Acorn Poisoning in Cattle

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In the last ten days of October, 1973, in the Hanover clinic for cattle diseases, among the other patients admitted were six ½ to 1½-year-old calves and an eight-year-old pregnant cow presenting symptoms which had not been seen in previous years. The course of the disease lasted two to twelve days. The cow, because of the progressively worsening course of the disease, had to be slaughtered before laboratory research could be undertaken; on the basis of the autopsy findings and the subsequently gathered information from the owner, it could be assumed that it suffered from the identical disease. This will be set forth in the following synopsis.

Clinical Appearances and Laboratory Findings

Including the six other animals (none related), five came directly from the pasture, and two, because of their illness, had been placed in stalls a few days before being brought to the clinic. At this time the preliminary reports stated that all appeared ill from a few days to two weeks; the most important symptoms noted were lack of appetite (four times), diarrhea (two times), colic or emaciation (one time each). Examination showed pronounced apathy, a fixed stance with lowered head, strongly arched back, and tightly stretched abdominal muscles, moderate to marked emaciation, rough haircoat, sunken eyes, crusty ocular secretion on the medial eyelid, and slimy nasal discharge as well as a dry muzzle and mouth. The turgidity of the skin was clearly reduced. The frequency of respiration and pulse was demonstrably higher in two calves and the cow; in the rest of the patients these signs, as well as the body temperature in all animals, remained within normal range. The specific observations of the separate organic systems resulted in the following findings. (See Table 1.)

Circulation: Heartbeat, at first, 76 to 120 per minute; later, in four animals, bradycardia (48 to 68 per minute); depending on the degree of illness the episclera was injected.

Digestive system: Almost complete inappetence (with the exception of case No. 5) with increased thirst. Rumen motility was greatly reduced or atonic; the contents (also with the exception of case No. 5) were found, on deep palpation of the left abdominal wall, to be no longer stratified, but thinly viscous and pulpy, intermixed, or entirely liquid. By rumenotomy (four animals) and/or by necropsy (six animals) it was shown that this liquefaction could have possibly depended on a damming up in the omasum; the contents were of a dark olive brown color; there was a striking stale-sour odor ("like abomasum contents"); also a lowered pH of the contents. The glucose fermentation test and the methylene blue test showed greatly to completely inactive rumen fluids; there was a raised total acidity and relatively high chloride contents; the entrance to the second stomach was "inflexibly" open. The feces of all the patients, except for case No. 6, at least occasionally, were thinly viscous to soupy, usually foul-smelling, black or golden brown to a mustard-like color (a mixture of blood or bile pigment). Parasitologic analysis showed, with the exception of case No. 5, which had intestinal-trichostrongyloids, no parasites present. Liver function tests showed at best a slight increase in the total bilirubin content in the serum (a maximum of 0.4 mg%) as well as a moderately active increase in the SGOT level (50, maximum 164 mU/ml).

Urinary function: More often than not the test results showed a bright, clear and lightly frothy urine, a regular strikingly lowered specific gravity, a slight to moderate albumin content, as well as exhibiting positive glucose findings. The urine sediment contained epithelial cells in all tests. The pH value of the urine was usually slightly acid.

Motor capacity and central nervous system: General sluggishness, stiff and hesitating gait; in the final stages, a fixed stance, with sideways head-shaking, staring, and teeth gnashing.

Metabolism: Hematocrit, 41% (dehydration); serum-urine contents, 391 mg% (uremia); serum-calcium picture, 6.8 mg% (hypocalcemia).

Progress: In spite of all attempts to treat the ailment through intensive measures (cleaning out of the rumen contents, and inoculation with rumen contents from healthy animals, a diet of hay, intravenous or subcutaneous doses of balanced electrolyte solutions, grape sugar and/or calcium borogluconate, charcoal powder or laxative *per os*), the condition of all the patients (with the exception of case No. 5) deteriorated, so that they were either expediently slaughtered (four animals) or died (one animal). Case No. 5 alone showed, with the gradual decrease of the serum-urea contents, increased appetite, return to normal of ruminal activity, and retrogression of the general symptoms, so that it could be discharged after a twelve-day stay in the clinic.

