

Outbreak of Peracute Urea Toxicosis in Sheep Following the Consumption of New Packages of Grain Barley

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Abstract: The objective of this report was to describe peracute urea poisoning deaths in 84 Ile de France and Merinos breed sheep due to following the consumption of new packages of grain barley on July 30, 2014 in İvrindi, Balıkesir, Turkey. The owner of the sheep and a local veterinarian reported that in the farm there were in total 800 sheep, and 325 of animals were fed with new packages of grain barleys and 84 of which were observed dead after feeding in three-four hours. Clinical signs in all affected animals prior to death were uneasiness and ataxia, incoordination, grinding teeth, salivation, severe struggling and bellowing, abdominal bloat, lateral recumbence, convulsions, and deaths occurred in one and half hour after first clinical signs. The presences of white urea particles were intensive especially at the bottom of the packages of barley. Urea, crude protein, nitrate and nitrite analysis were performed in barley samples. Analysed barley samples had quite high urea concentration (8.76%) and crude protein (27.81%). Anamnesis, clinical signs and laboratory analysis indicated peracute urea poisoning in sheep.

Key Words: Urea, peracute toxicosis, sheep.

Yeni Parti Tane Arpanın Tüketilmesini Takiben Koyunlarda Ortaya Çıkan Perakut Üre Zehirlenmesi

Özet: Bu çalışmanın amacı, 30 Temmuz 2015 tarihinde, Balıkesir'in İvrindi ilçesinde, yeni parti tane arpanın verilmesini takiben, Ile de France ve Merinos ırkı toplam 84 koyunun öldüğü perakut üre zehirlenmesini tanımlamaktır. 800 koyunun bulunduğu bu işletmede, yemlemede yeni parti arpanın yaklaşık 325 koyuna verilmesini takiben üç dört saat içinde 84 koyunun öldüğü yerel veteriner hekim ve hayvan sahibi tarafından bildirildi. Etkilenen hayvanlarda tedirginlik, ataksi, koordinasyon bozukluğu, dış gıcırdatması, salivasyon, şiddetli çabalama ve bağırta, karında şişkinlik, yere yan yatma ve konvülzyonları takiben yaklaşık bir buçuk saat sonra ölümlerin görüldüğü belirtildi. Yapılan incelemelerde beyaz renkli üre partiküllerinin arpa taneleri arasında özellikle çuval dibinde yoğun olarak bulunduğu gözlemlendi. Arpa numunesinin üre, ham protein, nitrat ve nitrit analizleri yapıldı. Arpa numunesinin yüksek oranda üre (%8.76) ve ham protein (%27.81) içerdiği tespit edildi. Bu olguda elde edilen anamnez bilgileri, klinik belirtiler ve laboratuvar analiz sonuçları koyunlarda üre ile perakut zehirlenmeyi işaret etmektedir.

Anahtar Sözcükler: Üre, perakut zehirlenme, koyun.

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Introduction

Nonprotein nitrogen (NPN) compounds can be utilized by ruminants to provide their protein nitrogen requirements. NPN compounds such as urea, ammonium acetate and ammonium sulphate are commonly used in ruminant rations because they are often more economical than natural protein. Feed grade urea [$\text{CO}(\text{NH}_2)_2$] is the cheapest, most effective, and most available nitrogen source. However, peracute and acute toxicity may occur under conditions of misuse^{8,12}. Urea can be added to molasses-based liquid products or into solid mineral blocks. Ammoniated beet pulp, citrus pulp, straw, silage, molasses, and rice hulls have also been used to supplement the protein content of a ruminant's diet. Ruminants can also be poisoned by gaining access to some fertilizers that can contain over 40% urea¹².

All mammalian species are potentially susceptible to NPN toxicities via the consumption of ammonium-containing feeds, if dose is high enough. Sheep and other ruminants are the most susceptible because their rumen contain urease and is an ideal environment for hydrolysis of urea, releasing carbon dioxide and ammonia. Urea toxicity varies with the age of ruminant animals. The very young ruminant has very low susceptibility because its rumen flora has not developed^{5,8}. Some factors can potentially predispose ruminants to poisoning with these compounds that include lack of adaptation to high NPN diets, low energy diets, high rumen pH, elevations in body temperature, dehydration, stress, concurrent disease, alterations in rumen microflora, and hepatic insufficiency^{4,5,8,12}. When urea is consumed by adult ruminants, it is rapidly disintegrated to ammonia in the rumen by bacterial urease. The ammonia is then utilized by the rumen bacteria, along with soluble carbohydrates, to synthesize amino acids and proteins. Most of the excess ammonia produced is protonated to the ammonium (NH_4) ion and is trapped in the rumen. If this excess ammonia production continues, the rumen pH increases. Ammonia poisonings occur when significant amounts of ammonia get absorbed systemically to the bloodstream¹². Excess ammonia has been shown to inhibit the citric acid cycle^{6,12}, to cause a lactic acidosis, and to interfere with cerebral energy metabolism and the sodium-potassium ATPase pump. The systemic metabolic acidosis is thought to be related to a hyperkalemia, and this situation can ultimately

cause to cardiac arrest¹². On the other hand, the primary cause of death was due to respiratory arrest⁴.

