Clinical, haematological and biochemical findings in Saanen goat kids with naturally occurring heat stroke

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Key words
Caprine, heat stroke, hyperthermia, blood biochemical parameters

Summary
Objective: Description of clinical, biochemical and haematological changes in Saanen goat kids post-exposure to a naturally occurring heat stroke. Material and methods: The experimental group consisted of goats of different age (1–4 months) and sex (8 males, 12 females) with an average weight of 7.2 ± 3.1 kg. Twenty clinically healthy, three to four months old, Saanen goat kids (sex-ratio 1:1) were used as a control. The average body weight in this group was 9.4 ± 2.6 kg. Case history, clinical signs, and results of haematology and blood biochemistry were documented in all goat kids. Results: Most common findings were hyperthermia, ataxia, muscle tremor and depression. Increased serum urea, creatinine, potassium and plasma lactate concentrations as well as an increase in aspartate aminotransferase and lactate dehydrogenase activity were observed in goats post-exposure to heat stroke when compared to the control group. Two goat kids died despite supportive treatment. Physical and biochemical blood parameters improved following treatment. Conclusion: This is the first study on heat stroke in Saanen goat kids. Heatstroke may be fatal in Saanen goat kids, despite appropriate treatment, and may lead to secondary complications. Plasma lactate concentration seems to be a reliable indicator for the prognosis of heat stroke in goat kids.

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Klinische, hämatologische und biochemische Befunde bei Saanen-Ziegenlämmern nach natürlich auftretendem Hitzschlag
Tierärzt. Prax 2009; 37 (G): 236–241
Received: November 17, 2008
Accepted: March 31, 2009

Schlüsselwörter
Ziegenlämmer, Hitzschlag, Hyperthermie, biochemische Parameter

Zusammenfassung

Introduction
Heatstroke 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 (9, 12, 19, 22, 44). Heatstroke is more common in the summer and especially when the environmental temperature and relative humidity are high and with prolonged exposure to direct sunlight (22). While retrospective studies have sporadically reported about environmental heat stroke in veterinary medicine (19, 44), various studies in human medicine have described several clinicopathological and pathological abnormalities (22, 48).

The aims of this study were to describe the clinical and clinicopathological abnormalities as well as the outcome in 20 Saanen goat kids with naturally-occurring heat stroke-related illness and to assess their association with mortality.

Tierärztliche Praxis Großtiere 4/2009
Material and methods

Admission of animals

The study was conducted on 20 Saanen goat kids that were found to have been affected by different degrees of heat stroke. The kids were of different ages, from different sexes and weight (Table 1). The animals were assigned to the pre-treatment group (n = 20). The same kids were re-evaluated as the post-treatment group (n = 18). Twenty healthy Saanen goat kids of different sex (n = 20) and body weight were used as the control group selected randomly from among the Saanen goat kids living on a different farm. All of the affected kids met the clinical criteria of heat stroke which was classified as moderate (n = 11) and severe (n = 9) based on clinical signs such as hyperthermia (moderate: 41.4–41.8 °C; severe: 41.9–42.1 °C), tachycardia (moderate: 144–180 bpm, severe: 180–196 bpm), tachypnea (moderate: 52–75/min; severe: 75–84/min). Severe depression, muscle tremor, ataxia and hypersalivation were also taken as the criteria for severe heat stroke. On all animals an examination of ruminal fluid was performed.

The goat kids included in the study were accommodated at a special farm located in Bursa, northwestern Turkey in July 2007. The owner said that all of them had been kept in a sheep pen and had been forgotten outside for a long period during the day. In different compartments of the same pen 63 sheep, 31 goats, and 32 kids were kept. All animals were fed with alfalfa, as forage wheat bran, sunflower meal and soybean meal, as well as a commercial mixture for lambs as concentrate which consists of ground corn, barley, calcium carbonate, and vitamin premix. The kids in the control group were given the same forage-concentrate ratio. All goats had free access to water. The living environment of the study kids was classified as moderate (n = 11) and severe (n = 9) based on clinical signs such as hyperthermia (moderate: 41.4–41.8 °C; severe: 41.9–42.1 °C), tachycardia (moderate: 144–180 bpm, severe: 180–196 bpm), tachypnea (moderate: 52–75/min; severe: 75–84/min). Severe depression, muscle tremor, ataxia and hypersalivation were also taken as the criteria for severe heat stroke. On all animals an examination of ruminal fluid was performed.