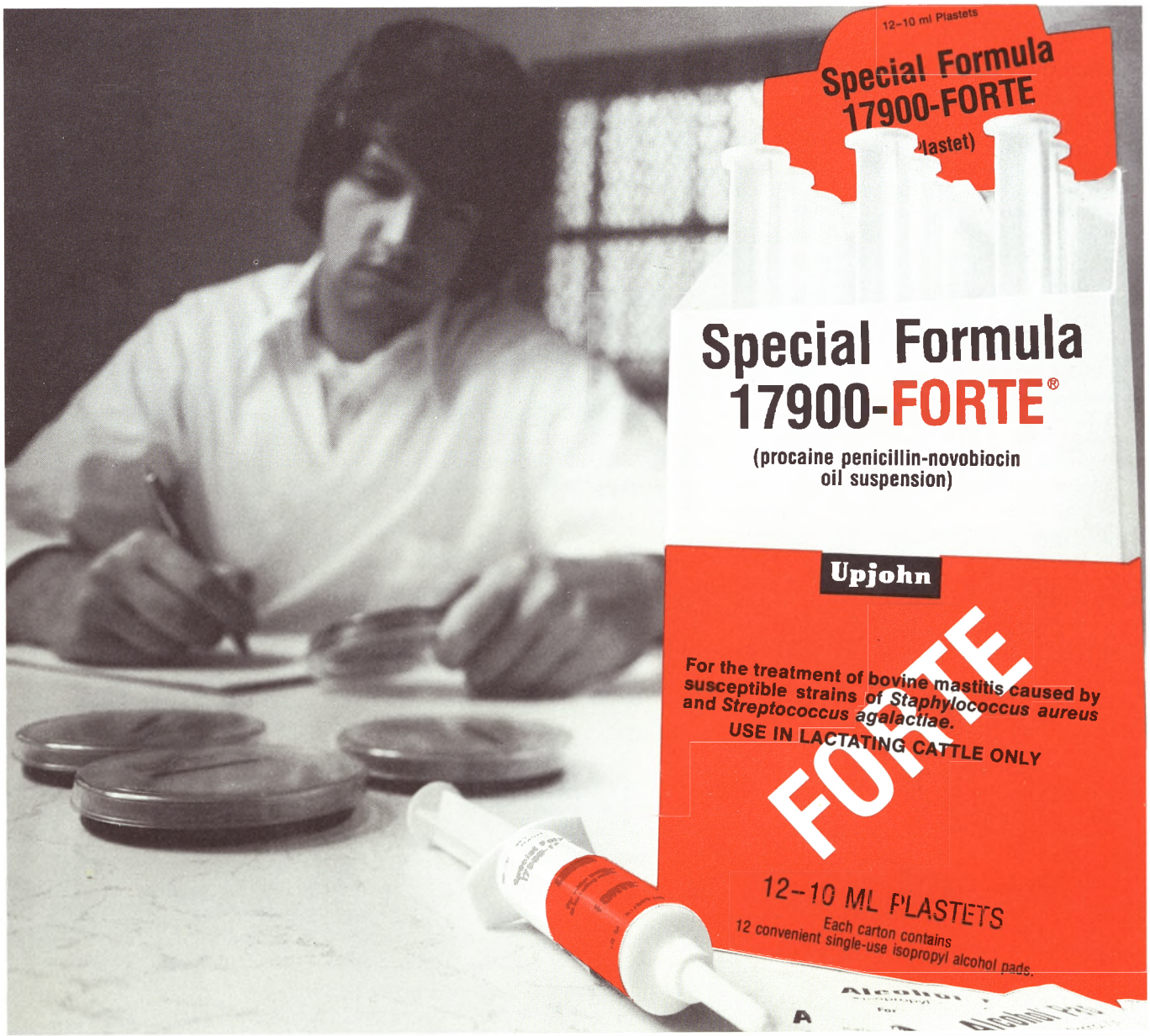

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Table 1: Summary of the Seven Patients' Anamnestic and Clinical Findings

