

## Oak leaf (*Quercus* spp.) intoxication in a sheep

### *Eikenblad- (Quercus spp.) intoxicatie bij een schaap*

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## ABSTRACT

A twenty-year-old ewe presented with acute lateral decubitus, tremor and dyspnea, followed by death. Post-mortem examination revealed a large amount of oak leaves in the rumen, reticulum and omasum. The duodenum had a focal hemorrhagic content and multifocal hemorrhages were present in several organs. Histopathological examination of the kidneys showed characteristic lesions of oak intoxication, such as intratubular hemorrhage, tubular degeneration and necrosis. This case report illustrates the importance to include oak intoxication in the differential diagnosis of acute death in sheep and the value of post-mortem and histopathological examination.

## SAMENVATTING

Een twintigjarige ooi vertoonde acute laterale decubitus, tremor en dyspnee, gevolgd door sterfte. Tijdens het post-mortemonderzoek werd een grote hoeveelheid eikenbladeren aangetoond in de pens, net- en boekmaag. Een deel van het duodenum bevatte een hemorrhagische inhoud en er waren multifocaal bloedingen aanwezig in verschillende organen. Bij het histopathologisch onderzoek van de nieren werden karakteristieke letsels van eikintoxicatie waargenomen, zoals intratubulaire bloedingen, tubulaire degeneratie en necrose. In deze casuïstiek wordt aangetoond dat eikintoxicatie dient ingesloten te worden in de differentiaaldiagnose van acute sterfte bij schapen en wordt de waarde van autopsie en aanvullend histologisch onderzoek benadrukt.

## INTRODUCTION

Oak (*Quercus* spp.) poisoning occurs sporadically in many species worldwide, although cattle seem to be the most affected species (Bausch and Carson, 1981). Intoxication is mainly observed in spring and autumn. All parts of the oak are potentially harmful as they contain tannins. When ingested, tannins can induce nephrotoxicity, gastroenteritis, hemolytic anemia and in high doses, hepatotoxicity (Segelmeier, 2017). The signs are usually subacute and appear several days after ingestion. In severe cases, death may occur within 24 hours after the onset of clinical signs (Pérez et al., 2011; Newman, 2012). In this case report, a rather atypical case of oak intoxication is described, as it caused acute death without any previous clinical signs in an elderly ewe during wintertime after ingestion of fallen oak leaves.

## CASE DESCRIPTION

### Case history

A female Zwartbles sheep of approximately twenty years old was presented in December to the local veterinarian with acute lateral decubitus, heavy tremor and dyspnea with froth coming out of its mouth. There was no previous history of illness. The ewe had an enlarged udder but this was considered normal by the owner. Symptoms were not observed in the three other sheep sharing the pasture. After ruling out an esophageal obstruction through intubation, the veterinarian administered dexamethasone (Rapidexon®, Dechra, the Netherlands) and hyoscine-N-butylbromide dipyrone (Buscopan®, Boehringer Ingelheim, UK). The ewe died within a few minutes after treatment.

## Necropsy and sampling

Due to suspicion of intoxication, the ewe was referred for necropsy one day later at the Laboratory of Veterinary Pathology, Faculty of Veterinary Medicine, Ghent University.

The ewe had a body condition score (BCS) of 4.5/5. Gross examination revealed soil and a large amount (estimated as >50% of the total content) of incomplete leaves in the rumen, reticulum and omasum, which were confirmed as oak leaves after botanical research at the Laboratory of Pharmacology and Toxicology of the Faculty of Veterinary Medicine, Ghent University (Figure 1). The abomasum contained no oak leaves, but a moderate amount of brown fluid content with a large amount of soil. The distal duodenum had a focal area with hemorrhagic content, multifocal serosal hemorrhages and a focal hemorrhage in the adjacent mesoduodenum (Figure 2). A swab for bacteriological examination was taken from the hemorrhagic content of the distal duodenum by a small sterile incision. Multifocal hemorrhages were also present in the epicardium, lungs, pleura and mesentery. The liver was pale with a slight orange appearance on cut surface. The lungs were congested, edematous and showed rib impressions. The enlarged udder was filled with a large amount of pus. The other organs did not show any gross abnormalities. Small tissue samples were taken from the liver, lung, kidneys, brain, ileum and the hemorrhagic part of the duodenum and were fixed in 4% buffered-formalin solution for further histological examination. Feces from the rectum were collected for parasitological examination.