Laboratory methods

Blood samples were obtained through jugular venipuncture in vacuum tubes with EDTA (2 ml) (Hema&Tube®, Turkey) as anticoagulant for haematology and lactate measurement and plain tubes (10 ml) (Hema&Tube®, Turkey) for serum biochemistry. Serum and plasma samples were separated by centrifugation (3000 rpm for 20 minutes at 20 °C). Samples with visible haemolysis were excluded from the study. Haematological parameters, including total white blood cell count (WBC count) and differential cell counts, haematocrit (HCT), haemoglobin (Hgb), red blood cell count (RBC), mean corpuscular haemoglobin concentration (MCHC), mean corpuscular volume (MCV) and platelet counts were measured within one hour after sampling using an automatic analyzer (Cell-Dyne 3500®, Abott Inc., USA).

Some serum biochemical analyses were measured within two hours using an autoanalyzer (RA-XT®, Bayer Inc, Germany). Parameters included sodium, potassium, calcium, phosphorus, and the activity of lactate dehydrogenase (LDH) (3 hours after sampling). Furthermore, the concentrations of serum urea, creatinine, the activities of aspartate aminotransferase (AST) and creatine phosphokinase (CK) (Roche Diagnostic, Germany) were assessed within one hour using a Reflotron® (Boehringer&Mannheim Inc., Germany; also delivered by Hoffman La Roche). Plasma lactate levels were measured within one hour with a lactate kit (Spinreac®, Spain) at 505 nm using a colorimetric spectrophotometer (Unicam 8625 UV/VIS®, Cambridge, UK) at 37 °C following the plasma extraction process. We adjusted the instrument to zero with distilled water. The working instructions, reagents and the samples were incubated for 5 minutes at 37 °C. Absorbance of the samples, standard solution and blank were read. The results were calculated according to the following formulae: samples/standard × 10 = lactate concentration (mg/dl) for conversion to mmol/L: (mg/dl) lactate × 0.111 (conversion factor)

Diagnosis

Diagnosis of heat stroke was finally made based on the clinical sings including severe depression, tachycardia, tachypnea, muscle tremor, incoordination, mild to moderate ataxia, hypersalivation, and laboratory findings. In differential diagnosis, floppy kids syndrome, nutritional muscular dystrophy, watery mouth were considered but some of the epidemiologic, clinical and laboratory findings such as normal body temperature for nutritional muscular dystrophy, abomasal dilatation, hypothermia, leukopenia, severe hypoglycemia for water mouth and abnormal ruminal fluid examination for ruminal acidosis ruled out these diseases.

Treatment

All affected animals were brought indoors in a cool environment to avoid high humidity and the adverse effects of sunlight. Ascorbic
acid (Redoxon®, Roche Inc.; 500 mg/animal, i. v., once daily for 3 days) and balanced isotonic saline solution (0.09%, Isotonic NaCl®, Dexter, Eczabasi Inc., Istanbul Turkey, 50–70 ml/kg BW, i. v.) were administered to all affected kids based on percent dehydration which had been estimated by clinical findings including CFT, peripheral pulse quality, decreased urine output, dry mucous membranes, sunken eyes, mental depression, mildly or moderately decreased skin turgor, and dry mucous membranes isotonic saline solution. Afterwards they were sprayed with a water hose until their body temperature reached 39.5 °C (40). Body temperature of the animals was closely monitored (35).

Statistical analysis

Data were expressed as mean ± SE and analyzed using variance analysis. Turkey tests were used to test the differences among groups. A difference value of p < 0.05 was considered significant. All statistical analyses were performed using the statistical package SPSS version 13.0 (SPSS Inc., Illinois, USA) (42).

Results

Values of rectal temperature, heart rate, and respiratory rate in affected animals (pre-treatment and post-treatment group) and control animals are shown in Table 2. The most consistent clinical signs observed in the pre-treatment group included hyperthermia, severe depression, tachycardia, tachypnea, muscle tremor, incoordination, mild to moderate ataxia, and hypersalivation. All of the affected goat kids displayed the above mentioned neurological signs. The temperature-humidity index was calculated as 35.72 (31). Two kids were found dead following supportive treatment. A pathological examination could not be performed due to rapid decomposition. The examination of ruminal fluid including pH, infusorians, sediment, flotation were normal in all animals.

