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Poisoning by (Conium maculatum) and liver deficiency in a bovine. Clinical case

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Abstract

In the majority of cases, poisonings in livestock occur accidentally. The primary causes of poisoning are associated with the ingestion of toxic plants, which contain a range of substances including alkaloids, nitrates, saponins, and others, that can lead to fatal outcomes. The objective of the investigation was to ascertain the cause of death of the bovine. The animal in question was a Simmental breed heifer, aged one year and two months, weighing 250 kg live, and displaying symptoms consistent with Conium maculatum poisoning. The results of the autopsy demonstrated the following: the external examination revealed slight profuse hemorrhage and icteric mucosa on the lips, a pale and icteric flaking muzzle, pale and icteric vulvar and ocular mucosa, and small hemorrhagic vesicles focused at the level of the cross, hip, and dewlap. Additionally, the abdomen was found to be slightly enlarged with a dull sound. The internal examination revealed the presence of generalized jaundice, icteric pleural tissue, icteric myocardium, cardiomegaly with flaccidity in the left ventricle, and icteric peritoneum in the abdomen. Additionally, the rumen and intestines exhibited apparently normal contents. The abomasum and liver exhibited adhesions with necrotic tissue, hepatomegaly, necrotic tissue at the level of the common bile duct, rupture of the gallbladder with the content distributed throughout the bile ducts, and an abundant blackish liquid with a putrid odor. Laboratory tests to rule out bluetongue and bovine vesicular stomatitis were conducted at the National Agrarian Health System (SENASA) and yielded negative results for both diseases. It was determined that the bovine had died as a result of poisoning by hemlock and liver deficiency.

Keywords: *Bovine, grazing, poisoning, conium maculatum.*

Resumen

Las intoxicaciones en el área pecuaria en la mayoría de los casos se producen de manera accidental, las principales causas de intoxicación se relacionan con la ingestión de plantas tóxicas que contienen alcaloides, nitratos, saponinas, entre otras, desencadenando la muerte. El objetivo de la investigación fue determinar la causa de muerte del bovino. Fue una vaquilla de raza Simmental con presencia de sintomatología a intoxicación por *conium maculatum*, de un año y dos meses de edad de nombre Carla, de 250 kg de peso vivo. Como resultado a la necropsia se obtuvo: al examen externo labios con ligera

hemorragia profusa y mucosa ictérica, morro descamado cetrino, pálido, ictérico, mucosa vulvar y oculares pálidas e ictéricas, pequeñas vesículas hemorrágicas focalizadas a nivel de la cruz, anca y papada, abdomen ligeramente aumentado de tamaño con sonido mate. Al examen interno se encontró ictericia generalizada, tejido pleural ictérico, miocardio ictérico, cardiomegalia con flacidez en el ventrículo izquierdo, en el abdomen presencia de peritoneo ictérico, rumen e intestinos con contenido aparentemente normal, adherencias en el abomaso e hígado con tejido necrótico, hepatomegalia, tejido necrótico a nivel de conducto colédoco, ruptura de la vesícula biliar, con el contenido diseminado con fibrina y tejido necrótico dentro de los conductos biliares, abundante líquido de color negruzco con olor putrefacto. Las pruebas para descartar lengua azul y estomatitis vesicular bovina fueron procesadas en el laboratorio del Sistema Nacional de Sanidad Agraria (SENASA), siendo negativas para ambas enfermedades. Concluyendo muerte del bovino: intoxicación por cicuta y deficiencia hepática.
Palabras clave: Bovino, pastoreo, intoxicación, *conium maculatum.*

INTRODUCTION

The economic losses incurred in the livestock sector due to plant poisoning are significant, resulting in the death of animals and a reduction in production (García, Santos & Capelli, 2016). A diverse range of plant-derived compounds possess toxic properties that can harm animals (R. Odriozola, 2018). Grazing animals typically consume toxic plants to varying degrees, often exhibiting no discernible symptoms or only mild indications. However, this is contingent on the animal's idiosyncrasies. In this instance, only a Simmental breed heifer manifested indications of poisoning by hemlock (Conium maculatum). Food poisoning is defined as any poisoning caused by the ingestion of toxic substances, microorganisms, metals, additives, hormones, or other foreign substances present in food products (Pinillos et al., 2003). The hogweed-like hemlocks, Conium maculatum, and the aquatic hemlock, Cicuta virosa, are commonly consumed by mistake and can be lethal (Pinillos et al., 2003). It is an annual or biennial herb, reaching a height of up to 2 m, with a fistulous stem that is stained purple at the base (in some cases, the petioles also exhibit purple spots) (Renobales & Sallés, 2001).