Bacteriological examination of the hemorrhagic duodenal content revealed the presence of *Clostridium perfringens* amongst other clostridia. In the feces, nonpathogenic *Eimeria* spp. (OPG=100), *Capillaria* (EPG=200) and *Strongyloides* (EPG=1800) were detected.

## Histology

Histopathological examination of formalin-fixed and paraffin-embedded kidney samples demonstrated large areas of tubular epithelial degeneration and necrosis. Approximately 40% of cortical and 80% of medullar tubuli contained intraluminal erythrocytes and sloughed necrotic epithelial cells (Figure 3). Some tubules had intraluminal granular casts, and a mild amount of distal tubules showed mineralization of the tubular epithelium. Overall, the glomeruli were ischemic and multifocally, there were small cortico-medullar and medullar hemorrhages present. The duodenum showed multifocal hemorrhages in the serosae and adjacent mesoduodenum with mild, multifocal infiltration of neutrophils. The mucosa was autolytic, impeding further examination. In the liver, moderate numbers of neutrophils were multifocally dispersed in the sinusoids. The lungs were congested

with some small hemorrhages in the parenchyma. The brain showed no significant histopathological abnormalities.

## Differential diagnosis

Important differential diagnosis for acute unexpected death in sheep can be clostridial enterotoxemia, copper poisoning, bloat, severe abomasal parasitism, such as *Haemonchus contortus* and bacterial septicemia. (Maxie and Miller, 2016). Hemorrhagic enteritis and multifocal serosal hemorrhages are classically observed with *Clostridium perfringens* enterotoxemia (Uzal et al., 2016). The presence of *Clostridium perfringens*, amongst others clostridia, could support this diagnosis in the present case. However, tubular degeneration and intratubular hemorrhage are not common features in clostridial enterotoxemia.

*Clostridium perfringens* type D mainly causes acute death in sheep and so-called 'pulpy kidney' on gross examination. Pulpy kidney is best regarded as a poorly understood form of accelerated autolysis. Diagnosis of *Clostridium perfringens* type D enterotoxemia is based on demonstration of epsilon toxin of this bacterium in the gut and histopathological examination of the brain (Uzal et al., 2016). Gross and histopathological findings of the kidneys and brain rendered this etiology less likely, thus toxin detection was not performed.

Other nephrotoxic agents, which cause acute tubular injury are some types of drugs, heavy metals, mycotoxins and other plants such as high oxalate-containing vegetation (Cianciolo and Mohr, 2016). Most of these nephrotoxic agents were ruled out by the clinical history of the ewe. Sheep are predisposed to chronic copper intoxication due to their low ability of biliary copper excretion. Animals die from an acute toxic copper crisis when liver necrosis and hemolysis occur (Cullen and Stalker, 2016). This can result in hemoglobinuric nephrosis, which is not easily differentiated from renal tubular degeneration and intratubular hemorrhage. However, liver necrosis and copper accumulation were not observed histologically in the present case.

Tympanic distention of the forestomachs (bloat) occurs sporadically in sheep and is caused by esophageal obstruction, intake of highly fermentable feed, ruminal adhesions or peritonitis (Uzal et al., 2016). It can result in acute death and typical signs are enlarged gas-filled forestomachs as well as vascular congestion of head and neck, and esophageal bloatline at necropsy. All of these signs were absent in the present case.

Death by *Haemonchus contortus* abomasitis is related to anemia and hypoproteinemia (Uzal et al., 2016), which were unremarkable at necropsy. Bacterial septicemia was also excluded by the lack of gross and histological lesions.

In the present case, the most likely diagnosis was considered acute oak intoxication due to the large



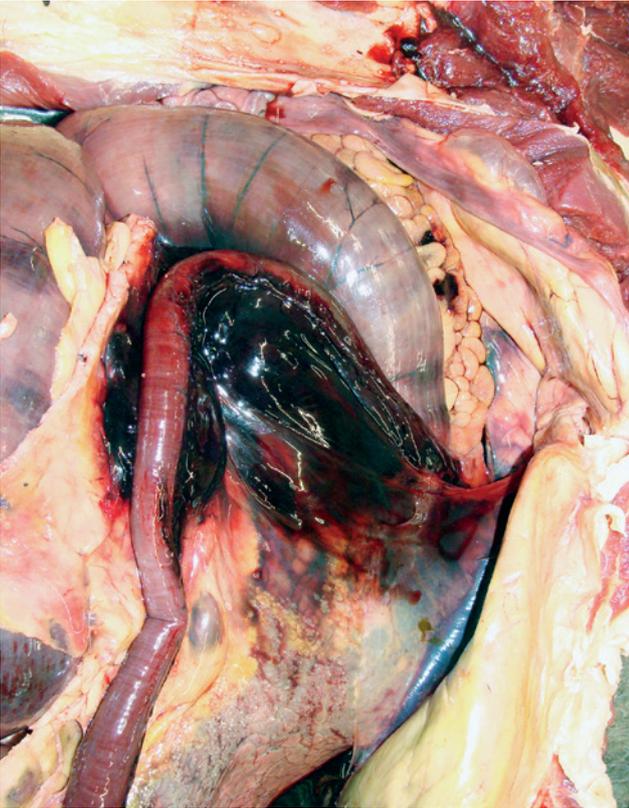
**Figure 1. Incomplete oak leaves found in the forestomachs of the ewe during necropsy.**

