Case Report - Chronic oak toxicity (Quercus suber) in beef cattle in the south of Portugal: 17 cases (2014-2018)

C. Frias,1 DVM; P. B. A. Simões,1 DVM, MVM, MRCVS; J. Cota,1 DVM, PhD; H. Pissarra,1 DVM, MD, MSc; T. P. Nunes,1 DVM; C. A. Hjerpe,2 DVM; M. S. Lima,1 DVM, PhD, Dipl. ECBHM
1 CIISA – Centre for Interdisciplinary Research in Animal Health, Faculty of Veterinary Medicine, University of Lisbon, Portugal
2 School of Veterinary Medicine, University of California, Davis, Davis, CA 95616
Corresponding author: Dr. Patricia Simões, patriciasimoes@fmv.ulisboa.pt

Abstract

Oak toxicity in cattle results from ingestion of acorns, buds, leaves, sprouts, and saplings from several species of trees in the genus Quercus, all of which contain high concentrations of tannins. Consumption of acorns by ruminants, principally between late summer and early winter, is common in the south of Portugal, where there is a high prevalence of Quercus suber. Oak poisoning can lead to progressive damage to the kidneys and may result in renal failure and death. This study involved 17 beef cows that died of chronic oak poisoning related to ingestion of Quercus suber. The most relevant findings from necropsy examinations were abdominal fluid accumulation, atrophic/fibrotic kidneys, mesenteric and sub-mandibular edema, absence of body fat reserves and muscle atrophy, and weight loss. The most significant histopathologic finding was chronic interstitial nephritis. Elevated blood urea nitrogen, creatinine, and K+ values and reduced albumin values were found in 4 cows examined ante-mortem. Straight-bred Mertolengo cows appeared to be highly resistant to oak toxicity.

Key words: beef cattle, Quercus suber, chronic oak toxicity, chronic interstitial nephritis

Résumé

La toxicité du chêne chez les bovins résulte de l’ingestion de glands, de bourgeois, de feuilles, de germes et de gaules de plusieurs espèces d’arbres du genre Quercus, qui contiennent tous de fortes concentrations de tanins. La consommation de glands par les ruminants, principalement entre la fin de l’été et le début de l’hiver, est courante dans le sud du Portugal, où la prévalence du Quercus suber est élevée. L’empoisonnement au chêne peut entraîner des lésions progressives des reins et entraîner une insuffisance rénale et la mort. Cette étude a porté sur 17 vaches de boucherie mortes d’un empoisonnement chronique au chêne lié à l’ingestion de Quercus suber. Les résultats les plus pertinents des examens d’autopsie étaient l’accumulation de liquide abdominal, les reins atrophiques / fibrotiques, l’œdème mésentérique et sous-mandibulaire, l’absence de réserves de graisse corporelle et d’atrophie musculaire et la perte de poids. La découverte histopathologique la plus importante était une néphrite interstitielle chronique. Des valeurs élevées d’azote uréique sanguin, de créatinine et de K+ et des valeurs réduites d’albumine ont été trouvées chez 4 vaches examinées ante mortem. Les vaches de race pure Mertolengo semblaient très résistantes à la toxicité du chêne.

Introduction

Oak toxicity is well documented in several parts of the world, including the UK, USA, South Africa, India, Spain, and Portugal. In the south of Portugal oak trees (Quercus suber and Quercus ilex) are common in most beef cattle pastures. Oak poisoning occurs sporadically in some years, and not in others. In southern Portugal, this toxicity tends to occur whenever there is a large crop of acorns that fall from trees in pastures, namely during late summer and fall, which provides an opportunity for cattle to consume excessive amounts of acorns. Outbreaks of acute acorn poisoning in cattle are well documented in the literature. The onset of clinical signs and death can occur from 1 day to 3 to 4 weeks following an acorn drop.

Chronic acorn toxicity has not been characterized as well as acute poisoning. To our knowledge, chronic oak poisoning has only been reported 7 times previously, and the clinical aspects of those cases were not always thoroughly characterized and documented. Chronic oak toxicity is a protracted debilitating illness caused by decompensating renal function over a period of weeks. The most likely principal toxic agent in Quercus spp is tannic acid or other tanins, which become hydrolyzed in
the rumen to gallic acid, pyrogallol, and other compounds. Evidence infers, but does not firmly establish, that tannic acid or its derivatives are the principal toxic agents for oak poisoning in cattle. The level of toxicity of oak tannins appears to be variable, and poisoning can occur regardless of the plant part consumed. It has been suggested that young leaves are more toxic than mature leaves. The kidney is clearly the most susceptible organ to tannic acid effects in domestic ruminants, especially cattle. Tannic acid toxicosis causes renal disease and subsequent kidney failure.