Rectal temperature of the dying kids had been between 41.7 °C (range: 41.4–42.1) and 42.1 °C before death. Eighteen of the kids recovered in 3 days after supportive treatment. Laboratory data for pre-treatment, post-treatment, and control group are presented in Table 2. Statistical differences (p < 0.05) between the pre-treatment group and the control group were detected for the following parameters: rectal temperature, respiratory rate, heart rate, WBC, neutrophils, lymphocytes, monocytes, eosinophils, MCHC, AST, HCT, potassium, lactate, urea, creatinine and LDH. Plasma lactate levels of surviving goat kids ranged from 2.9 to 3.4 mmol/L. The two goat kids that died had higher plasma lactate levels (3.75 mmol/L and 3.81 mmol/L, respectively).

Discussion

Heat stroke is a medical emergency requiring rapid diagnosis and treatment. This potentially life-threatening condition occurs mostly when environmental temperatures are high and is characterized by hyperthermia, tachypnea, tachycardia and changes in mental status following heat exposure (9, 12, 19, 20). The various forms of heat related illness represent a spectrum from the mildest form of heat stress to heat exhaustion and finally to heat stroke. However, heat stress refers to the feeling of discomfort and sluggishness when one exercises in unusually hot conditions. By definition, the body core temperature is not elevated, and the only negative effect is that one’s performance may be very well below normal (26, 48). The clinical signs observed in our patients were consistent with the symptoms of heat stroke as reported by various authors for other species (9, 12, 19, 20, 26). Yet, as to our knowledge, there are no studies on the effects of heat stroke on Saanen goat kids. Hence, the authors are of the opinion that the present study might be significant in the field of veterinary medicine.

Cattle, sheep and goat can be severely affected by the thermal environment, especially in hot conditions. The responses of these animals to heat depend on many factors such as breed, season and age. Heat production and respiratory evaporative heat loss have been presented as functions of three variables, i.e. hypothalamus temperature, spinal cord temperature and air temperature (25). Physiological thermolytic pathways under heat stress incorporate an increase in respiratory and cutaneous evaporative cooling (28), an increase in peripheral to splanchnic blood flow ratio have been implicated in goats (40, 41) and the suppression of thermogenic hormone production (6). Low humidity and air movements are important to allow evaporation of sweat and convection of heat (2, 14, 15, 17, 25). Dehydration and increase of electrolyte concentration in the body fluid of mammals exposed to heat will reduce
their thermoregulatory evaporation and allow the body temperature to rise (41). This readjustment in thermoregulation has been observed in both panting and sweating species 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 goat, progressive dehydration leads to suppressed sweating and increased panting (8). In the present study, we found that all of the kids in the pre-treatment group had high respiration rates and heart rates. This finding is consistent with the thermoregulatory mechanism in goats as reported in previous studies (2, 25).

Muscle degenerations observed in any cases may be exacerbated because of deficiency of Vitamin E and selenium (5). In the present study, vitamin E and selenium levels could not be evaluated but we suggest that measurements of vitamin E and selenium levels may be valuable in evaluation of goat kids suffering from heat stroke due to negative effects of vitamin E and selenium deficiency on muscle.

Rectal temperatures varied between 38.9 °C and 40 °C under thermo neutral conditions in goats. The high rectal temperature (hyperthermia) is the main finding in the initial phase of heat stroke (34). In the present study, an air temperature of 41 °C was measured and the temperature humidity index was estimated to be 35.72 (31) which indicates severe heat stroke. We found that the rectal temperatures of the kids in the pre-treatment group were significantly higher than those of the control group. However, there was no statistical difference between the control group and the post-treatment group.

