	Mean values of lactate dehydrogenase (U/lt) in healthy and heat stroke suffered cattle	68
<b><u>15</u></b>	Mean values of creatinine kinase (U/lt) in healthy and heat stroke suffered cattle	69

<b><u>16</u></b>	Mean values of blood urea nitrogen (mg/dl) in healthy and heat stroke suffered cattle	69
<b><u>17</u></b>	Mean values of serum creatinine (mg/dl) in healthy and heat stroke suffered cattle	70
<b><u>18</u></b>	Mean values of serum lactate (mmol/lt) in healthy and heat stroke suffered cattle	70
<b><u>19</u></b>	Mean values of serum glucose (mg/dl) in healthy and heat stroke suffered cattle	71
<b><u>20</u></b>	Mean values of serum sodium (mmol/lt) in healthy and heat stroke suffered cattle	71
<b><u>21</u></b>	Mean values of serum potassium (mmol/lt) in healthy and heat stroke suffered cattle	72
<b><u>22</u></b>	Mean values of serum calcium (mg/dl) in healthy and heat stroke suffered cattle	72
<b><u>23</u></b>	Mean values of serum phosphorous (mg/dl) in healthy and heat stroke suffered cattle	73



### LIST OF APPENDICIES

<b>Appendix. No.</b>	<b>Title</b>	<b>Page No.</b>
1	History sheet – Clinical and therapeutic studies of heat stroke in cattle	i
2	Temperature, pulse and respiration in healthy cattle (n=10)	iii
3	Temperature, pulse and respiration in heat stroke suffered cattle before treatment (n=16)	iv
4	Temperature, pulse and respiration in heat stroke suffered cattle after treatment (n=16)	v

5	Haematological parameters of healthy cattle (n=10)	vi
6	Haematological parameters of heat stroke suffered cattle before treatment (n=16)	vii
7	Haematological parameters of heat stroke suffered cattle after treatment (n=16)	viii
8	Differential leucocyte count of healthy cattle (n=10)	ix
9	Differential leucocyte count of heat stroke suffered cattle before treatment (n=16)	x
10	Differential leucocyte count of heat stroke suffered cattle after treatment (n=16)	xi
11	Biochemical parameters in serum of healthy cattle (n=10)	xii
12	Biochemical parameters of serum in heat stroke suffered cattle before treatment (n=16)	xiii
13	Biochemical parameters of serum in heat stroke suffered cattle after treatment (n=16)	xiv
14	Biochemical parameters of serum in healthy cattle (n=10)	xv

15	Biochemical parameters of serum in heat stroke suffered cattle before treatment (n=16)	xvi
16	Biochemical parameters of serum in heat stroke suffered cattle after treatment (n=16)	xvii

**Appendix 1: History sheet – Clinical and therapeutic study of heat stroke in cattle**

Case No..... Date.....

Owner’s name and address.....

Ph. No..... Breed..... Age.....

Gender..... Body Weight..... Ticks-Present/Absent.....

Feeding- Concentrate/ Dry / Green roughage.....

Lactating/ Dry.....Housing- Loose/Intensive.....

Exposure to high environment temperature-yes/no.....

**Previous history of any ailment:**

**Clinical Findings**

Temperature (°F) .....Respiration (/min) ..... Pulse rate (/min)..... Dehydration—On the basis of skin tainting, skin elasticity (%).....

Conjunctival mucous membrane- Normal/Congested/Anaemic/Pale..... Appetite-Normal/Anorexia.....Muzzle-Dry/Wet.....

Ruminal motility ..... (../2min.)... Rumination-Present/Absent.....

Defecation-Normal/Constipation/Diarrohea.....

Urination-oliguria/polyuria/normal.....Urine colour-Normal/pale/Red.....

Hyper- salivation-yes/no .....

Animal standing/sternal recumbency /Lateral recumbency..... Polydipsia/Normal.....Behaviour-dull/excited/bright.....

Hair Coat-Dry /wet ..... Milk production (kg) – normal/decreased General Condition.....