At necropsy examination of acute cases, the kidneys are slightly swollen and pale with petechiae on the surface that extend into the cortex. In the gut, there is congestion and mucosal erosions of varying severity and location, extending from the mouth to the colon, and deep ulcerations and hemorrhages are sometimes evident. Although renal lesions are always present, lesions in other tissues may or may not be present. The objective of this case study was to describe multiple cases of chronic oak poisoning in beef cattle on a farm in the south of Portugal. This is the first report in the literature describing chronic oak poisoning in beef cattle due to ingestion of tannins present in Quercus suber, the tree referred to as 'The cork oak'. This species of tree is indigenous to the Mediterranean region, and is the source of much of the world's natural cork supply.

**Materials and Methods**

This case study took place on a 12,500 acre (5,000 ha) farm located in Alcácer do Sal, 50 miles (80 km) south of Lisbon, Portugal. There were approximately 1,000 head of cattle, 600 of which were adult cows, on the farm. The cows were pure Mertolengo (a Portuguese beef breed; 300 cows) and crossbred Mertolengo with Blonde d’Aquitaine and/or with Charolais and/or with Limousin breed influence (300 cows). The other 400 animals were yearling heifers and weaned calves. The cows were divided into 5 groups, with approximately 120 cows per group. There were also 10 Limousin bulls and 2 Mertolengo bulls in the herd.

Each group of cows rotated between different pastures (6 to 7 pastures per group). Each parcel of pasture was approximately 250 acres (100 ha) in size. Some pastures had natural grasses (6,200 acres; 2,500 ha) and forages and others were cultivated grasses and legumes (1,235 acres; 500 ha). The amount of available forage was similar for all 5 groups of cattle, as well as the exposure to oak. During the winters of 2016 and 2017, the cows were supplemented with corn silage (4.4 lb [2 kg]/d) and straw (2.2 lb [1 kg]/d) because of drought. The dominant trees in the pastures were oaks (Quercus suber) and pines (Pinus pinela).

Cowseal acorns after they drop from the trees (September to January) and can browse young trees, buds, sprouts, and leaves, which are available all year long. The quantity of acorns available and/or consumed varies from year to year, depending on weather and unknown factors. Cattle are not known to consume any parts of pine trees. The cows were vaccinated against bovine viral diarrhea virus and Clostridium perfringens type B, C, and D, chauvoei, hemolyticum, novyi, septicum, and tetani twice a year (May and November). The cows were dewormed with ivermectin in May, and combination ivermectin and closantel in November, beginning at 1 year of age.

This investigation began in November 2014 and extended through November 2018, and involved 17 adult cows. Fourteen of the cows were found dead in the pastures, and the remaining 3 were very sick and were euthanized because of the severity of clinical signs. A blood sample was collected from the caudal vein of the 3 cows that were euthanized, and from 1 cow that had just died (#485); creatinine, albumin, blood urea nitrogen (BUN), and potassium concentrations were measured. Ocular fluid (aqueous or vitreous fluid) was collected from 1 cow (#117), which had been dead for about 1 hour, to determine creatinine, albumin, and BUN levels.

Field necropsies were performed as soon as possible after the cows were found dead, and thin slices of kidney, liver, and large intestine were fixed in 10% formalin and sent to the pathology laboratory of the "Faculdade de Medicina Veterinaria, Lisboa" for histopathologic examination. These tissues were selected because acute oak toxicity in cattle is characterized by renal and gastrointestinal alterations, which can be confirmed by necropsy and histopathologic examination.

The mortality rate, from all causes, among the 600 adult cows on this farm during the study period (2014 – 2018) was 8.8% per year, totaling 264 cows that died of all causes. This death rate is well above the average (<2%) for herds raised under similar conditions in this part of Portugal. The mortality rate in the calves was 3%/year, which was less than most other beef cattle herds in Portugal, as well as in most well managed beef herds in the other western countries of the world.

**Statistical Analysis**

In order to test the association between the mortality rate and the genetics of the cows in the herd (straightbred Mertolengo vs crossbred Mertolengo), a Fisher exact test was performed.

**Results**

Cow data, serum chemistry values, gross necropsy findings, and major histopathologic findings are shown in Tables 1 to 4 and Figures 1 and 2.

All 17 cows that died were cross-bred Mertolengo cattle, with up to as much as 50% parentage from the Blonde d’Aquitaine and/or Charolais and/or Limousin breeds. There
was a highly significant difference in mortality rates between straight-bred Mertolengo cows (zero) and the crossbred cows (17 or 5.7%); *P*<0.0001.