Table 3
Haematological and biochemical findings (mean ± SE) in animals with heat stroke and in control animals

<table>
<thead>
<tr>
<th></th>
<th>Animals with heat stroke</th>
<th>Control group (n = 20)</th>
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<tbody>
<tr>
<td></td>
<td>Pre-treatment group (n = 20)</td>
<td>Post-treatment group (n = 18)</td>
</tr>
<tr>
<td>WBC (×10⁹/L)</td>
<td>18.0 ± 1.6a</td>
<td>11.9 ± 1.1bc</td>
</tr>
<tr>
<td>Neutrophil (×10⁹/L)</td>
<td>15.8 ± 0.02a</td>
<td>6.05 ± 0.03bc</td>
</tr>
<tr>
<td>Lymphocyte (×10⁹/L)</td>
<td>1.9 ± 0.03a</td>
<td>5.6 ± 0.04bc</td>
</tr>
<tr>
<td>Monocyte (×10⁹/L)</td>
<td>0.036 ± 0.004a</td>
<td>0.02 ± 0.001ab</td>
</tr>
<tr>
<td>Eosinophil (×10⁹/L)</td>
<td>0.93 ± 0.006a</td>
<td>0.71 ± 0.004ab</td>
</tr>
<tr>
<td>RBC (10¹²/L)</td>
<td>12.6 ± 0.1</td>
<td>12.2 ± 0.2</td>
</tr>
<tr>
<td>Hgb (g/L)</td>
<td>97 ± 0.2</td>
<td>90 ± 0.3</td>
</tr>
<tr>
<td>HCT (l/l)</td>
<td>0.319 ± 0.4a</td>
<td>0.245 ± 0.9bc</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>19.2 ± 1.0</td>
<td>17.9 ± 0.2</td>
</tr>
<tr>
<td>MCHC (g/L)</td>
<td>345 ± 2.3a</td>
<td>339 ± 1.8ab</td>
</tr>
<tr>
<td>Platelet (×10¹²/L)</td>
<td>321 ± 10.1</td>
<td>343 ± 9.2</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>475.3 ± 21.3a</td>
<td>319.3 ± 10.9b</td>
</tr>
<tr>
<td>LDH (U/L)</td>
<td>615.8 ± 70.5a</td>
<td>429.5 ± 69.2b</td>
</tr>
<tr>
<td>CK (U/L)</td>
<td>288 ± 20a</td>
<td>123 ± 8.1bc</td>
</tr>
<tr>
<td>Creatinine (μmol/L)</td>
<td>167.96 ± 0.02a</td>
<td>114.92 ± 0.03b</td>
</tr>
<tr>
<td>Urea (mmol/L)</td>
<td>19.09 ± 4.6a</td>
<td>11.35 ± 2.3b</td>
</tr>
<tr>
<td>K⁺ (mmol/L)</td>
<td>7.2 ± 0.4a</td>
<td>6.3 ± 0.2ab</td>
</tr>
<tr>
<td>Ca (mmol/L)</td>
<td>2.2 ± 0.2</td>
<td>2.4 ± 0.2</td>
</tr>
<tr>
<td>P (mmol/L)</td>
<td>2.36 ± 0.7</td>
<td>2.6 ± 0.6</td>
</tr>
<tr>
<td>Na⁺ (mmol/L)</td>
<td>149.1 ± 3.9</td>
<td>146.3 ± 4.2</td>
</tr>
<tr>
<td>Lactate (mmol/L)</td>
<td>3.53 ± 0.35a</td>
<td>1.82 ± 0.07b</td>
</tr>
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</table>

a, b, c indicate a significant difference between pre-treatment group, post-treatment group and control group measurements (p < 0.05)

WBC = white blood cell, RBC = red blood cell count, Hgb = haemoglobin, HCT = haematocrit, MCV = mean corpuscular volume, MCHC = mean corpuscular haemoglobin concentration, AST = aspartate aminotransferase, LDH = lactate dehydrogenase, CK = creatine phosphokinase.

In previous studies in humans (3, 10, 18, 24, 29) rhythm disturbances including sinus tachycardia, atrial fibrillation, supraventricular tachycardia, and hypotension have been frequently described. Similarly, the kids in the pre-treatment group had higher heart rates when compared to the control group which suggests
that 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 (3, 24).

Patients with heat stroke present neurological symptoms of varying degrees and duration including ataxia, incoordination, lethargy, coma and seizures (3, 18). In the present study, all the affected kids had neurological signs including mild to moderate ataxia, incoordination, and muscular tremors. These neurological findings could be attributed to metabolic disarray, cerebral edema or ischemia (10). In eighteen of the kids, these neurological dysfunctions disappeared after supportive therapy. However, the neurological symptoms of non-surviving kids were more severe than in the others. They included seizure activity associated with whole body tremors, severe depression, and dilated pupilla. Despite supportive treatment, these two kids were found dead. Pathological examinations could not be performed due to rapid decomposition.