Taxonomic information
Kingdom: Plantae
Phylum: Magnoliophyta
Class: Magnoliopsida
Order: Apiales
Family: Apiaceae
Genus: Conium
Species: Conium maculatum L.
Common name: Cicuta (Lezama, 2014)

It is a highly toxic plant due to presence of alkaloids 2-pentypiperidine, coniine and g coniceine, neurotoxins involved in the poisoning and death of cattle because they stand out for being toxic compounds with potent neurotoxic and teratogenic action, the most susceptible species being cattle, pigs and to a lesser extent horses and sheep. (Lezama, 2014) (Odriozola, 2015). Poisoning by this plant has been reported in horses, pigs, goats, sheep and cattle. It can also cause congenital malformations in calves (Díaz, 2010). The symptoms of poisoning by consuming this plant are: difficulty eating, presence of foam in mouth, excessive salivation, dilation of the pupils, nervousness, muscle spasms, visual disturbances (Behnken & Durgan, 2023). Likewise, the clinical signs of liver failure in cattle are nonspecific and there may be considerable damage before they are evident (Araya, 1991). The objective of the study was to determine the cause of death of the cattle.

Investigation Development

Symptoms

A female Simmental bovine, 1 year and 2 months old, named Carla, weighing 250 kg, is reported.

Clinical picture evolution

On the first day she presented with restlessness, abdominal pain, lateral and rotary head movements. On the second day, lateral and rotational head movements, ear tossing, eyelid edema, disorientation, and signs of photosensitivity. On the third day, decreased eyelid edema, icteric and cyanotic snout with diffuse hemorrhage at the tip of the snout, chocolate colored urine, decreased appetite. On the fourth day, hemorrhagic snout, chocolate-colored urine, hemorrhagic rashes on the forelimbs, rashes on the skin of the chest, back, and hips. Blood comes out on palpation, ataxia, anorexia. Fifth day: hemorrhagic snout, chocolate-colored urine, hemorrhagic areas on the forelimbs, rashes on the chest, back, and hips that bleed when touched, ataxia, anorexia, dehydration. Sixth day: hemorrhagic snout, chocolate-colored urine, hemorrhagic areas on the forelimbs, rashes on the chest, back and hips that bleed when touched, ataxia, ruminal stasis, anemia, jaundice. In addition, 5 ml of blood was collected (in EDTA tubes) for analysis to rule out bluetongue and vesicular stomatitis. Seventh day: hemorrhagic snout, chocolate-colored urine, hemorrhagic areas on the front legs, rashes on the chest, back and hips that bleed when touched, ataxia, ruminal congestion, anemia, jaundice. Eighth day hemorrhagic snout, chocolate colored urine, hemorrhagic areas on the forequarters, skin rashes on the chest, back and hips that bleed when touched, ataxia, ruminal stasis, anemia, jaundice, pain in the flanks 9. - Hemorrhagic snout, chocolate-colored urine, hemorrhagic areas on the forequarters, rashes on the skin of the chest, back, and hips that bleed when touched, ataxia, ruminal stasis, anemia, jaundice, ataxia, prostration, prone position, and death.

Results of necropsy: On external examination, the body condition of (4) was observed, lips with slight profuse hemorrhage and icteric mucosa, scaly sallow, pale, icteric muzzle with slight profuse hemorrhage, pale and icteric vulvar and ocular mucosa, presence of small hemorrhagic vesicles located at the level of the cross, hip and dewlap, abdomen slightly enlarged with a dull sound. Internal examination revealed generalized jaundice, icteric pleural tissues, icteric myocardium, cardiomegaly with flaccidity of the left ventricle, presence of icteric peritoneum in the abdomen, rumen and intestines with apparently normal contents, adhesions in the abomasum and liver with necrotic tissue, hepatomegaly. Adhesions in the abomasum and liver with necrotic tissue, hepatomegaly, necrotic tissue at the level of the common bile duct, rupture of the gallbladder with contents spread with fibrin and necrotic tissue within the bile ducts, abundant blackish liquid with a putrid odor. Tests to exclude bluetongue and vesicular stomatitis were carried out in the laboratory of the National Agrarian Health System (SENASA) and were negative for both diseases.

The final diagnosis: death due to Conium maculatum intoxication caused by liver deficiency due to an increase in the size of the gallbladder.

Treatment

Day 1: 15 mL of sodium metamizole (500 mg/mL) was administered intramuscularly for pain and spasm at a daily dose for 3 consecutive days.

Second day: 6 ml dexamethasone was administered intramuscularly, 2.5 ml chloroprofenpyridamine maleate intramuscularly and 15 ml sodium metamizole.

