Oak poisoning has been recognized for over 300 hundred years. It occurs sporadically in many parts of the world. In the United States, most cases of oak poisoning are seen in the Southwest, Northeast, and Midwest. The leaves, buds, twigs, and acorns of many oak species are toxic, and all of them produce similar clinical signs and lesions when consumed in large enough quantities. Cattle are most often involved clinically, but sheep, horses, goats, rabbits, and quinea pigs are also susceptible.

Source

Oaks are woody perennial plants belonging to the genus *Quercus*. They grow in varying habitat, which allows them to be found in nearly all areas of the United States and Canada. The oaks range in size from shrubs to large trees. They have simple, alternate leaves with irregularly rounded lobes and produce an acorn, which is a matured, inferior ovary subtented by a cup of many reduced, overlapping scale leaves. Maturation characteristics of the acorn are used to divide oaks into two groups: white oaks and black oaks, both of which are toxic. Over 60 species of *Quercus* are known, all of which are potentially toxic. Most of the poisonings are from *Quercus gambelii* (Gambel's oak) and *quercus havardii* (shinnery oak).

Toxic Principles and Mechanism of Action

Tannins and their metabolites, gallic acid, and pyrogallol are now widely believed to be the major toxic principles of oak. Experimental feeding of tannins to rabbits and calves has produced clinical signs and lesions similar to those found in animals naturally poisoned with oak. Steers fed 50 grams of tannins daily for 14 days showed typical signs of oak poisoning, and the LD50 for tannin toxicity in rabbits is 6.9 gm/kg per day for 5 days.

Tannins are found throughout the oak plant, with higher levels found in the leaves, buds, twigs, and acorns. Acorns contain about 6–8 percent tannins. The tannins and their metabolites are absorbed from the G.I. tract and enter the blood. The exact mechanism of action is not known, but tannins are thought to be hepatotoxic and nephrotoxic by precipitation of proteins.

Predisposing Factors

Because oak poisoning occurs sporadically, appearing unexpectedly in animals that grazed the same pasture for many years without any adverse affects, there seem to be a number of related factors that must exist in order for oak poisoning to occur.

1. Season—Oak bud and oak leaf poisoning is usually seen in the spring, while acorn poisoning is seen in the autumn shortly after the acorns fall.
2. Geographic locations—Most of the acorn poisonings occur in the Midwest and Northeast in the autumn. However, leaf and bud poisonings are seen in the Southwest when cattle and sheep browse oak in the spring. Most oakbud poisonings occur on rugged heavily wooded pasture, where marginal grazing conditions exist.
3. Maturation of the oak—Young leaves and green acorns are more toxic than dry, mature leaves and mature acorns. This is probably due to the fact that
the tannin content decreases as the leaves mature, and also young leaves are considerably more palatable than mature leaves.

4. Weather conditions—Drought, or other circumstances leading to poor grazing conditions frequently exist at the time of oak bud poisonings. Acorn poisonings can be seen during years of heavy rainfall resulting in a heavy crop of acorns that soften and sprout, or when a windstorm brings the green acorns down prematurely.

5. Animal factors—Young animals, especially calves, are usually most affected even though cows and calves have access to the same pastures. Some animals seem to develop a taste for acorns and oak leaves and continually crave them, while other animals will not consume any. There also seems to be a difference in susceptibility between animals. Some animals can eat large quantities of acorns and show no ill effects, while others die after eating relatively small amounts. Animals on a poor plane of nutrition also are more susceptible to oak poisoning than well fed animals.

Clinical Signs

The major clinical signs are chiefly alimentary and urinary in nature. In most cases, the onset of initial signs are gradual and may not be observed by the herdsman. They include anorexia, depression, clear watery nasal discharge which may be blood stained, dry and crusted nose, and rumen atony. The body temperature is usually normal or subnormal. The rumen may become distended, and there may be an increase in pulse and respiratory rates. Elevation of tail head and lumbar spine with animals producing small hard balls of mucus-covered feces are frequent features. Constipation is followed in 2–10 days by a dark, thin, mucoid, often bloody diarrhea. Fragments of acorns may be present in the feces. Dehydration usually occurs and scleral vessels are markedly congested. Often there is subcutaneous edema of the neck, brisket, abdomen and perineum. The abdomen is distended due to ascites and a rapid shallow breathing due to hydrothorax are occasionally seen. Abortions have been observed following oak consumption.

As the disease progresses, animals show signs of polyuria and polydipsia and the urine becomes clear, with the urine specific gravity decreasing to the 1.008–1.014 range. Hemoglobinuria and a mild to moderate proteinuria with granular, cellular, and hyaline casts have been reported. Occasionally a mild glycosuria and occult blood are seen.

Cases of acute, subacute, and chronic oak poison have been reported. The acute cases are usually calves, which have been eating oak buds or acorns and die with a hemorrhagic enteritis and diarrhea after an illness of less than 24 hours. Most typical cases are subacute with animals showing signs in 3–10 days after consuming oak. Chronic disease results in a debilitating illness due to decompensated renal failure over a period of weeks. There have been reports of animals losing weight gradually for 19 months after being exposed and then dying of renal failure.

Morbidity rates vary from 1–2 animals in the herd showing signs, to the entire herd being affected. Mortality rates as high as 75–85 percent have been reported in cattle clinically affected with oak poisonings.