Other .....

**Haematology:-**

Hb (gm. %)..... PCV (%)..... TLC (Thousand/C.mm.....

Platelets (Thousand/C.mm.) .....DLC –N%..... E%..... B%..... M%..... L%..... TRBC (million/c.mm)..... Other.....

**Serum Biochemistry:-**

SGOT (U/lit).....SGPT (U/lit).....ALP(U/lit)..... LDH (U/lit).....

BUN (mg/dl)..... Creatinine (mg/dl)..... Creatinine Kinase (U/lit).....

Potassium (mEq/lit)..... Sodium (mEq/lit).....calcium (mg/dl)..... Phosphate (mg/100ml).....Blood Glucose (mg/dl)..... Lactate (mg/dl).....

**Treatment:**

Recovery: - .....

**Appendix 2: Temperature, Pulse and Respiration in healthy cattle**

**(n=10)**

S. No.	Temperature (°F)	Pulse Rate (Rate/Min)	Respiration Rate (Rate/Min)
1.	102.4	66	18
2.	101.2	58	23
3.	101.8	58	25
4.	101.6	56	26
5.	102.4	68	30

6.	101.8	62	24
7.	102.2	60	22
8.	102.8	64	26
9.	100.8	62	27
10.	102.4	68	24
Mean	101.94	62.20	24.50
± SE	± 0.1950	± 1.2792	± 0.9618
Range	100.8 - 102.8	56 - 68	18 - 30

**Appendix 3: Temperature, Pulse and Respiration in heat stroke suffered cattle before treatment (n=16)**

S. No.	Temperature (°F)	Pulse Rate (Rate/Min)	Respiration Rate (Rate/Min)
1.	107.2	120	110
2.	107.8	115	115
3.	107.6	112	110
4.	106.8	118	102
5.	107.8	120	105
6.	108.6	130	130
7.	107.3	127	100
8.	107.7	95	108

9.	107.6	108	127
10.	106.7	106	135
11.	107.6	118	120
12.	107.2	122	115
13	108.1	90	118
14.	106.4	121	120
15.	107.4	92	116
16.	108.5	125	125
Mean ± SE	107.52 ± 0.1450	113.69 ± 2.9892	115.50 ± 2.4859
Range	106.4 - 108.5	90 - 130	100 - 135

**Appendix 4: Temperature, Pulse and Respiration in heat stroke suffered cattle after treatment (n=16)**

S. No.	Temperature (°F)	Pulse Rate (Rate/Min)	Respiration Rate (Rate/Min)
1.	101.8	69	35
2.	101.9	68	39
3.	102.9	64	32
4.	101.7	64	30
5.	101.8	66	40
6.	102.5	65	29
7.	102.3	70	39
8.	102.2	65	31
9.	102.3	67	35
10.	102.2	60	33
11.	102.1	62	36
12.	101.8	61	40
13.	101.7	59	31
14.	102.1	69	38
15.	102.0	63	37
16.	101.8	70	38
Mean ± SE	102.07 ± 0.0794	65.12 ± 0.8564	35.19 ± 0.9066
Range	101.7 - 102.9	59 - 70	29 - 40

**APPENDIX 5: Haematological parameters of healthy cattle (n=10)**

S. No.	Hb (g/dl)	PCV (Lt/l)	TEC ( $10^{12}$ /lt)	TLC ( $10^9$ /lt)	Total Platelate count ( $10^9$ /lt )	MCH (pg)	MCV (fl)	MCHC (g/dl )
1.	12.4	38.4	8.44	8.85	400	14.69	45.49	32.29