Moderate heat stroke resulted in mild to moderate inflammatory responses which were self-limited and subsided after 36 hours with recovery of the animals (12, 13). We also found that the pre-treatment group had higher WBC and neutrophil counts (p < 0.05) when compared to the control group, which may be explained by inflammation associated with heat stroke.

Bouchama et al. (13) observed that animals with severe heat stroke displayed an oliguric renal failure with an increase in serum urea and creatinine levels, hypercarbaemia, hyperchloraemia, and mild increase or decrease in blood glucose, sodium, and potassium levels. Similarly, we found a mild to moderate increase in serum urea, and creatinine levels compared with the control group (p < 0.05). These increases may be associated with renal dysfunction in accordance with those reported by Dematte et al. (18) or with prerenal azotemia that might develop due to shock. Also, the high HCT would support at least a degree of dehydration as a contributing factor to azotemia.

Some studies (4, 21, 27, 47) in humans reported increased activity of AST, LDH, and serum alkaline phosphatase in patients with heat stroke. These increases may be associated with liver damage due to direct thermal injury and hypoxia secondary to splenic re-distribution (21). In the present study, CK, AST and LDH levels in the pre-treatment group were higher than in the control group. However, these enzymes are not liver-specific. Unfortunately, we could not evaluate necessary enzymes for differential diagnosis including sorbitol dehydrogenase and glutamate dehydrogenase activity. Therefore, we concluded that the rise in the enzyme activities may be associated with muscle damage.

Heat stroke may lead to mild to severe changes in serum sodium, potassium, calcium and phosphorus levels (13). Hypokalaemia 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. However, animals in the pre-treatment group had high potassium levels (7.2 ± 0.4 mEq/L) compared with the control group. This hyperkalaemia might be associated with sustained hyperthermia, hypoxia and hypoperfusion which lead to the failure of the magnesium-dependant Na⁺/K⁺-ATPase pump resulting in cellular potassium leakage (22).

While some studies (11, 45) have reported that acute hypophosphataemia and hypercalcaemia are observed in classical heat stroke and are probably related to the increased glucose phosphorylation seen in alkalotic conditions (22) a few authors (32, 36) have noted hyperphosphataemia due to resultant rhabdomyolysis associated with sustained hyperthermia, hypoxia and hypoperfusion. 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 hypercalcaemia and hyperphosphataemia (22). In the present study, phosphorus and calcium levels of the pre-treatment group were not found to be statistically different when compared to the control group and post-treatment group. This result may be due to the varying degrees of heat stroke observed in each kid and as a result of pathophysiologic process described above.

Heat stroke occurs in two types, water and sodium depleted, although in reality they often overlap (22, 30, 33). In humans heat exhaustion from sodium depletion occurs most often in unacclimated people who fail to replace sodium lost in sweat (7). On the other hand, a person with water-depleted heat exhaustion is hyponatraemic (16). Marathon runners with exertional collapse were reported to be hyponatraemic, hypernatremic and normonatraemic in some studies (16, 23). Furthermore, despite the higher HCT value in the pre-treatment group when compared to the control group, no statistical difference was found between the sodium values of the pre-treatment group and the control group which might be associated with moderate dehydration in kids.

If heat stroke is associated with dehydration reduced renal perfusion and therefore reduced excretion of hydrogen ions can worsen the situation. Formation of L-lactate from anaerobic glycolysis following tissue hypoperfusion has long been considered to be a cause of high anion gap acidosis in kids with heat stroke. In the present study, high plasma L-lactate level was the most important biochemical change. In humans this change is well described in heat stroke (29). High lactate levels can occur as a response to severe exertion, and lactate is rapidly converted to glucose by the liver. In the present study the plasma lactate level was significantly higher in pre-treatment group when compared to the control group. A negative correlation between increased plasma lactate and decreased survival rate has been reported (22, 28). Similarly, the two kids in the present study which died had higher plasma lactate levels than the surviving animals. Therefore, plasma lactate level might be an important indicator for prognosis.

Our study shows that Saanen goat kids are sensitive to this disease. It also displays that plasma lactate concentration can be a useful predictor in evaluation of prognosis for goat kids suffering from heat stroke. The assumed associations offer interesting approaches for future investigations.
References