### Discussion

In the south of Portugal, beef cattle often have access to large amounts of acorns that fall from trees like *Quercus suber* or *Quercus ilex* from late summer to early winter. The amount of acorns available can vary from year to year. During the entire year, the cows frequently have the opportunity to graze young oaks and foliage of mature oak trees. Bausch and Carson\(^1\) classified oak poisoning in cattle according to the season. Oak bud and oak leaf poisoning are usually seen in the spring, while acorn poisoning is seen in the autumn after the acorn fall drop.\(^1\)\(^,\)\(^16\) At low levels of intake, oak is an important forage, but as tannin levels increase in cattle diets, roughage digestibility may decrease.\(^18\)

As mentioned, the mortality rate on this farm during the period of the study (2014-2018) was much higher than the average for this part of the country. There are 2 major reasons for this. First, cows were underfed, especially during certain years due to drought. In some months of the year, when the pasture is very poor (June until November), there is no grass available, and supplemental feed is not provided. Secondly, these cows have access to acorns, buds, leaves, sprouts, and saplings throughout the year. On this farm, unlike others, the presence of young trees in the fields is scarce, which strongly suggests that the cows use them as a source of feed.

According to Neser et al.\(^14\), the toxicity of acorns has been poorly understood, since they may be completely harmless on some occasions, but highly toxic at other times. Feed intake restriction, as occurs in situations of overstocking, is one of the most important risk factors for the development of toxicosis.\(^8\)\(^,\)\(^17\) Other predisposing factors include sudden large acorn “drops” caused by severe windstorms and heavy rain, which shake tree branches and cause large quantities of acorns to fall over a short period of time.\(^1\) However, there is no available evidence to suggest that any of these particular predisposing circumstances occurred on this farm prior or during the time of this case study.

The initial clinical signs of acute oak poisoning in cattle include gauntness, listlessness, and constipation followed by diarrhea, excessive thirst, frequent urination (urine may be clear and colorless or, in some early cases, red-colored because of polyphenolic metabolites), and feces may be bloody or black and tarry.\(^2\)\(^,\)\(^12\) These clinical signs are a consequence of gastrointestinal and kidney lesions which can be confirmed by necropsy and histopathologic examination of tissues. However, because beef cows are on the pasture and not frequently checked or closely examined, the onset of initial clinical signs may not always be observed by herdsmen.\(^1\) Unfortunately, oak toxicosis is very difficult to reproduce experimentally for reasons not well understood.\(^17\) In this case study, the clinical signs most consistently observed were weight loss and submandibular edema (Figure 3). Cows were usually noted to be dull, although according to the herdsmen an aggressive behavior was sometimes observed.

Elevated blood urea nitrogen and creatinine support a diagnosis of oak toxicity, and they may be extremely elevated.\(^22\) However, 75% loss of glomerular function is necessary to impact BUN and creatinine concentrations.\(^22\) Consequently,

### Table 1. Cow identification, year of death, age, and breed for cows that died of oak toxicity.

<table>
<thead>
<tr>
<th>Cow ID (year of death)</th>
<th>Age (years)</th>
<th>Breed</th>
</tr>
</thead>
<tbody>
<tr>
<td>280 (November 2014)</td>
<td>4</td>
<td>Crossbred</td>
</tr>
<tr>
<td>F115 (December 2014)</td>
<td>9</td>
<td>Crossbred</td>
</tr>
<tr>
<td>F114 (January 2015)</td>
<td>9</td>
<td>Crossbred</td>
</tr>
<tr>
<td>265 (January 2015)</td>
<td>5</td>
<td>Crossbred</td>
</tr>
<tr>
<td>123 (April 2015; euthanized)</td>
<td>14</td>
<td>Crossbred</td>
</tr>
<tr>
<td>485 (June 2015)</td>
<td>4</td>
<td>Crossbred</td>
</tr>
<tr>
<td>281 (November 2015)</td>
<td>6</td>
<td>Crossbred</td>
</tr>
<tr>
<td>185 (December 2015)</td>
<td>6</td>
<td>Crossbred</td>
</tr>
<tr>
<td>6929 (January 2016)</td>
<td>4</td>
<td>Crossbred</td>
</tr>
<tr>
<td>239 (April 2016)</td>
<td>7</td>
<td>Crossbred</td>
</tr>
<tr>
<td>277 (April 2016)</td>
<td>7</td>
<td>Crossbred</td>
</tr>
<tr>
<td>206 (January 2017)</td>
<td>12</td>
<td>Crossbred</td>
</tr>
<tr>
<td>204 (March 2018)</td>
<td>14</td>
<td>Crossbred</td>
</tr>
<tr>
<td>2174 (April 2018; euthanized)</td>
<td>5</td>
<td>Crossbred</td>
</tr>
<tr>
<td>050 (June 2018; euthanized)</td>
<td>5</td>
<td>Crossbred</td>
</tr>
<tr>
<td>117 (November 2018)</td>
<td>11</td>
<td>Crossbred</td>
</tr>
<tr>
<td>191 (November 2018)</td>
<td>10</td>
<td>Crossbred</td>
</tr>
</tbody>
</table>