Case No.	1 calf	2 calf	3 calf	4 calf	5 calf	6 calf	7 cow
Length of time on pasture with a stand of oak trees	3 mos.	3 mos.	> 2 mos.	4 wks.	4 wks.	3 wks.	6 wks.
No. of sick/simultaneously with other pastured animals	3/7	3/7	1/6	1/9	2/14	2/3	1/5
Length of illness before admittance to the clinic	1 wk.	1 wk.	2 wks.	5 days	1 wk.	1 day	3 days
Length of hospitalization	7 days	5 days	2 days	6 days	12 days	2 days	1/2 day
Final disposition	slaughtered	died	died	slaughtered	slaughtered	slaughtered	slaughtered
Rumen contents (flank palpation)	viscous intermixed	predominantly liquid	predominantly liquid	liquid	stratified	predominantly liquid	entirely liquid
Rumen pH	7.1*	(very acid)	7.5*	6.4	6.6	6.7	4.5
Methylene blue test (No. of minutes)	7.5	/	12	/	4.5	> 15	> 60
Glucose test (ml CO_2 within 30/60 min.)	0.02/0.3	/	0.4/0.6	/	0.6/0.7	0/0	0/0
Infusoria contents (estimated) capacity	(+) -	/	+ - 30% dead	/	(+) - 40% dead	(+) - dead	-
Total acidity of rumen fluids (Titration units)	35.8	/	/	/	26.4	29.2	28.0
Chloride capacity of the rumen fluids (mval/1)	/	/	/	/	19.4	33.7	88.7
Indication of a damming up of the abomasum contents in the omasum	+(0,2)	+(0,2)	+(2)	+(0,2)	?	+(2)	+(0,2)
Diarrhea	(+)	+	+	(+)	(+)	-	only golden brown mucous
Urine:	clear	clear	clear	clear	clear	clear	slightly cloudy
pH	6.9 (→ 6.5)	7.0	5.9	7.6	6.6 (→ 7.4)	6.7	/
specific gravity (g/ml)	1.0009	1.010	1.013	1.010	1.010 (→ 1.001)	1.018	/
albumin	+(+)-	+--	++-	+--	++- [-(+)--]	++-	++-
glucose (%)**	2***	2	-	0.1	0.25	0.25***	/
sedimentation	kidney epithelial cells among others	platelets	as No. 1	as No. 1	as No. 1	/	/
other	-	-	-	-	-	-	bilirubinoid ketone bodies
Serumuria (mg %)	376 (→ 210)	404	211	513 (→ 435)	290 (→ 53)	554	/
Serum creatinine (mg %)	/	/	/	45	8.1 (→ 3.7)	/	/
Serum calcium (mg %)	7.1 (→ 9.0)	5.0	8.7	6.3	8.0 (→ 9.3)	5.8	/
Hematocrit (%)	40	39	51	36	38	41	/

Clarification: * = drawn from the findings (containing saliva); ** = determined by glucose test strips/Boehringer-Mannheim; *** = after receiving glucose solution; (+) = occasionally; ? = possible (not operated on); / = not examined; u.a. = fatty degenerate cells, separate leuco- or erythrocytes and/or epithelial cells; - = later; (O) = discovered during rumenotomy; (2) = discovered at autopsy.

Discussion of Etiology

Apart from some not yet described symptoms (glucosuria, hypocalcemia, liquefaction of the rumen contents), the observed aspects of the disease corresponded fully and entirely to poisoning by "green oak leaves" (in spring: "mal de brout"; "oak brush disease"; "oak bud poisoning") or through acorns (in the fall: "brûlure par les glands"; "acorn poisoning"), as described in the literature, of all the oak meadows of Southeast Europe (1, 19), England (7, 12, 15, 23), Holland (2), France (5, 22), Scandinavia (20) and the United States (3,6,9,10,11,13,14,16,17,18). Save for the clinical manifestations and the season of the year in the results of the individual cases, the following facts are indications for considering acorn poisoning: The owner of case No. 5 mentioned previously that oaks grew in his pasture; with the rumenotomy and/or with the slaughter of animals No. 1 through 4, 6 and 7, acorn residue was found to be part of the rumen contents; in some oak leaves were found; the attending veterinarian stated that in cases Nos. 1 and 2 the necropsy results showed that the animals fell ill with similar symptoms, which rapidly terminated in bleeding from the kidneys and the urinary tract.

