Poisoning by crude oil in sheep and goats

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SUMMARY

This paper reports spontaneous cases of poisoning by crude oil in sheep and goats. The outbreak occurred on a ranch in the municipality of Areia Branca, Rio Grande do Norte state, northeastern Brazil. At total of fourteen sheep and goats died or were euthanized in two days. In the pasture, there were several oil extraction wells with cement foundation allowing the accumulation of rainwater and production water. Clinical evaluation revealed lateral recumbency with paddling, pupil dilatation, blindness, cyanotic mucosae, severe dehydration, tachycardia, severe dyspnea, rumen atony and bloat. Lesions were observed in the lungs, kidneys, spleen, liver, gastrointestinal tract, heart, and central nervous system. One ewe also presented ovarian teratoma. The association of epidemiological, clinical and pathological findings was crucial for confirming the diagnosis.

Keywords: Aliphatic hydrocarbons, aromatic hydrocarbons, crude oil, small ruminants.

RÉSUMÉ

Empoisonnement par le pétrole brut chez les ovins et les chèvres

Cet article rapporte des cas spontanés d'intoxication par le pétrole chez les moutons et les chèvres. La maladie est apparue à Areia Branca, RN, Brésil. Quatorze ovins et caprins sont morts ou ont été euthanasiés en deux jours. Dans les pâturages, il y avait plusieurs puits servant à l'extraction de pétrole dont la fondation cimentée permettait l'accumulation des eaux de pluie et de production. L'évaluation clinique a révélé un décubitus latéral avec signes de pédalage, mydriase, cécité, cyanose des muqueuses, déshydratation sévère, une tachycardie, une dyspnée, une atonie sévère de la panse avec ballonnement. Les lésions ont été observées dans les poumons, les reins, la rate, le foie, le tube digestif, le cœur et le système nerveux central. Un mouton a également eu un tératome de l'ovaire. La combinaison de confirmation épidémiologique, clinique et pathologique est essentiel pour le diagnostic.

Mots clés: Aliphatiques, hydrocarbures aromatiques, pétrole, petits ruminants.

Introduction

Crude oil is composed of aliphatic and aromatic hydrocarbons as well as resins and asphaltenes [2]. A common form of poisoning by petroleum and oil is through oil spills to the environment, affecting wild animals [11]. Major oil spills may result in significant environmental impact [3]. In livestock, most reports of poisoning occur by refined products [1, 5, 8, 20]. However, poisoning may be promoted by crude oil when animals have access to the fields of petroleum extraction [9, 12], or contamination of food by oil may occur during processing and transportation of food [7].

Numerous episodes of poisoning in cattle with petroleum and other oilfield substances have been reported [5-8], but there was no report of spontaneous poisoning by crude oil in sheep and goats. In small ruminants, there have been reports of ingestion of bunker "C" fuel oil (a residual fuel oil) [13] and surface water contamination with extrusions of natural gas condensate by rain water [1], causing fatalities in sheep. In goats, the reports include ingestion of kerosene [16] and diesel fuel leaking from an overturned truck tanker [20]. This paper aims to describe spontaneous cases of poisoning in sheep and goats by crude oil waste from extraction wells.

Case Report

We observed a spontaneous outbreak of crude oil poisoning on a ranch in the municipality of Areia Branca, Rio Grande do Norte state, Northeastern Brazil. Fourteen of 183 small ruminants died or were euthanized in two days in July 2011 (dry period in the region). The flock was composed of about 55 goats and 128 sheep of mixed breed and different ages grazing in native pasture (*caatinga*) and without dietary supplementation. The livestock was raised extensively and gathered once a week in a corral for counting. In the pasture, there were several oil extraction wells with cement foundation allowing the accumulation of rainwater and production water (defined as the water that is brought to the surface during the pumping and production of oil and natural gas) [6]. During oil extraction, hydraulic pumping causes regular dripping of crude oil in the accumulated water. During a visit to the farm, it was possible to find numerous oil extraction wells with the above characteristics (figure 1). We reiterate that the oil wells initiated extraction in November 2010; approximately ten months earlier of the outbreak. It is also notable that in July 2011, the dry season was intense and just one other source of drinking water was available in the property.

Figure 1: The accumulation of water mixed with crude oil at the ground of an oil extraction well.