### Table 2. Serum chemistry values from cows with oak toxicity that were euthanized (n=3, ID 123, 2174, 050), died just before necropsy (n=1, ID 485), and ocular fluid from cow that died just before necropsy (n=1, ID 117).

<table>
<thead>
<tr>
<th>Cow ID</th>
<th>BUN (mg / dL)</th>
<th>Creatinine (mg / dL)</th>
<th>Albumin (g / dL)</th>
<th>Potassium (mmol / L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>123</td>
<td>140</td>
<td>6.6</td>
<td>2.1</td>
<td>6.6</td>
</tr>
<tr>
<td>485</td>
<td>627</td>
<td>23</td>
<td>2.1</td>
<td>nq</td>
</tr>
<tr>
<td>2174</td>
<td>59</td>
<td>1.9</td>
<td>1.8</td>
<td>4.3</td>
</tr>
<tr>
<td>050</td>
<td>130</td>
<td>5.4</td>
<td>2.4</td>
<td>8.1</td>
</tr>
<tr>
<td>117*</td>
<td>130</td>
<td>4.4</td>
<td>nq</td>
<td>nq</td>
</tr>
<tr>
<td>Reference range(^*)</td>
<td>10 - 25</td>
<td>0.5 - 2</td>
<td>2.5 - 3.8</td>
<td>3.6 - 4.9</td>
</tr>
</tbody>
</table>

\(nq\) = not quantified
\(^*\) Values from cow #117 were from ocular fluid
it may be difficult to evaluate the significance of renal lesions found in individual dead cows during necropsy examination. In uncomplicated acute tubular injuries, regeneration of epithelial cells generally begins after about 7 to 10 days following renal damage. In mild cases, full recovery of architecture may occur within 2 to 3 weeks, with longer recovery periods being required for more severe renal injury. According to Dixon et al\textsuperscript{15} that show that compensatory weight gain can occur in steers after an outbreak of oak bud toxicosis, the cows of this study were never weighed, so this phenomenon, if it occurred, was not detected. Five dead cows had acorns in the rumen; 2 of these cows died in November, 1 in December, 1 in January, and 1 in March. Ten of these cows (60\% of the cows) died while they had plentiful access to acorns, but little grass. However, the other 7 cows (40\%) died when there was an abundance of grass available (March to May).

One of the difficulties facing a clinician during episodes of chronic oak poisoning is trying to predict the outcome in apparently unaffected animals. Cows on this farm had contact with acorns in different stages of maturity (from September to January), and young trees, leaves, buds, and stems during the rest of the year, but most of them never showed clinical signs. It is not clear why cows in spring or early summer died from oak poisoning when there was ample grass available. Three cows (\#281, \#185, \#191) died in late autumn/early winter without finding acorns in their rumen. It is possible that these cows had a foraging preference for leaves from young oak trees, which are available all year around. It would

Table 3. Necropsy findings from cows suspected with oak toxicity.

<table>
<thead>
<tr>
<th>Cow</th>
<th>Fluid accumulation</th>
<th>Gl tract lesions</th>
<th>Kidney</th>
<th>Mesenteric edema</th>
<th>Submandibular edema</th>
<th>Acorns in rumen</th>
<th>Weight loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>280</td>
<td>5 L (abdomen)</td>
<td>Small ulcers</td>
<td>Autolysis</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(abomasum)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F115</td>
<td>No</td>
<td>Hemorrhagic</td>
<td>Autolysis</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>jejunitis,</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>blood</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F114</td>
<td>Yes – nq (pericardium)</td>
<td>Edema of the abomasum folds</td>
<td>Autolysis</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>265</td>
<td>No</td>
<td>No</td>
<td>Autolysis</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>123</td>
<td>No</td>
<td>Edema of the abomasum folds</td>
<td>Autolysis</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>485</td>
<td>Yes - nq (abdomen)</td>
<td>Edema of the abomasum folds</td>
<td>Autolysis</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>281</td>
<td>No</td>
<td>No</td>
<td>Autolysis</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>185</td>
<td>No</td>
<td>Hemorrhagic</td>
<td>Autolysis</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>6929</td>
<td>No</td>
<td>Jejunitis</td>
<td>Autolysis</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>239</td>
<td>10 L (abdomen)</td>
<td>Edema of the abomasum folds</td>
<td>Autolysis</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>277</td>
<td>No</td>
<td>Hemorrhagic</td>
<td>Autolysis</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>206</td>
<td>No</td>
<td>No</td>
<td>Autolysis</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>204</td>
<td>No</td>
<td>No</td>
<td>Autolysis</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2174</td>
<td>20 L (abdomen)</td>
<td>Edema of the abomasum folds</td>
<td>Fibrosis</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>050</td>
<td>2 L (abdomen)</td>
<td>Edema of the abomasum folds</td>
<td>Fibrosis</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>117*</td>
<td>5 L (abdomen)</td>
<td>Edema of the abomasum folds</td>
<td>Fibrosis</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>191</td>
<td>No</td>
<td>No</td>
<td>Fibrosis</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