To insure that these observations were about acorn poisoning, the pastures involved were checked. It proved that only the calf in case No. 6 had been pastured with adult animals, and that it, like the dry

pastured cow in case No. 5, had not been regularly observed. The presence of oaks *(Quercus robur s. pedunculata)* occurred on all pastures in lesser or greater number (from 5 to 150); they grew either thickly near the enclosed pasture (cases No. 1 and 2) or in the pasture itself (the rest of the cases). Further, these places produced a rich crop of acorns, which, often following particularly stormy weather, and often in large masses, and even though not yet ripe, fell to the ground in the pastures. In four cases (Nos. 3, 4, 6 and 7) the cows were seen by the owners or relatives to be actually eating the acorns; at the time they considered this observation of no great importance. In the case of No. 5, there was large amount of feces which contained the undigested residue of acorns.

To check against the toxicity of the acorns, a healthy 385 kg calf was fed a total of 9 kg of ripe acorns *(Quercus robur s. pedunculata).* part mixed with water and given by mouth (intubated), and part fed mixed with "Troblako." The general condition of the animal remained unchanged in the period following. Its heart rate on the fifth day sank to 48-50 beats per minute, on the twentieth day it was again 80 beats per minute. The rumen contents were viscous and intermixed. The fecal consistency remained medium to thickly pulpy; from the third to the fifth day it was noticeably dark and slime-covered; from the sixth to the eighth day it contained undigested acorn particles. The laboratory findings of the body fluids showed, with the exception of the temporary lowering of the specific gravity of the urine to 1.006 on the third day, that the urine was bright and clear and there was no noteworthy change from the norm.

The physical findings were disclosed in part in the autopsy room (cases Nos. 1, 4, 6 and 7) and in part in the dissecting lab (cases Nos. 2 and 3). These are listed in Table 2. Heart findings ranged from a fibroplastic pericarditis (two times) to a subendocardial bleeding. Two of the animals had not only an alveolar edema, but also an alveolar interstitial emphysema. Submucosal petechiae were found near the pillars of the rumen in case No. 2. The abomasum of all the animals necropsied presented an abomasitis ranging from a medium to a high catarrhal hemorrhagic abomasitis, from (in case No. 1) an edema of the mucous membrane to ulceration. In cases Nos. 2 and 6 the strong urine odor of the abomasum was striking. In the intestines the enteritis (medium to high) was of a catarrhal or of a hemorrhagic character. In case No. 1 there was such a massive discharge of fibrin in the intestinal lumen that the final intestinal feces lay in an up to 3 mm fibrin bed. Edema of the gall bladder wall was found (five times); fibroplastic perihepatitis (one time) and fatty degeneration (two times). The bile fluids were mostly of a golden brown and of a slimy-slick consistency. In two animals there was bleeding in the renal fat layers or petechial bleeding of the renal surface; moreover, the kidneys of more than half the cases were larger than normal. The spleen of case No. 3 showed subcapsular petechiae and distinct follicular hyperplasia; normally this organ was unchanged.