2.	11.2	36.2	6.42	8.15	324	17.44	56.38	30.93
3.	9.2	28.8	6.39	7.45	450	14.39	45.07	31.94
4.	9.8	30.4	6.89	7.55	500	14.22	44.12	32.23
5.	10.6	33.2	7.18	8.25	510	14.76	46.23	31.92
6.	10.3	32.8	6.32	7.35	600	16.29	51.89	31.40
7.	11.8	36.0	7.68	8.55	350	15.36	46.83	32.77
8.	11.6	37.2	6.86	6.95	410	16.90	54.22	31.18
9.	8.2	26.8	5.84	6.15	575	14.04	45.89	30.59
10.	11.7	36.4	7.43	7.35	555	15.74	48.99	32.14
Mean ± SE	10.68± 0.4168	33.62± 1.2323	6.94± 0.2420	7.66± 0.2544	467.40 ± 28.6909	15.38± 0.3725	48.51± 1.3380	31.74± 0.2171
Range	8.2 -12.4	26.8-38.4	5.84 - 8.44	6.15 - 8.85	100 - 800	14.04 - 17.44	44.12 - 56.38	30.59 - 32.77

**APPENDIX 6: Haematological parameters of heat stroke suffered cattle before treatment (n=16)**

S. No.	Hb (g/dl)	PCV (lt/lt)	TEC ( $10^{12}$ /lt)	TLC ( $10^9$ /lt)	Total plateletes ( $10^9$ /lt)	MCH (pg)	MCV (fl)	MCHC (g/dl)
1.	11.4	35	6.85	8.95	200	16.64	51.09	32.57
2.	11.2	34.	6.87	8.65	285	16.30	49.49	32.94
3.	11.2	34	6.98	7.75	295	16.04	48.71	32.94
4.	12.1	37	7.68	6.55	300	15.75	48.17	32.70
5.	11.5	36	8.67	8.35	312	13.26	41.52	31.94
6.	11.2	35	8.98	6.35	378	12.47	38.97	32.00
7.	10.8	33	6.34	9.55	352	17.03	52.05	32.72
8.	11.6	32	7.25	6.45	288	16.00	44.13	36.25

9.	12.3	39	6.45	6.25	272	19.06	60.46	31.53
10.	11.7	34	7.34	5.75	370	15.94	46.32	34.41
11.	12.8	38	6.98	8.98	385	18.33	54.44	33.68
12.	11.6	35	5.95	9.23	287	19.49	58.82	33.14
13.	10.9	37	6.23	8.45	394	17.49	59.39	29.45
14.	12.5	38	6.67	7.89	292	18.74	56.97	32.89
15.	10.8	33	6.97	8.32	300	15.49	47.34	32.72
16.	11.1	35	6.94	8.45	340	15.99	50.43	31.71
Mean± SE	11.54 ± 0.1466	35.31 ± 0.4899	7.07 ± 0.1962	7.87 ± 0.2930	315.62 ± 12.3424	16.50 ± 0.4615	50.52 ± 1.5295	32.72 ± 0.3469
Range	6.5-8.7	20-30	4.98-8.98	5.75 - 9.23	200-394	12.47-19.49	38.97-60.46	29.45-36.25

**APPENDIX 7: Haematological parameters of heat stroke suffered cattle after treatment (n=16)**

S. No.	Hb (g/dl)	PCV (lt/lt)	TEC (10 <sup>12</sup> /lt)	TLC (10 <sup>9</sup> /lt)	Total platelete (10 <sup>9</sup> /lt)	MCH (pg)	MCV (fl)	MCHC (%)
1.	11.5	33	7.80	6.25	380	14.74	42.30	34.84
2.	10.5	32	6.30	7.56	450	16.66	50.79	32.81
3.	11.6	34	6.51	8.25	428	17.81	52.22	34.11
4.	10.5	32	7.98	7.34	400	13.15	40.10	32.81
5.	11.6	35	7.67	6.56	390	15.12	45.63	33.14
6.	12.4	38	6.90	8.12	470	17.97	55.07	32.63
7.	10.8	33	6.85	6.56	350	15.76	48.17	32.72
8.	10.6	32	6.50	6.87	358	16.30	49.23	33.12
9.	11.2	34	7.12	7.45	325	15.73	47.75	32.94
10.	10.8	31	7.23	7.78	365	14.93	42.87	34.83
11.	11.2	34	7.45	7.34	375	15.03	45.63	32.94
12.	10.9	32	6.89	7.24	382	15.82	46.44	34.06
13.	11.1	33	6.78	6.98	432	16.37	48.67	33.63
14.	12.0	37	6.97	8.23	428	17.21	53.08	32.43
15.	10.2	31	6.34	6.67	440	16.08	48.89	32.90