nq = not quantified
* Values from cow #117 were from ocular fluid
be very costly to prove this theory, as it would require hiring someone to monitor cows every day for the better part of a year.

Three cows (#F115, #185, #277) died suddenly without showing signs of sickness. Necropsy of cow #F115 revealed large intraluminal blood clots in the small intestine; lesions resembled hemorrhagic bowel syndrome which has been associated with Clostridium perfringens type A. Cows #185 and #277 showed marked hemorrhagic enteritis lesions which could be associated with overgrowth of Clostridium spp. Some of the cows in this study showed rapid autolysis (Table 3), which can also be associated with the proliferation of Clostridium perfringens within the small intestine. These cows, however, were vaccinated twice each year for Clostridium perfringens type B, C, and D. In spite of these findings, it is significant that the 3 cows that died suddenly had histopathologic evidence of chronic renal lesions, suggestive of acorn poisoning (Table 4).

One cow (#281) had oxalate crystals in the kidney (renal tubules). There were at least 4 plants on the farm that can accumulate potentially toxic amounts of oxalates (Amaranthus spp, Chenopodium spp, Rumex spp, and Oxalis pes-caprae). The populations of Amaranthus spp and Chenopodium spp on the farm were low, and considered to be an insignificant risk. However, at the beginning of the winter after the first fall rains, the cows did have access to significant populations of Rumex spp and Oxalis pes-caprae. The kidney from this cow, in addition to oxalate crystals, had lesions characteristic of oak poisoning (chronic interstitial nephritis).

Gross lesions at necropsy were not uniformly present in all affected cows (Table 3). Eight cows showed submucosal edema (Figure 3). Twelve cows showed marked edema of the abomasal folds, 6 had mesenteric edema, 6 had variable volumes of excess fluid in the peritoneal cavity, 5 had acorns inside the rumen, 6 had marked weight loss without any fat inside the abdominal cavity, 3 showed lesions of enteritis, 1 had abomasal ulcers, and 1 had excess pericardial fluid. Because necropsies of many of these cows were not performed on the day of death, there was usually some autolysis present in the internal organs.

Visual examination of the internal organs in the majority of the necropsies did not reveal any major gross pathologic changes. Nevertheless, in some cases, liver, lung, or intestinal samples were taken for histopathological examination when these tissues were reasonably free of severe autolysis. In 4 of the cows necropsied, the carcass had a strongly uremic odor (#280, #239, #2174, #117). Whether or not a cow with renal failure survives is dependent on the severity of