The previous week, all animals were counted and found to be healthy before being released in the paddock. After a 24-h period, the rancher found 12 mature sheep dead and two agonizing (one ewe and one doe). One ewe among the dead and the two agonizing animals were sent to the Veterinary Hospital for clinical and pathological evaluation. One agonizing goat died on the way to hospital. Clinical evaluation of the ewe revealed lateral recumbency with paddling, pupil dilatation, blindness, cyanotic mucosae, normal rectal temperature (38.7°C), severe dehydration (>12%), tachycardia, severe mixed dyspnea, rumen atony and bloat. Hematology showed no alterations in RBC and serum protein, while WBC revealed moderate neutrophilia (8175 neutrophils/µL; reference: 4000–6000 cells/µL) and mild monocytosis (763 monocytes/µL; reference: 40–700 cells/µL). Biochemical assays showed elevated serum level of urea (110 mg/dL; reference: 8–20 mg/dL), normal creatinine values (0.8 mg/dL; reference: 1.2–1.9 mg/dL), and a slight increase of gama glutamiltransferase activity (63 U/L; reference: 20–52 U/L). Cerebrospinal fluid (CSF) collected through cerebellar-medullary puncture was analyzed. The presence of fibrin clots and high density (1.030) were the abnormalities noted. A CSF sample was submitted to microbiological assays.

Due to the agonic state and poor prognosis, the sheep was submitted to euthanasia *in extremis*. A total of three animals (two ewes and a doe) were necropsied. At necropsy, the gross findings included extensive subcutaneous edema. The lungs were congested and with presence of marked edema. Liver, spleen and kidneys showed severe congestion. A potent gasoline-like odor was detected in the gastrointestinal tract in all animals. This odor originated most strongly from the rumen and abomasum. There was diffuse mucosal hyperemia over the rumen and reticulum, besides easy shedding of the mucosa. The heart was flaccid, and the presence of blood clots in the ventricle chambers was noted. Adrenal glands showed a marked increase in volume. The brain appeared pale with severe edema, swelling with flattening of the gyri, and narrowing of the cerebral sulci. The cerebellum was shifted caudally with herniation through the *foramen magnum*. The cut surface of the cerebral cortex showed malacia areas with softening consistency and blackish coloration. The diagnosis of petroleum hydrocarbon toxicosis was suggested by the strong gasoline-like odor in the ruminal and abomasal contents. Toxicologic analyses were not performed. One of the ewes also showed approximately 4 L of serous peritoneal fluid, and the left ovary presented a large encapsulated tumor (figure 2). Gross examination revealed an ovoid neoplasm with pinkish-gray color, firm, smooth surface, with diameters of 7.5–9.2 cm, weighing approximately 350 g. After slitting, the presence of cystic or multilobular mass with pale or yellowish areas besides of friable tissue and reddish coloration in the center was observed. Fragments of the organs were collected and fixed in buffered formalin 10%, embedded in paraffin, sliced to a thickness of 5 mm and stained with hematoxylin and eosin.

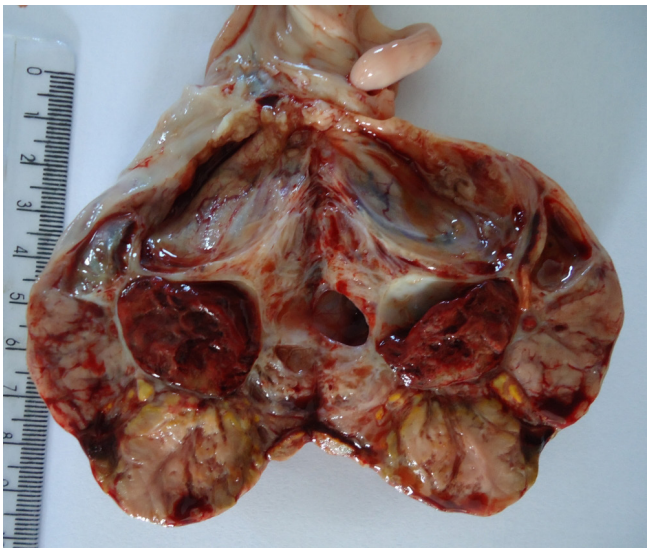

Figure 2: Teratoma from a ewe poisoned by crude oil.