16.	11.8	36	6.45	6.89	360	18.29	55.81	32.77
Mean± SE	11.17± 0.1478	33.56 ± 0.4997	6.98 ± 0.1269	7.25 ± 0.1503	395.80 ± 9.9754	16.06 ± 0.3260	48.29 ± 1.0802	33.29 ± 0.1858
Range	8.1-12.0	25-36	6.50-8.51	6.25-8.25	325-470	13.15-18.29	40.10-55.81	32.43-34.84

**Appendix 8: Differential leucocyte count of healthy cattle (n=10)**

S. No.	Lymphocyte	Monocyte	Neutrophils	Eosinophils	Basophils
--------	------------	----------	-------------	-------------	-----------

1.	62	2	32	4	0
2.	62	3	30	5	0
3.	65	4	30	1	0
4.	62	0	34	4	0
5.	70	2	25	3	0
6.	68	3	27	2	0
7.	67	4	24	5	0
8.	64	3	30	3	0
9.	64	3	32	1	0
10.	62	2	33	3	0
Mean	64.60±	2.60±	29.70±	3.10±	0
± SE	0.9092	0.3711	1.0651	0.4582	
Range	62-70	0-4	24-34	1-5	0-0

**Appendix 9: Differential leucocyte count of heat stroke suffered cattle before treatment (n=16)**

S. No.	Lymphocyte	Monocyte	Neutrophils	Eosinophils	Basophils
1.	50	4	45	1	0
2.	55	3	40	2	0
3.	56	2	41	1	0
4.	58	1	39	2	0

5.	45	4	48	3	0
6.	43	2	51	4	0
7.	53	3	40	4	0
8.	59	5	35	1	0
9.	54	2	42	2	0
10.	49	2	46	3	0
11.	48	3	47	2	0
12.	51	1	45	3	0
13.	43	4	52	1	0
14.	46	4	46	4	0
15.	45	3	49	3	0
16.	50	1	47	2	0
Mean ± SE	50.31 ± 1.2491	2.75 ± 0.2997	44.56 ± 1.1318	2.37 ± 0.2633	0
Range	43-59	1-5	35-52	1-4	0-0

**Appendix 10: Differential leucocyte count of heat stroke suffered cattle after treatment (n=16)**

S. No.	Lymphocyte	Monocyte	Neutrophis	Eosinophils	Basophils
1.	59	1	36	4	0
2.	58	2	36	4	0
3.	60	3	34	3	0
4.	62	3	33	2	0
5.	59	2	35	4	0
6.	61	2	32	3	0
7.	59	3	36	2	0
8.	60	2	37	1	0
9.	62	4	33	3	0
10.	58	4	37	1	0

11.	57	3	38	2	0
12.	63	2	30	3	0
13.	59	3	36	2	0
14.	61	3	33	3	0
15.	62	3	34	1	0
16.	62	3	34	3	0
Mean ± SE	60.12 ± 0.4319	2.69 ± 0.1920	34.62 ± 0.5144	2.56 ± 0.2495	0
Range	57-63	1-4	32-38	1-4	0-0