<table>
<thead>
<tr>
<th>Cow</th>
<th>Kidney</th>
<th>Liver</th>
<th>Other organs</th>
</tr>
</thead>
<tbody>
<tr>
<td>280</td>
<td>Interstitial nephritis; glomerular degeneration.</td>
<td>nd</td>
<td>Moderate abomasitis</td>
</tr>
<tr>
<td>F115</td>
<td>Fibrocystic interstitial nephritis</td>
<td>Multifocal necrotic hepatitis caused by bacteria (microabscesses)</td>
<td>nd</td>
</tr>
<tr>
<td>F114</td>
<td>Chronic interstitial nephritis</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>265</td>
<td>Chronic interstitial nephritis</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>123</td>
<td>Chronic interstitial nephritis; glomerular amyloidosis.</td>
<td>Periportal necrosis</td>
<td>Interstitial pneumonia</td>
</tr>
<tr>
<td>485</td>
<td>Chronic glomerulonephritis; interstitial fibrosis.</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>281</td>
<td>Chronic interstitial nephritis; deposition of oxalate crystals.</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>185</td>
<td>Chronic interstitial nephritis.</td>
<td>Microabscesses</td>
<td>nd</td>
</tr>
<tr>
<td>6929</td>
<td>Interstitial fibrosis.</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>239</td>
<td>Chronic interstitial nephritis; parenchymal calcification.</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>277</td>
<td>Chronic interstitial nephritis</td>
<td>Focal necrosis</td>
<td>nd</td>
</tr>
<tr>
<td>206</td>
<td>Chronic interstitial glomerulonephritis</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>204</td>
<td>Interstitial fibrosis; multifocal tubular calcification</td>
<td>Necrosis</td>
<td>nd</td>
</tr>
<tr>
<td>2174</td>
<td>Interstitial fibrosis; multifocal tubular necrosis; glomerular atrophy.</td>
<td>Congestion and focal necrosis</td>
<td>nd</td>
</tr>
<tr>
<td>050</td>
<td>Chronic interstitial nephritis</td>
<td>Sinusoidal congestion</td>
<td>Necrotic enteritis (cecum)</td>
</tr>
<tr>
<td>117</td>
<td>Chronic interstitial nephritis.</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td>191</td>
<td>Chronic interstitial nephritis; tubular necrosis; tubular calcification.</td>
<td>nd</td>
<td>nd</td>
</tr>
</tbody>
</table>

nd = not determined
the renal lesions, and the ability of the renal tubular cells to regenerate.\textsuperscript{19} Interestingly, the histopathological examination of tissues revealed remarkably consistent changes that were confined almost exclusively to the kidneys (Table 4). These changes were compatible with chronic renal tissue injury, in particular the amount of fibrotic interstitial tissue, glomerular damage, mononuclear inflammatory cell infiltration, and in some cases, calcification of renal tubules and parenchyma (Figure 2).

Previous reports described a syndrome that affects calves born from dams that ingested large numbers of acorns under poor forage conditions during the second trimester of pregnancy. These calves, referred as "acorn calves", have very short leg bones, may have abnormal hoof development, and may have a short or long, narrow head.\textsuperscript{12,20} There are no reports of this problem on the farm in this case study.

An important observation from this study was that only crossbred cows were affected, in spite of the fact that the Mertolengo cows made up approximately 50\% of the cows in the herd. In Spain, there are also reports suggesting that some indigenous breeds, including the black Avilena, the Morucha, and the Retinta, do not appear to be susceptible to oak poisoning.\textsuperscript{8}

According to Smith,\textsuperscript{20} young cattle (under 440 lb [200 kg]) are often more severely affected than adult cattle. This was not confirmed in our study, since the youngest cow dying was 4-years of age. One explanation for this is that young cattle are often moved to good quality pasture when they are weaned, and not grazed on marginal pastures where they are exposed to oak trees and acorns. Portuguese farmers recognize the increased requirements for maintenance and growth in weanling calves, thus they are provided better quality forages.

Removing animals from oak-infested areas or reducing stocking rates to allow greater forage availability during oak budding and early leaf growth is the most reliable method for preventing oak toxicosis.\textsuperscript{2} Removal of cattle from pastures when the acorn drop is abundant can be adequate therapy for mildly affected animals.\textsuperscript{1} Supplemental feeding may be required during at-risk periods on ranches where oak poisoning is a problem.\textsuperscript{19}

Several treatments have been shown to be effective for treating cows with acute acorn poisoning.\textsuperscript{2,12} Oral administration of 5 to 10\% calcium hydroxide solutions may have beneficial effects by neutralizing the tannins in the rumen. Active charcoal polyvinylpyrrolidone and polyethylene glycol (PEG, 10g/day) have also been reported to be protective against dietary tannins.\textsuperscript{7} However, we envision 2 problems

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image1}
\caption{Severe fibrosis of a kidney from a cow with chronic oak toxicity, presenting granular irregular surface and abnormal greyish coloration.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image2}
\caption{Histological alterations in the kidney (H&E staining; 40x). Exuberant interstitial fibrosis, tubular necrosis (†), glomerular atrophy (arrow), formation of cylindrical hyaline casts (circle) and renal tubule dilation (†). Multifocal mononuclear inflammatory cells infiltration (*). Aspects associated with interstitial glomerulonephritis with tubular necrosis, compatible with chronic oak toxicity.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image3}
\caption{Cow with suspected oak toxicity showing weight loss and a marked submandibular edema.}
\end{figure}
with implementing these therapeutic measures. First, we doubt that the kidney lesions observed in many cows in the present study would be reversible. Second, the long distance between many of the pastures and the corrals where working facilities were located made treatment attempts impractical. An alternative option is to feed pelleted rations containing 10 to 15% calcium hydroxide to the cows as a preventive measure, as long as the pellets are palatable.\textsuperscript{2,12} The strategy adopted on this farm was to identify the cows that were losing weight and had early signs of submandibular edema, remove them from the pasture, and feed them a mixed grain/hay ration, and eventually market them for slaughter after they have improved body condition scores.