amount of oak leaves in the forestomachs, focal hemorrhagic duodenitis and extended renal tubular necrosis with intratubular hemorrhage.

## DISCUSSION

Oak (*Quercus* spp.) poisoning of animals occurs sporadically and worldwide. In springtime, intoxication occurs mostly due to ingestion of buds, young leaves and blossoms. In autumn, it is common due to consumption of acorns. All oak species, trees and shrubs, should be considered potentially toxic. Especially young leaves, buds and acorns have high levels of toxic components, which are hydrolysable tannins and their metabolites including tannic acid, gallic acid and pyrogallol (Baush and Carson, 1981; Sebastian et al., 2007). It is thought that tannins such as pyrogallol produce oxidative radicles that can denaturize proteins and alter protein interactions (Stegelmeier, 2017). In the present case, the owners of the ewe confirmed that there was an oak shrub present close to the pasture. Although this case happened in December and oak leaves have less amounts of tannins, the ingestion of large quantities of leaves, as found in the forestomachs at necropsy, is very likely the cause of intoxication.

An important predisposing factor, which contributes to intoxication is species. Cattle are the most common reported species affected by oak intoxication. Less frequently, intoxication occurs in horses and occasionally in sheep and goats. Swine seem to be rather resistant (Panter et al., 2007). Rabbits and guinea pigs are also susceptible (Bausch and Carson, 1981). A possible explanation why cattle are more affected could be their gastric anatomy, which provides them to uptake a large amount of oak material in one feed. Another predisposing factor is the concentration of toxins present in the leaves. Oak toxicity is considered dose-dependent. Intoxication often occurs during periods of drought when there is limited foliage and thus when food resources are scarce. When animals are fed an amount of oak material less than 50% of their total food intake, it will usually prevent poisoning (Panter et al., 2007). In the present case, the oak leaves were considered to be more than 50% of the content in the rumen, reticulum and omasum. In an experimental study of Pérez et al. (2011), it was shown that intoxication of cattle was only elicited when administration of oak leaves was preceded by a severe feed restriction period. The reason why feed restriction favors intoxication still remains unclear. As the ewe in the present case had a BCS of 4.5/5, it



**Figure 2.** Focal hemorrhage in the mesoduodenum at necropsy.

is unlikely that there was a long period of food restriction preceding the intake, but a short period cannot be ruled out. Other factors, such as season, maturation of the oak and geographic location, also play an important role, as these factors may affect the amount of toxic components the oak contains. Additionally, animal factors such as young age can contribute to a higher risk for intoxication. Some animals also seem to develop a taste for acorns and oak leaves, so they consistently search and consume them. Furthermore, there seems to be individual differences in susceptibility between animals of the same age and species (Bausch and Carson, 1981). Taken together, the large amount of ingested oak leaves by the ewe of the present case has resulted in intoxication and death. Since leaves were not present in the intestinal tract, it is likely that the leaves were ingested in a short period. However, it remains unknown whether the other three sheep on the pasture also consumed oak leaves and why only one sheep showed clinical signs as described.

In the present case, signs were only observed shortly before death and were interpreted as agonal signs. The onset of initial signs is nonspecific and can be easily missed by the owner or herdsman (Bausch and Carson, 1981). Clinical signs are mainly caused by renal and gastrointestinal damage and can include anorexia, polyuria and polydipsia, hemorrhagic diarrhea, and also ventral edema may be present. Signs

usually occur subacute and appear several days after ingestion (Blowey and Waever, 2011). In autumn, fragments of acorn may be present in the feces. In occasional severe cases such as the present case, death may occur within 24 hours. Rarely, chronic cases may occur (Bausch and Carson, 1981). Ante-mortem blood examination in oak intoxication can reveal abnormalities associated with renal disease, such as elevated creatinine, blood urea nitrogen (BUN) and potassium. Oxidative damage to erythrocytes can also cause hemolytic anemia (Davis and Warren, 2017). Urine examination usually shows low gravity and proteinuria (Pérez et al., 2011). The treatment for oak intoxication is symptomatic and preventing further access to oak plants. Extra awareness is needed after violent storms, as leaves and acorns may end up on the pasture of animals (Blowey and Waever, 2011).