Table 2: Summary of the Pathologic and Anatomic Findings, Cases 1 to 4, 6 and 7

Case No.	1	2	3	4	6	7
Macroscopic changes						
Skin	dry	dry	dry	dry	dry	n.s.f.
Heart	pericarditis, fibroplasia	pericarditis, fibroplasia, subendocardial bleeding	subendocardial bleeding	n.s.f.	n.s.f.	n.s.f.
Lungs	n.s.f.	alveolar edema, interstitial and alveolar emphysema	same as No. 2	n.s.f.	n.s.f.	n.s.f.
Liver and gall bladder	edema of the gall bladder wall	same as No. 1	perihepatitis edema of the gall bladder wall	edema of gall bladder wall	same as No. 4, fatty liver	fatty liver
Bile color	green	gold-brown	gold-brown	gold-brown	gold-brown	/
Bile condition	slimy mucous	slimy mucous	slimy mucous	slimy mucous	slimy mucous	/
Spleen	n.s.f.	n.s.f.	follicular hyperplasia subcapsulated petechia	n.s.f.	n.s.f.	n.s.f.
Renal fat layer	n.s.f.	petechia	n.s.f.	n.s.f.	n.s.f.	/
Renal surface	n.s.f.	petechia	enlarged, pale	n.s.f.	petechia	/
Abomasum	catarrhal abomasitis, edema of the mucous membrane	catarrhal abomasitis to hemorrhagic	ulcerative abomasitis	catarrhal abomasitis	catarrhal abomasitis	catarrhal abomasitis, hemorrhagic
Intestines	catarrhal enteritis to fibrinose	catarrhal enteritis	catarrhal enteritis to hemorrhagic enteritis	catarrhal colitis	catarrhal enteritis	catarrhal enteritis to hemorrhagic enteritis

Histological findings: For the microscopic research, parts of the heart, liver, gall bladder, kidneys, abomasum and small intestine were fixed in a 10% formalin solution. After paraffin fixation tissue slices were stained with hematoxylin-eosin, the kidney sections were separately subjected to the PAS-reaction. In Table 2 are shown the histological findings of the liver, abomasum, small intestine and kidneys.

The myocardium in case No. 1 showed great hyaline membrane degeneration. With the exception of case No. 4, the RHS of the liver showed constant medium to high grade activity; more than once this organ showed some degree of fatty degeneration. In three cases a slight nonpurulent hepatitis was shown. Only in two cases did the microscopic examination of the gall bladder show a medium edema or infiltration with lymphocytes. The macroscopic findings in the abomasitis also showed histologic confirmation–in case No. 1 it had a clearly purulent character. The most prominent changes were found in the kidneys, where the changes were much alike. Often renal damage could be demonstrated in several developmental stages in the same organ; in the region where the urine first is formed, tannin and the products of its degeneration exerted strong inflammatory stimuli on the glomeruli and the proximal tubule segments. The epithelia of the nearby tubule segment, along with the basal membrane, showed, in the initial phases, an edematous swelling. In a continuing course there was a stronger albumen (or protein) exudation which wavered between tubular epithelial and basal membrane; the basic cells were separated and collected in the lumen of the tubules. Later, individual sections of the tubules were found filled with a PAS-positive mass. Nearby the exudate contained leuco- and lymphocytes in a varying quantity. There also developed, as a rule, a medium to high grade interstitial edema.

Discussion of Clinical and Etiological Factors

After the appearance of symptoms, all laboratory findings, all laboratory findings and accompanying circumstances pointed without doubt to acorn poisoning. The agreement of the data found in the literature also included the following noteworthy facts: As a rule, in spite of common pasturage, only certain cows or only a small part of the herd became ill with this disease, and certainly almost always the younger animals which seemed to eat the fallen acorns with special greed (under certain conditions it was the only food); most of the rest of the herd paid the acorns little or no attention (3,5,13). In addition, acorn poisoning in cattle was common only in those years in which there was an unusually rich crop of acorns, and then usually after earlier violent storms or rough weather, when an unusual amount of acorns would fall on the pasture (3,5,13); the timely connection between the sudden (acorn-caused) deaths and the stormy weather led both veterinarians and owners to believe that lightning could be the cause of death (3). Ripe acorns are nonpoisonous, at least substantially less toxic than the unripe acorns (and than green oak leaves). A tolerance test by Fowler and Richards (1965) displayed the obvious characteristics, but without serious indications of poisoning since the cattle were fed only ripe acorns. A pail full of green acorns should, after four days of feeding, be sufficient to present symptoms of poisoning (4). Oak leaves were poisonous during the same vegetative season; three to four weeks after the buds open the cattle no longer find the leaves appetizing (9). The poisonous constituent in acorns and oak leaves is tannin (4,7,8,14). Smith, Jones and Hunt (1972), from their observations, doubt that the symptoms depend on the effect of tannin alone.