Third day: 6 ml dexamethasone intramuscularly, 2.5 ml chloroprofenpyridamine maleate intramuscularly.

Fourth day: 250 ml of 5% glucose was administered intravenously, 25 ml of 5 mg carbazochrome sodium sulfonate, Vit. K3 (menadione sodium bisulfite) 10 mg, meloxican at a concentration of 20 mg/ml at a dose of 7. 5 ml per kg of live weight, chloroprofenpyridamine maleate 2.5 ml intramuscularly, oxytetracycline L.A at a dose of 25 ml per kg of live weight at a concentration of 200 mg/ml and diminazene diaceturate, antipyrine, vitamin B12 at a dose of 20 ml.

Fifth day. 25 ml of sodium carbazochrome sulfonate 5 mg, vitamin K3 (menadione sodium bisulfite) 10 mg, meloxican at a concentration of 20 mg/ml at a dose of 7.5 ml per kg of live weight,

chloroprofenpyridamine maleate 2.5 ml intramuscularly, physiological saline solution 250 ml were administered.

Day Six. 1 bag of Vit. A (retinol palmitate) 10,000 IU, Vit. D3 (cholecalciferol) 20,000 IU, Vit. E (alpha tocopherol acetate) 500 mg, Vit. B1 (thiamin hydrochloride) 5 mg, Vit. B2 (riboflavin 5 sodium phosphate) 10 mg, Vit. B5 (calcium pantothenate) 2 mg, Vit. B6 (pyridoxine hydrochloride) 2 mg, Vit. B12 (cyanocobalamin) 0.2 mg, DL-methionine 2000 mg, L-lysine (L-lysine hydrochloride) 100 mg, Probiotics 2. 5 Billion, Prebiotic (Inulin) 620 mg, Sodium Bicarbonate 30,000 mg, Calcium Carbonate 15,000 mg, Zinc Sulfate (Heptahydrate) 2500 mg, Cobalt Sulfate (Heptahydrate) 250 mg, Manganese Sulfate (Nomohydrate) 250 mg, Green Ammonium Iron Citrate 1000 mg, Ginger Powder 7500 mg, Gentian Extract 10,000 mg, Yeast Extract 7500 mg, Dimethylpolysiloxane 2250 mg, Colloidal Kaolin (Hydroxylated Aluminum Silicate) 5000 mg, Starch 5000 mg, Dextrose (Monohydrate) 5000 mg, Excipients 100 g.

25 ml of carbazochrome sodium sulfonate 5 mg, Vit. K3 (menadione sodium bisulfite) 10 mg, Meloxican at a concentration of 20 mg/ml at a dose of 7.5 ml per kg of body weight.

Each 1 ml contains: vitamin B12 (cyanocobalamin-0.012 mg), cobalt acetate-0.5 mg, tryptophan-2. 5 mg, histidine-5 mg, citric acid-5 mg, DL-methionine-10 mg, iron (ammonium citrate-20 mg), sodium cacodylate-30 mg, thiamine hydrochloride-5 mg, riboflavin-5-phosphate-2 mg, nicotinamide-50 mg, pyridoxine-hydrochloride-10 mg, sodium glycerophosphate-20 mg, excipients q.s./1 mL.

Day seven: 15 ml meloxisan, 25 ml hemostop, 10 ml hematopan gold were applied.

Eighth day: 250 ml glucose 5%, 15 ml hematopan gold, 15 ml meloxisan, 2 ml histapro, 20 ml oxytetracycline, 25 ml hemostop.

Day nine: 15 ml meloxisan, 2 ml histapro, 25 ml hemostop, in the afternoon 250 ml glucose 5%, 15 ml hematopan gold.