Clinical Pathology

Marked elevation of blood urea nitrogen (45–320 mg/dL) is seen in almost 100 percent of cases. Other abnormalities associated with the renal insufficiency caused by oak poisoning are: increased creatinine, hyperphosphatemia (7.0–20.3 mEq/L), hypocalcemia (3.5–4.2 mEq/L), hyponatremia, hyperkalemia, and a hypoalbuminemia along with increases in SGOT, SGPT, PCV, and hemoglobin.

Gross Pathology

Gross post-mortem findings are mainly what would be expected from the symptoms and can be put into four categories: (1) gastroenteritis, (2) hemorrhages, (3) edema and ascites, and (4) renal lesions.

The gastritis and enteritis range from catarrhal to hemorrhagic and usually involve the terminal intestine most severely. In advanced cases, 2–3 cm ulcers maybe seen on the mucosal surface of the G.I. tract. These ulcers appear to be caused by coagulative necrosis. Later, a diphtheritic pseudomembrane forms over the necrotic surface. The esophagus and pharynx may also show varying degrees of ulceration. An inconsistent fin-
 ding is the presence of acorns and/or oak leaves in the rumen or gut contents.

The hemorrhages are mostly of petechial and ecchymotic dimensions. They are constantly found on the epicardial and endocardial surfaces, serosal surfaces of G.I. tract, peritoneal and subperitoneal tissues, perirenal tissues, lungs, and lymph nodes. The liver is occasionally pale and mottled and shows a moderate degree of toxic hepatitis.

Subcutaneous edema is frequently found over the pelvic limbs and along the ventral body wall. The mesenteric lymph nodes are often swollen and edematous. Probably the most striking gross lesion is gelatinous blood-tinged perirenal edemas usually found in acute cases. In the more subacute or chronic cases, streaks of hemorrhage or brown hemosiderin deposits may be seen in the perirenal region, reflecting resolved edema. Hydrothorax, hydropericardium, and ascites are very dramatic findings and can be seen in about one half of the cases. The edema and blood-tinged fluid in the body cavities can be explained as a result of the hypoalbuminemia.

In acute cases, the kidneys are pale and swollen with a smooth surface. Petechial hemorrhages are seen on the surface and scattered throughout the cortex. The cut surface is moist, the cortex pale, and the medulla is usually congested. In more chronic cases, the kidneys become pale, shrunken, and irregularly dimpled, due to tubular degeneration and atrophy.

Histopathology

The constant microscopic change seen in the kidneys is a massive coagulative necrosis of the proximal convoluted tubules. There is sloughing of the tubule lining into the lumen to form a pink-staining mass of material composed of epithelial cells and protein. The distal tubules are often dilated and contain hyaline, granular, or cellular casts. In the acute stages, the interstitial tissue can be edematous and contain focal hemorrhages. Later, the cortical interstitium has an increase in fibrous connective tissue and contains small aggregates of lymphocytes and plasma cells. The glomeruli show little change and the medulla remains normal except for congestion. These microscopic lesions seen in the kidney are very characteristic of oak poisoning and considered by some to be pathognomonic.

Diagnosis

Clinical signs of gradual anorexia, rumen atony, constipation, followed by diarrhea and signs of renal disease in cattle exposed to oak buds or acorns could be considered a potential oak poisoning. By finding increases in SGOT and BUN, and by grossly seeing the typical gastrointestinal and renal lesions found in oak poisoning, a tentative diagnosis could be reinforced. The diagnosis could be confirmed by finding the characteristic coagulative necrosis of the proximal tubules microscopically, or by finding oak leaves or acorns in the G.I. tract.

Oak poisoning may be confused with *Amaranthus retroflexus* (pigweed) toxicosis, which produces similar clinical signs and similar gross and microscopic lesions. These two diseases could be differentiated by determining if the animals were exposed to oak or pigweed.

Treatment and Prevention

There is no specific antidote for oak poisoning, so treatment is largely symptomatic. Mineral oil and rumen stimulants along with parenteral fluids to correct dehydration and electrolyte imbalances are usually indicated. The toxic material could be removed by performing a rumenotomy to prevent further absorption of toxins.

The best prevention is to keep animals from being exposed by removing the source of oakbud or acorns, or by removing the animals from the source. If this is not possible, animals could be fed a supplemental ration containing 10–15% calcium hydroxide (hydrated lime) during periods of extreme hazard in an attempt to inactivate ingested tannins.

Case Report

On October 30, 1979, we were called to necropsy a four month old, 350 pound Hereford bull calf that had died the night before. The owner had noticed the day before that the calf was very stiff and stood with its legs tucked under its body and its back arched. The calf was also very reluctant to nurse.

On postmortem exam, a blood-tinged fluid was found in the abdominal and thoracic cavities and the lungs were collapsed. The kidneys were pale and swollen, and had
numerous subcapsular petechial hemor­rhages. A large amount of edema was found in the fat and connective tissue surrounding the kidneys. The mucosa of the abomasum was hyperemic and the small intestine showed varying degrees of hemorrhagic enteritis. Parts of acorns and acorn shells were found in the rumen, omasum, and abomasum.

The calf along with 20 other cows and calves were being pastured on a timber pasture that contained numerous oak trees, which had just produced a heavy crop of acorns. A diagnosis of oak poisoning was made on the gross lesions and the history of the animal being exposed to acorns. It was recommended to the owner that the cattle be removed from the pasture to prevent further exposure, but this was not feasible at this time. No other animals showed any clinical signs or died.

REFERENCES
Smith, HA. The Diagnoses of Oak Poisoning in a Cow and a Sheep. Southwestern Vet, 13, 1959, p. 34.