Diagnosis of oak intoxication is usually based on a history of access to oak plants or on necropsy findings (Blowey and Waever, 2011). Detection of pyrogallol in blood or tissue has limited diagnostic value as it is rapidly metabolized (Stegelmeier, 2017). In the present case, the diagnosis was concluded post-mortem based on a history of access to a nearby oak shrub, typical necropsy findings, such as the large amount of oak leaves found in the stomachs, the presence of multifocal hemorrhages and hemorrhagic content of the duodenum and the histopathological findings such as extensive renal proximal tubular necrosis with intratubular hemorrhage. Especially the presence of tubules with intratubular hemorrhage distinguishes this poisoning from that of most other causes. Also the observed extensive tubular degeneration and necrosis, ischemic glomeruli and intraluminal sloughed epithelial cells and casts are considered typical (Cianciolo and Mohr, 2016). Although microscopically, the kidneys were severely affected, macroscopic changes such as pallor and enlargement with occasional cortical petechial hemorrhages and perirenal edema, which have been described in oak intoxication (Cianciolo and Mohr, 2016), were not observed. As endothelial cells are a main target for binding of the oak tannins and their metabolites, vascular leakage may be present (Newman, 2012). In this case, hemorrhages were present in several organs, such as the gastrointestinal tract, lungs and heart, which are typical locations. Also the presence of the focal hemorrhagic duodenitis fits the diagnosis of oak poisoning. Gastro-intestinal ulcerations were not observed in this case but are also common, which can be secondary to uremia due to renal failure, but direct irritant effect of oak leaves cannot be ruled out (Pérez et al., 2011).

In the present case, *Clostridium perfringens* was cultured from the focal hemorrhagic duodenal content. The diagnosis of clostridial enterotoxemia is not straightforward and is based on the demonstration of large numbers of bacteria and enterotoxines as well as gross and histopathological lesions of hemorrhagic necrotizing enteritis (Uzal et al., 2016). Confirmation

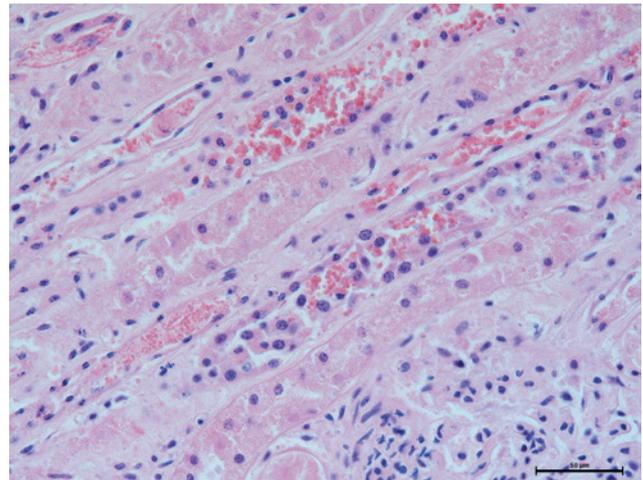
of the hemorrhagic necrotizing enteritis by histology was not conclusive due to post-mortem autolysis of the intestinal mucosa. As *C. perfringens* belongs to the intestinal flora, sudden changes in the intestinal environment can result in proliferation and toxin production (Uzal and Songer, 2008). In the present case, such change could be related to the abnormal gastric content. On the other hand, clostridia tend to overgrow other intestinal bacteria rapidly after death. Since also other clostridia species were isolated at necropsy and sampling was done one day after death, the presence of *C. perfringens* was regarded as a post-mortem overgrowth.

## CONCLUSION

Acute death in sheep can occur due to ingestion of oak leaves. Post-mortem findings such as the presence of a large amount of oak leaves in the stomachs can be indicative. Confirmation can be obtained by gross and by histopathological examination demonstrating multiple hemorrhages and renal tubular necrosis with luminal hemorrhage.

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**Figure 3. Multiple tubuli with desquamated epithelial cells and intraluminal intact and fragmented erythrocytes. Histological image of the renal cortex, hematoxylin and eosin staining, 400x.**

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