**Appendix 11: Biochemical parameters in serum of healthy cattle (n=10)**

S.NO.	AST (U/lt)	ALT (U/lt)	ALKP (U/lt)	Lactate (mmol/lt)	LDH (U/lt)	Creatinine (mg/dl)	CK (U/lt)	BUN (mg/dl)	Glucose (mg/dl)
1.	78.86	22.60	80	0.60	698.00	0.50	36.65	14.36	65
2.	96.33	25.66	60	1.76	692.00	0.61	35.00	16.30	60
3.	80.94	27.32	140	1.23	876	0.78	56.23	16.50	45
4.	82.69	11.00	30	1.24	978	0.76	95.45	18.60	58
5.	95.47	33.42	45	1.79	1123	0.87	87.89	15.30	75
6.	100.22	18.52	62	1.56	1234	0.59	150.78	9.94	70
7.	88.67	26.36	85	0.89	1445	0.68	108.24	12.20	72
8.	78.00	21.48	110	1.00	1323	0.91	109.78	17.30	68
9.	91.78	40.00	93	2.20	967	1.10	280.00	8.90	66
10.	132.00	25.82	54	2.11	856	0.95	275.12	13.60	59
Mean±SE	92.49 ±4.7832	25.22 ±2.3679	76.50 ±9.4130	1.44 ±0.1598	1019.2 ±77.0168	0.77 ± 0.0557	123.51 ±26.5901	14.30 ±0.9515	63.80 ±2.6038
Range	78-132	11-40	30-140	0.6-2.2	692- 1445	0.50-1.10	35-280	6.0-27	45-75

**Appendix 12:**  
serum

**Biochemical  
parameters of  
in heatstroke**

**suffered cattle before treatment (n=16)**

S.No.	AST (U/lt)	ALT (U/lt)	ALKP (U/lt)	Lactate (mmol/lt)	LDH (U/lt)	Creatinine (mg/dl)	CK (U/lt)	BUN (mg/dl)	Glucose (mg/dl)
1.	387	65	146	10.39	1450	1.20	283	35	40
2.	377	53	112	3.78	1478	1.50	285	40	50
3.	354	65	86	5.26	1123	1.64	290	30	55
4.	290	96	98	5.61	1269	1.45	300	27	60
5.	305	83	124	7.95	1180	2.78	289	30	52
6.	287	88	108	4.30	1257	3.12	296	35	45
7.	416	71	86	5.50	1400	4.12	302	50	47
8.	346	75	74	7.40	1378	3.56	307	37	48
9.	450	123	94	4.50	1410	2.89	290	42	54
10.	406	79	106	12.0	1378	2.76	310	45	68
11.	330	120	84	11.13	1224	1.89	295	65	44
12.	345	60	110	6.87	1350	3.60	315	33	50
13.	350	130	92	5.89	1490	4.50	311	38	52
14.	380	56	80	8.96	1379	5.52	301	45	56
15.	375	90	84	9.67	1468	4.95	302	33	46
16.	358	108	114	7.56	1450	3.95	282	47	57
Mean ± SE	359.75± 10.7726	85.12 ± 5.9605	99.87± 4.5568	7.29± 0.6150	1355.25± 27.2066	3.08± 0.3199	297.37± 2.4951	39.5± 2.2998	51.50± 1.6677

Range	287-450	53-130	74-146	3.78-12	1123-1490	1.2-5.52	282-311	27-65	40-68
-------	---------	--------	--------	---------	-----------	----------	---------	-------	-------

**Appendix 13 Biochemical parameters of serum in heatstroke suffered cattle after treatment (n=16)**

S.No.	AST (U/lt)	ALT (U/lt)	ALKP (U/lt)	Lactate (mmol/lt)	LDH (U/lt)	Creatinine (mg/dl)	CK (U/lt)	BUN (mg/dl)	Glucose (mg/dl)
1.	134	35	128	3.30	1240	1.2	270	25	60