Chronic oak toxicity has not been reported in Portuguese dairy cattle. There are relatively few dairy cattle on pasture in southern Portugal, and those that are on pasture are fed diets comprised of various grain and hay mixes, therefore little to no exposure to oak buds, leaves or acorns.

**Conclusions**

Chronic oak poisoning of beef cattle in Portugal occurs primarily in cows that are at least 2 years of age and nursing a calf, and is associated with ingestion of the acorns, buds, leaves and/or stems of oak trees of the species *Quercus suber*, and less commonly *Q. ilex*. On this farm, crossbred Portuguese Mertolengo cattle did not appear to be susceptible to this toxicity, presumably the result of their unique genetics. All 17 cases in this study occurred in crossbred Mertolengo cattle that were less than 50% Mertolengo. The other (>50%) genetic influence was mostly Blonde d’Aquitaine, Charolais, or Limousin.

Clinical diagnosis can be challenging, and requires a combination of indepth knowledge of on-farm feeding and animal management, physical examination of affected cows, as well as biochemical analysis of serum samples from affected animals, and gross and histopathologic examinations of kidney tissues from fatal cases. The main risk factors appear to be inadequate nutrition and poor pasture management that forces lactating cows to eat plants (i.e., *Q. suber* and *Q. ilex*) that they would not ordinarily want to graze or browse, in order to meet their dietary nutritional requirements.

**Endnotes**

\textsuperscript{a} Bovilis® BVD, MSD Animal Health, Lda., 2770-192 Paço de Arcos/Portugal
\textsuperscript{b} Covexin 8® , Zoetis Portugal, Lda., 2740-271 Porto Salvo/Portugal
\textsuperscript{c} Virbamec®, Virbac Portugal Laboratórios, Lda., 2710-693 Sintra/Portugal
\textsuperscript{d} Closamectin®, Norbrook Laboratories, Ltd, BT35 6QX Newry/Northern Ireland
\textsuperscript{e} Rx Daytona, Randox Laboratories Limited, Crumlin, UK
\textsuperscript{f} i-Stat, Sensor Devices Incorporated, Waukesha, WI, USA

**Acknowledgements**

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The authors declare no conflict of interest.

**References**

Moraxella bovoculi Bacterin

World’s First Commercially Available Moraxella bovoculi Pinkeye Preventative!

- 8 Different M. bovoculi isolates
- Cost Effective for all Cattle Herds
- Proven Safety Record
- More Convenient and Autogenous Programs


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**From the LEADERS in pinkeye prevention!**

Addison Biological Laboratory, Inc. announces the approval of the world’s first commercial Moraxella bovoculi vaccine for the prevention of pinkeye in cattle. The USDA conditionally licensed product is the first of its kind. Previously, the only method of prevention against Moraxella bovoculi was autogenous vaccines. This vaccine signifies a breakthrough in prevention for the large number of veterinarians and herd owners battling the challenging problem of pinkeye caused by Moraxella bovoculi. This product license is conditional, effective and potency have not been fully demonstrated.

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**Cystorelin (gonadorelin)**

By Merial

For treatment of cystic ovaries in dairy cattle.

For use with Cooperstandin® in a pituitary degradative estrus cycle to allow for timed artificial insemination (TAI) in lactating dairy cows (30 days postpartum to 12 months postpartum).

**CAUTION:** CooperSyringe® L-1 (labeled to be used with this product) is not available in the U.S. and is currently only licensed in Canada.

**DESCRIPTION:**

Cystorelin is a sterile solution containing 40 mcg/mL of gonadorelin (as gonadorelin acetate diacetate) equivalent to 40 mcg/mL of gonadorelin diacetate tetrahydrate (30 mcg/mL of gonadorelin). There is no preservative added to this product.

**INDICATIONS:**

- Cystic Ovaries
- Luteal Cysts
- Pyometra or Chronic Endometritis

**DOSAGE AND ADMINISTRATION:**

- Cysts Ovaries

  1. Administer 100 mcg (as gonadorelin diacetate tetrahydrate) intramuscularly (IM) into a large muscle mass (abductor muscle) of the lateral thorax or gluteal muscle of the hind leg.

  2. Administer 50 mcg (as gonadorelin diacetate tetrahydrate) IM 5 days after the cyst resolution occurred.

**WARNINGS AND PRECAUTIONS:**

- Dark field in animals.
- Keep out of reach of children.