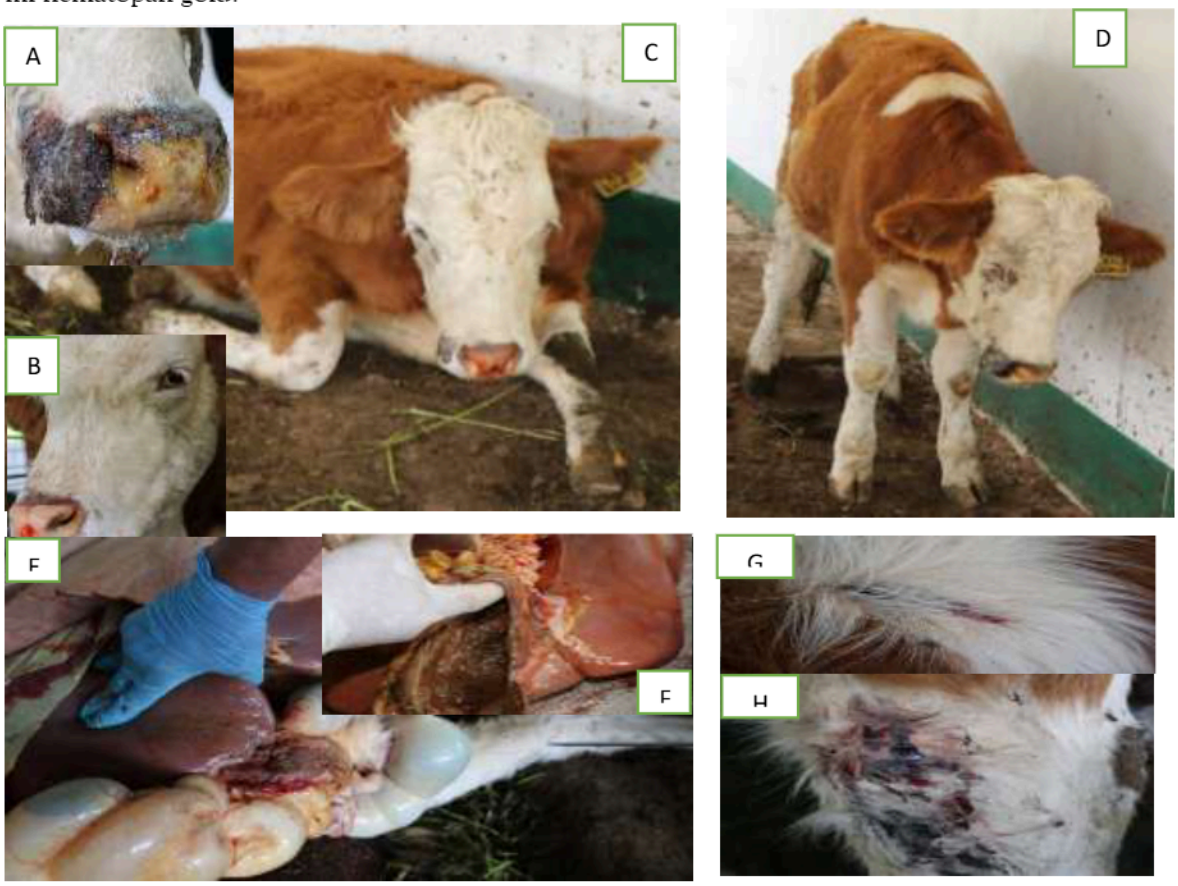

Figure 1. Clinical picture A. Sallow, icteric scaly muzzle. B. Muzzle with slight, profuse hemorrhage. C. Prostration and anorexia. D. Ataxia, weakness. E. Adhesions in the liver, small intestine and rumen, hepatomegaly. F. Rupture of the gallbladder. G and H. Hemorrhagic vesicles on the skin at the height of the withers and knee.

Figure 2. Conium maculatum A. Flowering. B. Leaf structure. C. Complete plant.

CONCLUSIONS

1. **Cause of Death Determination**

The bovine's death was caused by *Conium maculatum* poisoning, which led to severe liver deficiency. This diagnosis was confirmed through necropsy, revealing characteristic signs such as hepatomegaly, necrotic liver tissue, gallbladder rupture, and bile accumulation in the bile ducts.

2. **Importance of Differential Diagnosis**

Laboratory tests ruled out infectious diseases such as bluetongue and vesicular stomatitis, focusing the diagnosis on poisoning by a toxic plant.

3. **Clinical Evolution and Specific Symptoms**

The clinical case demonstrated the progressive development of symptoms characteristic of *Conium maculatum*poisoning, including jaundice, anorexia, ataxia, prostration, chocolate-colored urine, and hemorrhagic skin lesions. These signs evidenced multisystem damage.

4. **Treatment Limitations**

Despite medical treatment involving analgesics, anti-inflammatory drugs, vitamins, intravenous fluids, and other medications, it was not possible to reverse liver damage or control the poisoning, highlighting the rapid progression of this condition.

5. **Impact of Toxic Plants on Livestock**

This case underscores the importance of identifying and removing toxic plants such as *Conium maculatum* from grazing areas. Economic losses associated with livestock deaths due to poisoning emphasize the need for preventive measures and risk management programs in the livestock sector.

6. **Need for Awareness and Education**

Educating farmers about the risks of toxic plants and implementing management strategies to minimize livestock exposure is essential to prevent similar cases in the future.

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Conflicts of interest

The authors declare that they have no conflict of interest in this research.

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