2.	170	46	90	3.98	1350	1.11	275	30	65
3.	135	39	72	3.80	1398	0.98	282	32	58
4.	136	32	84	3.90	1200	0.89	297	24	70
5.	140	40	83	2.50	1456	0.78	289	20	58
6.	125	33	103	2.90	1478	1.66	290	26	62
7.	210	26	66	3.23	1289	1.50	285	35	63
8.	146	60	64	3.50	1234	1.34	360	40	59
9.	140	34	90	2.90	1232	1.45	310	38	58
10.	126	29	104	2.89	1101	1.56	278	21	64
11.	128	38	80	3.50	1298	1.68	287	28	60
12.	138	42	103	3.56	1345	0.59	292	32	56
13.	110	45	92	1.78	1234	1.34	275	33	68
14.	130	36	65	3.00	1001	2.00	273	45	70
15.	131	43	70	2.67	1190	1.89	265	25	58
16.	129	41	62	2.50	1060	1.50	260	18	65
Mean ± SE	139 ± 5.4754	38.68 ± 1.9359	84.75 ± 17.8728	2.89 ± 0.1434	1256.62± 32.2229	1.34 ± 0.0957	286.75 ± 5.6218	29.5 ± 1.8307	62.12±1.0893
Range	110-210	26-60	62-128	1.78-3.98	1001-1478	0.59-2.00	260-360	20-45	56-70

**Appendix 14: Biochemical parameters of serum in Healthy cattle (n=10)**

S.No.	Na (mmol/l)	K (mmol/l)	Ca (mg/dl)	P (mg/dl)
1.	135.23	4.67	8.45	4.70
2.	134.00	4.00	8.97	4.86
3.	140.54	4.98	9.87	5.78
4.	143.34	5.67	9.78	6.87
5.	134.58	5.46	9.56	6.86
6.	140.89	5.36	10.23	7.45

7.	144.00	4.89	10.50	5.83
8.	138.26	4.78	9.76	5.23
9.	139.78	5.23	10.98	8.90
10.	136.98	5.70	8.20	6.98
Mean± SE	138.76 ± 1.0646	5.07± 0.1566	9.63 ± 0.2631	6.35 ± 0.3931
Range	132-152	3.9-5.8	9.7-12.4	5.6-6.5

**Appendix 15: Biochemical parameters of serum in heatstroke suffered cattle before treatment (n=16)**

S.No.	Na (mmol/l)	K (mmol/l)	Ca (mg/dl)	P (mg/dl)
1.	131.00	3.92	8.23	5.20
2.	131.73	3.88	8.45	5.50
3.	130.00	3.98	7.80	4.58
4.	129.78	3.98	7.90	4.23
5.	130.79	4.00	8.10	4.34
6.	131.67	4.55	7.90	4.00
7.	130.43	3.92	8.50	5.34
8.	129.87	4.14	8.98	4.99
9.	130.56	4.53	7.89	5.00
10.	128.7	4.45	7.56	5.45
11.	133.45	4.51	9.50	5.55
12.	129.35	4.64	9.30	5.23
13.	129.99	4.78	8.00	4.98
14.	130.67	4.89	8.78	5.12
15.	130.23	4.30	7.67	5.24

16.	131.23	4.12	8.12	4.67
Mean ± SE	130.59 ± 0.2682	4.28 ± 0.0813	8.29 ± 0.1403	4.96 ± 0.1140
Range	128.70-133.45	2.11-3.78	7.56-9.50	4-5.55

**Appendix 16: Biochemical parameters of serum in heatstroke suffered cattle after treatment (n=16)**

S.No.	Na mEq/lt	K mEq/lt	Ca mg/dl	P mg/dl
1.	135	4.98	10.21	5.67
2.	140	4.87	8.28	5.8
3.	145	5.20	9.89	5.89
4.	146	5.45	9.78	6.24
5.	152	5.23	9.67	6.12
6.	150	4.29	10.23	4.98
7.	130	4.12	9.23	5.78
8.	134	3.72	8.99	5.89
9.	128	3.45	9.27	6.22
10.	130	4.23	9.89	6.45
11.	129	4.12	10.56	5.90

12.	131	4.00	10.25	5.50
13.	125	4.90	10.23	5.12
14.	145	4.83	8.99	5.98
15.	146	4.24	9.00	5.99
16.	136	4.56	9.12	5.23
Mean ± SE	137.62 ± 2.0951	4.51 ± 0.1397	9.59 ± 0.1536	5.79 ± 0.0999
Range	125-152	3.45-5.45	8.28-10.56	4.98-6.45