**ADVERSE REACTIONS:**

No withdrawal period or withdrawal time is required when used according to the labeling.

**USE OF SYMCHSURE:**

- The intravenous or intramuscular dosage of CYSTORELIN is 100 mcg of gonadorelin diacetate tetrahydrate (2 mL) per cow.

**PRODUCT INFORMATION:**

Cystorelin is not intended for use in human medical treatment.

**REFERENCES:**

- 91
- 17
- 5-oxoPro-His-Trp-Ser-Tyr-Gly-Leu-Arg-Pro-Gly-NH2—
- 13
- 75
- 7.47 mg
- 17
- O
- q.s.
- 13

**Cystorelin®**

Equivalent to 250 mcg cloprostenol/mL

**Cystorelin®**

Equivalent to 500 mcg cloprostenol/mL

**SYNCHSURE®**

(dopamine releasing)

For treatment of cystic ovaries in dairy cattle.

For use with Cooperstandin® in a pituitary degradative estrus cycle to allow for timed artificial insemination (TAI) in lactating dairy cows (30 days postpartum to 12 months postpartum).

**CAUTION:** CooperSyringe® L-1 (labeled to be used with this product) is not available in the U.S. and is currently only licensed in Canada.

**DESCRIPTION:**

SYNCHSURE is a sterile solution containing 1 MCU/mL of dopamine releasing (44 mcg/mL) equivalent to 1 MCU/mL of dopamine releasing (33 mcg/mL) equivalent to 500 mcg of dopaminergic releasing (44 mg/mL) equivalent to 500 mcg of dopaminergic releasing (33 mg/mL) of dopamine releasing. There is no preservative added to this product.

**INDICATIONS:**

- Cystic Ovaries
- Luteal Cysts

**DOSAGE AND ADMINISTRATION:**

- Cysts Ovaries

  1. Administer 2 mL (100 mcg) of SYNCHSURE® intramuscularly (IM) or intravenously (IV) into a large muscle mass (abductor muscle) of the lateral thorax or gluteal muscle of the hind leg.

  2. Administer the second SYNCHSURE® injection 5 days after the cyst resolution occurred.

**WARNINGS AND PRECAUTIONS:**

- Dark field in animals.
- Keep out of reach of children.

**ADVERSE REACTIONS:**

No withdrawal period or withdrawal time is required when used according to the labeling.

**USES OF SYMCHSURE:**

- Controlled Breeding: SYNCHSURE can be used to synchronize estrus and eliminate the possibility of post-injection bacterial infection. Antibiotic therapy should be given prophylactically to reduce the risk of the infection. Number of estrous cycles usually indicated.

**CONVERSION CHARTING:**

- For comparison of SYNCHSURE to other existing in the market.

**SAFETY AND TOXICITY:**

- At 50 and 100 times the recommended dosage, SYNCHSURE was not found to be toxic in the rat, rabbit, or monkey. Further studies are needed to assess whether or not SYNCHSURE is toxic at the maximum recommended dosage. SYNCHSURE was demonstrated to be non-carcinogenic and non-mutagenic in the rat bioassay.

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**PRECAUTIONS:**

- Systemic absorption should be employed to decrease the possibility of post-injection bacterial infection. Antibiotic therapy should be given prophylactically to reduce the risk of infection. For technical assistance, avoid injection site reactions. The use of alternating sites in the same bovine is not recommended. For technical assistance in Japan, please contact Merial Toyama K.K. at 1-81-11-473-2880 or by email, synch@merial.co.jp. For technical assistance in Korea, please contact Merial Korea Ltd. at 82-31-223-4100 or by email, korea@merial.co.jp.
It’s easy to be confident that your cows will get pregnant when you use Cystorelin® (gonadorelin) and Synchsure™ (cloprostenol sodium) together. They’re an effective combination for reproductive efficiency. So, after use, this test is more of a formality.

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IMPORTANT SAFETY INFORMATION FOR CYSTORELIN: Do not use in humans. Keep this and all drugs out of the reach of children.

IMPORTANT SAFETY INFORMATION FOR SYNCHSURE: FOR ANIMAL USE ONLY, NOT FOR HUMAN USE. KEEP OUT OF REACH OF CHILDREN. Women of child-bearing age, asthmatics, and persons with bronchial and other respiratory problems should exercise extreme caution when handling this product. In the early stages women may be unaware of their pregnancies. SYNCHSURE is readily absorbed through the skin and may cause abortion and/or bronchospasms: direct contact with the skin should therefore be avoided. Accidental spillage on the skin should be washed off immediately with soap and water.

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