The onset of clinical signs can be extremely quick and peracute, from ten minutes to as much as four hours after consumption of toxic level of urea and other NPN compounds^{5,8,12}. Commonly reported clinical signs in affected ruminant animals exhibit uneasiness and ataxia, incoordination, grinding teeth, salivation, muscle tremors, severe struggling and bellowing, labored breathing, kicking at the abdomen indicating abdominal pain, and bloat. The progression of clinical signs is generally quite rapid and lateral recumbency, convulsions and death typically ensue within four hours^{4,5,8,12}. Toward the terminal stages, vomiting is especially common in sheep. There are no characteristic lesions of urea-NPN poisoning⁸. Necropsy findings in urea poisonings include odour of ammonia can detect when open the rumen of animal which has recently died. Pulmonary oedema, congestion, and petechial haemorrhages are rather common findings^{5,8}.

Case History

The owner of the farm addressed to the Faculty of Veterinary Medicine of Uludag University on July 31, 2014 to report the deaths of sheep. In the farm, there were 800 sheep aging three-four years old. The farmer noticed the beginning of poisoning after administration of new packages of grain barleys to the sheep approximately at 19:00 pm in Ivrindi, Balıkesir, Turkey. According to farmer's commitments the first group animals composed from 230 Ile de France breed sheep were fed with barley and exhibited poisoning signs after a while. Upon the occurrence of clinical poisoning signs, the second group animals including 170 Merinos breed sheep were stopped to feeding barley. Clinical signs of toxicity were observed after 15-20 minutes of ingestion of barley. The local veterinarian and the owner reported the poisoning of a total of 84 sheep, of which, 70 were from first group 14 were from second group in three-four hours. The poisoned animals were died within two hours after the initiation of first clinical signs. Slightly affected 25-30 animals were survived after symptomatic treatment (intravenous infusion of saline solutions) by the veterinarian. Commonly observed clinical signs were uneasiness and ataxia, incoordination, grinding teeth, salivation, severe struggling and

bellowing, abdominal bloat, lateral recumbency and convulsions prior to death. In addition, vomiting was also shown in some sheep (Figure 1).



Figure 1. Vomiting in one of the death sheep.
Şekil 1. Ölen koyunların birinde görülen kusma.

Abdominal bloat and recumbency was also presented in Figure 2. Suspected barley samples were collected for examination and toxicological analysis. In examination, the presences of white urea particles were observed especially at the bottom of the packages of barley (Figure 3). Urea, crude protein, nitrate and nitrite analysis were performed in barley samples. Urea and crude protein levels of barley samples were determined by the method of Turkish Standards Institution¹³ and the method of AOAC¹, respectively. The mix barley sample was had high rate urea (8.76%) and crude protein (27.81%). Nitrate and nitrite levels were determined by spectrophotometric analysis¹⁰.



Figure 2. Abdominal bloat and recumbent position in death sheep.
Şekil 2. Ölen koyunlarda görülen karın şişliği ve yatma pozisyonu.



Figure 3. Ürea particles in the package of barley.

Şekil 3. Arpa çuvalı içindeki üre partikülleri.

Discussion

Toxic and lethal doses are difficult to establish in ruminants due to all the predisposing factors that can enhance or decrease their sensitivity to this compounds cycle¹². Urea is generally recommended in ruminant rations at a rate of approximately 3% of the grain ration or about 1% of the total ration. In the present case, the urea concentration of barley (as based on grain ration) (8.76%) was approximately three folds higher than reported levels in the veterinary literature. Urea has been reported to cause toxic effects when administered to ruminant animals at higher doses and depends on predisposing factors such as lack of adaptation to high NPN diets and low energy diets. Urea doses of 8.76% in grain ration has been described to cause lethal effects in adult sheep^{4,5,8}. In the case described here, urea was administered to sheep at higher amount with barley and this farm sheep were had been lack of adaptation to high NPN diets because the sheep were not fed urea previously. Therefore, the amount of urea administered in these animals should be responsible for the intoxication. Clinical signs in the sheep of the present report were similar to those reported previously literatures^{4,5,8,12} and from field cases of urea toxicosis and included uneasiness and ataxia, incoordination, grinding teeth, salivation, severe struggling and bellowing, abdominal bloat, lateral recumbency and convulsions prior to death. Vomiting which was observed in this case (Figure 1), did not harmonise with previously published report⁸. Clinical signs of sheep were similar. In urea-NPN toxicities, although there are some lesions such as pulmonary oedema, congestion, and petechial haemorrhages,

there are no characteristic lesions⁸. Post mortem examination (except abdominal bloat) and ammonia in blood, serum and ruminal fluid could not be carried out in this case due to timing problem and lack of approach of owner and the veterinarian to the poisonings. Determined nitrate and nitrite levels in barley sample were 20 mg/kg NO₃-N and 1.5 mg/kg NO₂-N, respectively. These levels are much lower than the levels (800-2000 NO₃-N) which lead to subacute and chronic toxicity in ruminants³.

The anamnesis information, clinical symptoms, demonstration of urea in grain barley packages and level of urea in consumed barley confirmed peracute urea poisoning as the cause of sheep deaths.

In field intoxications, the toxicological diagnosis is a path that must be taken especially when it allows to detect and to eliminate the intoxication source in order to save other animals and humans from being exposed to the same toxic substances^{9,11}. To the best of our knowledge, although there is a report² with urea poisoning of six lambs in sheep 84 of sheep were dead in this case in sheep in Turkey. The feed company supplying barley with urea should be made a mistake for this produced party. This sheep farm did not use urea to these sheep for protein source before this case; this situation may have been a contributing factor. Urea poisoning treatment is often not possible because of its rapidity. However, five percent acetic acid or vinegar should be orally infused into the affected animal; 0.5-1.0 litre in sheep and goats and 2-6 litres in cattle. This should be immediately followed by a large volume (15-30 litres) of cold water^{6,7,8,12}.

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