Histopathological examination revealed toxic acute tubular nephrosis characterized by degeneration and necrosis of tubular epithelium, presence of intratubular granular casts and inclusion bodies (figure 3). Extensive foci of hemorrhage, edema, and coagulation necrosis of cardiac fibers were observed in the heart. In the liver, the presence of hepatocytes swelling with necrosis foci, megalocytosis, intense sinusoidal congestion, and accumulation of biliar pigment in the hepatic parenchyma was noted (figure 4). The presence of pre-neoplasic lesions in the liver (anisokaryosis, alteration of nucleus-cytoplasm ratio, mitosis figures and intense degree of nuclear hypercromasia) was also observed. Rumen and reticulum showed hydropic degeneration of epithelial cells. Cell depletion with thinning of the germinal centers and lymphoid follicles was observed in the spleen. In the cerebellum, degenerated Purkinje cells (eosinophilic retracted cells with chromatolysis) and moderate loss of these cells were observed. Brain revealed polioencephalomalacia (PEM) characterized by the presence of a segmental laminar neuronal necrosis with acidophilic cytoplasm and pycnotic nuclei (red neurons), cytoplasmatic vacuoles, gliosis, perineural and perivascular dilatation. Thalamic nuclei also

presented segmental laminar neuronal necrosis and the presence of basophilic, homogeneous, and hyaline clots with chromatin shifted to the cell periphery, suggesting inclusion corpuscle (figure 5). The ovoid neoplasm in the ovary of one ewe microscopically showed predominance of dense and loose connective tissue with foci of cardiac; luteinized ovarian, adipose, osseous and glandular tissues; cysts lined by single columnar epithelium, blood vessels, mononuclear leukocyte cells with predominance of macrophages and few follicles and neurons. The presence of undifferentiated pleomorphic multinucleated cells with giant nuclei and evident nucleoli was also observed. These findings are characteristic of ovarian teratoma.

It is also important to state that the flock was sold out in the months subsequent of the outbreak because it was cheaper to the owner than building a fence in every oil extraction well. This action made it impossible to follow up the flock regarding new cases of chronic diseases due to the non-lethal ingestion of crude oil.

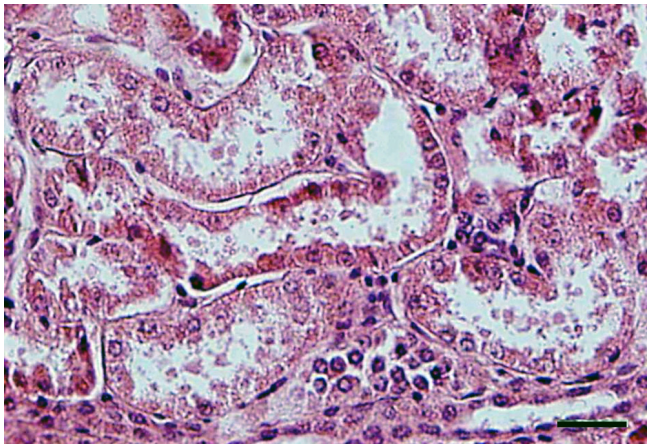

Figure 3: Kidney from a sheep poisoned by crude oil showing degeneration and necrosis of tubular epithelium with presence of intratubular granular casts. Hematoxylin and eosin. Bar = 10 µm.

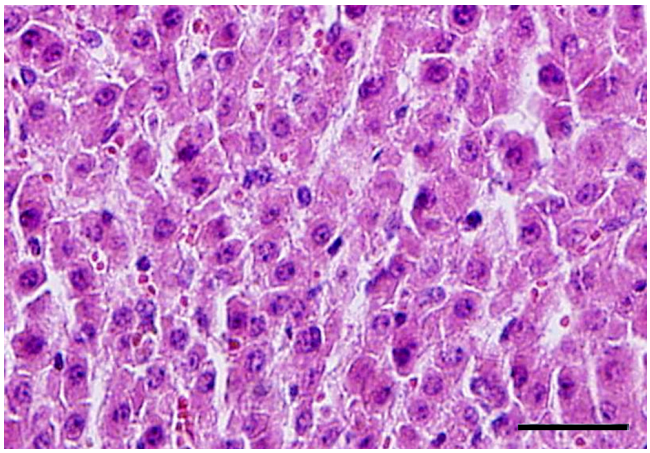

Figure 4: Liver from a sheep poisoned by crude oil showing hepatocytes necrosis, degeneration, and megalocytosis. Hematoxylin and eosin. Bar = 25 µm.

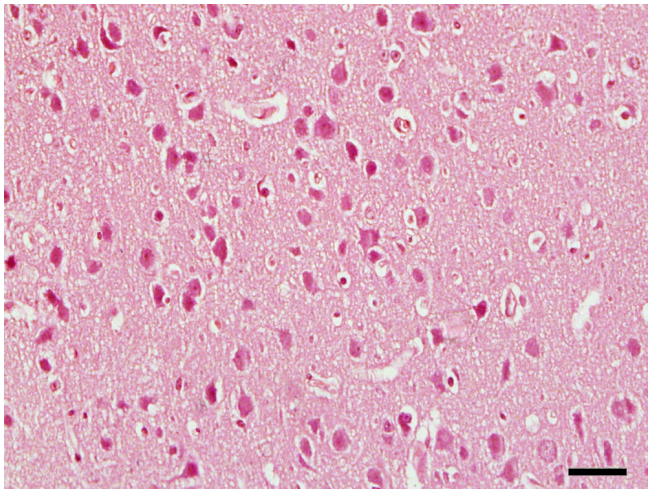

Figure 5: Thalamic nuclei from a sheep poisoned by crude oil showing segmental laminar neuronal necrosis with acidophilic cytoplasm and picnotic nuclei (red neurons), cromatolisis, and presence of intracytoplasmatic vacuoles. Hematoxylin and eosin. Bar = 10 µm.