With all the other authors, the length of illness varied from a few days to more than one month (3,6,7,11,17); the clinically manifested renal injury developed within ten to fourteen days (6,22). With individual patients events proceeded more or less along a protracted, designated course. In this we observed the high-grade emaciation and the gross diarrhea (with only small amounts of acorn residue found in the rumen) which were noted in the observations of Towers (1950) as well as Kingery (1963). The absence of the subcutaneous edema in the subcutis of the pendant portions of the body, mentioned more than once in the literature (9,13,17,18,19,22) is perhaps due to the fact that it dealt with acorn and not with oak leaf poisoning. The hypocalcemia and glucosuria described in this kind of poisoning are probably the result of renal failure. The unsuccessful course of treatment confirms corresponding results of Boughton and Hardy (1936) as well as Kingery (1963); generally 75-85% of the cases end fatally. Usually only individual animals were involved, and in ordinary years the pastures with stands of oak trees did not present a problem; for that reason acorn poisoning in this country has, up to now, often passed unrecognized, and the true situation underrated. All the owners who were questioned had never observed these same symptoms before on the same pasture; one of the owners thought the relationship between the ingestion of acorns and the illness was absolutely out of the question. Therefore, it appeared necessary to broach veterinarians and owners and call their attention to the latent danger of cattle on pasture with stands of oaks. The recognition of acorn poisoning is, with hindsight, and given the symptoms and accompanying conditions, not too difficult to diagnose under practice conditions if the veterinarian only considers the possibility. In the normal course of treatment it is particularly the severely emaciated, those who rejected other food and had other alimentary failures, which hardly have a chance to recover, and are never again worth anything to the owner. An effective preventive measure is not to allow the animals access to unripe acorns (and to green oak leaves) at certain seasons (by rotating cattle to oak-free pasturage, or where a storm could not pile acorns or leaves up on pasture land).

Anatomical and histological pathology: The cause of the subcutaneous edema of the ventral abdominal wall and dependent parts as well as the increase in the pleural, pericardial and peritoneal fluids (1,3,6,-10,13,14,17,18) described in the literature, was, in some of the research material, not readily determined because of the protracted course of the illness or because it was not fully determined if it dealt with acorn poisoning (rather than as a consequence of the ingestion of oak leaves). The ascertained changes in the heart muscle (hyalin-bunched, bleached in color) and the distinctive edema of the lungs are in close accord with the findings of Smith (1959). A liver fatty degeneration, which Llewellyn (1962) described, was noted in only one case.

Of pathogenic importance, suggesting an enlargement of the gall bladder (1,6) was the demonstration of activation of the RHS of the liver as a manifestation of the reaction of these organs to a toxic stimulant, as was also the characteristic slimy-slick condition of the mostly gold-brown colored bile, which appeared in 4 or 5% of the animals investigated. Pathological changes of the rumen pillars were seen which agreed with Tower's observations (1950). A preponderant number of authors (1,5,6,7,-13,14,17,19,23) described similar findings, a catarrhal to hemorrhagic inflammation of the abomasum, from which ulceration of the mucous membrane is possible (17). Likewise it was demonstrated that the intestine, and especially the base of the small intestine, had an inflammation ranging from catarrhal to hemorrhagic; the ammonia odor of the abomasum was noticeable. In regard to the macroscopic findings of the kidneys, several authors (2,6,10,13,14,16) noted (as we did also in case Nos. 1 and 2) bleeding in the kidney fat layer as well as petechia in the kidney surface area. The enlargement of these organs, as was observed in case No. 3, was reported by Boughten and Hardy (1936) as well as by Kingsbury (1964). Above all, the material presented by Smith (1959) and our own investigations demonstrated PAS-positive effusion of the proximal tubule segment to be of pathogenic significance. The pathological-anatomical and histological findings confirm, therefore, the clinical diagnosis of acorn poisoning.

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