Discussion

The outbreak occurred during the dry season when food and water are scarce in the region, and the contaminated water in the cement foundation of the extraction wells was accessible to the animals. This incident reinforces the fact that ruminants are often at risk of ingesting contaminated food or water when proper feed and salt are not made available to them [9, 17].

Our results showed that crude oil poisoning did not alter hematological parameters of the ewe, as was described in cattle [6]. Some authors reiterate that leukocyte numbers increase as chemical pneumonia advances [6]. Elevated serum activity of GGT may be caused by acute hepatic dysfunction, as was also observed in an outbreak of poisoning by kerosene in heifers [5]. Histopathology confirmed liver damage in the present animals. The increased serum concentrations of urea indicated injury to renal tissue, which was confirmed through histopathology by the finding of toxic acute tubular nephrosis. The presence of clots in the CSF is indicative of bacterial contamination [18], although no microorganism seemed to growl in the microbiological assay. High density in CSF is an unusual finding in sheep with PEM, since the presence of normal protein concentrations with only a marginal increase in white cell concentration with predominance of lymphocytes is reported [18].

Clinical signs observed in the only alive ewe corroborate those in the literature [1, 5, 6, 17]. The volatile components of crude oil have anesthetic properties, are more irritating and are more likely to be aspirated into the lungs, causing aspiration pneumonia [6, 7, 9]. However, in the present report, dyspnea was observed clinically, and the only gross findings in the lungs were congestion and edema. Crude oil destroys rumen microflora and enzymatic activity, causing ruminal atony, and perhaps contributes to the onset of bloat [17]. Several

oil compounds are highly corrosive and when ingested cause direct damage to the wall of the rumen [8, 9]. Lesions in the rumen and reticulum were evident and characterized by diffuse mucosal hyperemia and easy shedding of the mucosa and degeneration of mucosal cells of the epithelial lining. Central nervous signs are linked to the pathological changes caused by PEM. The onset of PEM may be attributed to the ingestion of rainwater mixed with production water, which can induce salt (sodium ion) poisoning and sometimes can be high in sulfates [6]. The consumption of water high in sulfate by cattle have increased risk for thiamine-responsive PEM, and the risk increases with decreased dietary copper [10], while nervous system pathology presented by other studies was limited to hemorrhage into the cerebellar peduncles in cattle [6], and mild perivascular hemorrhage was seen in the pia mater and in the white matter of the cerebellum and cerebrum of sheep [1].

In a sheep, we observed the presence of ovarian teratoma. This is a rare neoplasm of germ cell origin in domestic animals. It is characterized by the presence of tissues derived from more than one of the three germ layers, such as integument and its appendages, teeth, or even bone tissue, adipose and nervous [14]. It is most commonly found in young animals [19], and its formation has been attributed to gene disruption that causes signaling defects in either oocytes or in the surrounding follicular granulosa cells [4]. Some cases of human ovarian teratoma were hereditary, but most cases occur sporadically [4]. The diagnosis of ovarian teratoma is unusual in sheep, and is considered an incidental finding at necropsy or slaughter houses [15]. This finding is associated with the fact that this neoplasm does not interfere with the reproductive functions of females and usually are asymptomatic [19]. Since the crude oil and petroleum products contain compounds suspected to be human carcinogens, meat or milk from exposed animals are not fit for human consumption. Also, it is possible that rumen microflora metabolize the chemicals found in crude oil or petroleum products, forming carcinogenic metabolites [17]. So the possibility of the pre-neoplasic lesion in the liver and the ovarian teratoma to be caused by the chronic ingestion of small quantities of crude oil could not be dismissed.

The access to crude oil extraction wells and other oilfield substances by livestock may result in acute poisoning, especially in cases that other sources of water are limited. Lesions were observed in the lungs, kidneys, spleen, liver, gastrointestinal tract, heart, and central nervous system. The association of epidemiological, clinical and pathological findings (mainly the potent gasoline-like odor detected in the gastrointestinal tract) was crucial for confirming the diagnosis.

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