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## Spontaneous poisoning by *Leucaena leucocephala* in a goat from Rio de Janeiro State, Brazil

### Intoxicação espontânea por *Leucaena leucocephala* em uma cabra, no Rio de Janeiro, Brasil

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#### -NOTA-

#### ABSTRACT

Poisoning of a goat by *Leucaena leucocephala* (Leg. Mimosoideae) in the State of Rio de Janeiro, Brazil, is described. Fresh leaves of the plant were ingested as the animal's main meal for at least 4 months before it developed total alopecia. At necropsy the thyroid was moderately enlarged. Histological examination revealed a decrease in the number of hair follicles (empty or in telogenic phase), vacuolation of keratinocytes of the epidermis and hair follicles, and superficial and follicular hyperkeratosis. Myxedema was found between skeletal and cardiac muscles fibers, in the kidneys, and in the submucosa/muscular layer of the digestive tract. The thyroid follicles were enlarged and filled with copious amounts of colloid (colloidal goiter). Although *L. leucocephala* is common in several tropical regions, natural poisoning by this plant in goats seems to be rare. It is the first report about spontaneous poisoning in goats by *L. leucocephala* in Brazil.

**Key words:** *Leucaena leucocephala*, goat, spontaneous poisoning, mimosine, 3,4-DHP.

#### RESUMO

Este trabalho descreve a intoxicação natural por *Leucaena leucocephala* em um caprino, no município de Maricá, RJ. No ano de 1997, o animal ingeriu voluntariamente as folhas da planta fresca como alimentação quase exclusiva, por pelo menos 4 meses, e, aos poucos, foi perdendo os pêlos até tornar-se totalmente alopecico. À necropsia, a tireóide estava moderadamente aumentada. O exame microscópico revelou decréscimo do número de folículos, além de folículos sem pêlos ou em fase telogênica, vacuolização de queratinócitos da epiderme e de folículos e acentuada

hiperqueratose superficial e folicular. Havia mixedema entre fibras musculares esqueléticas e cardíacas, no rim e na submucosa, e muscular do trato digestório. Os folículos tireoideanos estavam dilatados e preenchidos por grande quantidade de colóide ("bócio coloidal"). Embora essa planta seja freqüente em várias regiões tropicais, a ocorrência natural dessa intoxicação parece bastante rara e ainda não havia sido descrita em caprinos no Brasil.

**Palavras-chave:** *Leucaena leucocephala*, caprino, intoxicação natural, mimosina, 3,4-DHP.

Natural poisoning by *L. leucocephala* (Figure 1A), a leguminous fodder shrub found in tropical regions, has been described in sheep, cattle, pigs and horses (TOKARNIA et al., 2000) in many parts of the world. However, the poisoning does not occur in areas where the animals have the ability to detoxify the toxine 3-hydroxy-4(1H)-pyridone (3,4-DHP), with the help of the Gram-negative bacteria, *Synergistes jonesii*, which resides in the rumen (RINCÓN et al., 1998). Spontaneous poisoning by this plant in goats is not common, as only a textbook chapter describes it (RADOSTITS et al., 2000), but no original scientific report could be found. In Brazil, although the plant is widespread there is only one report of natural poisoning by *L. leucocephala* in sheep (RIET-CORREA et al., 2004). Some experimental studies have also been

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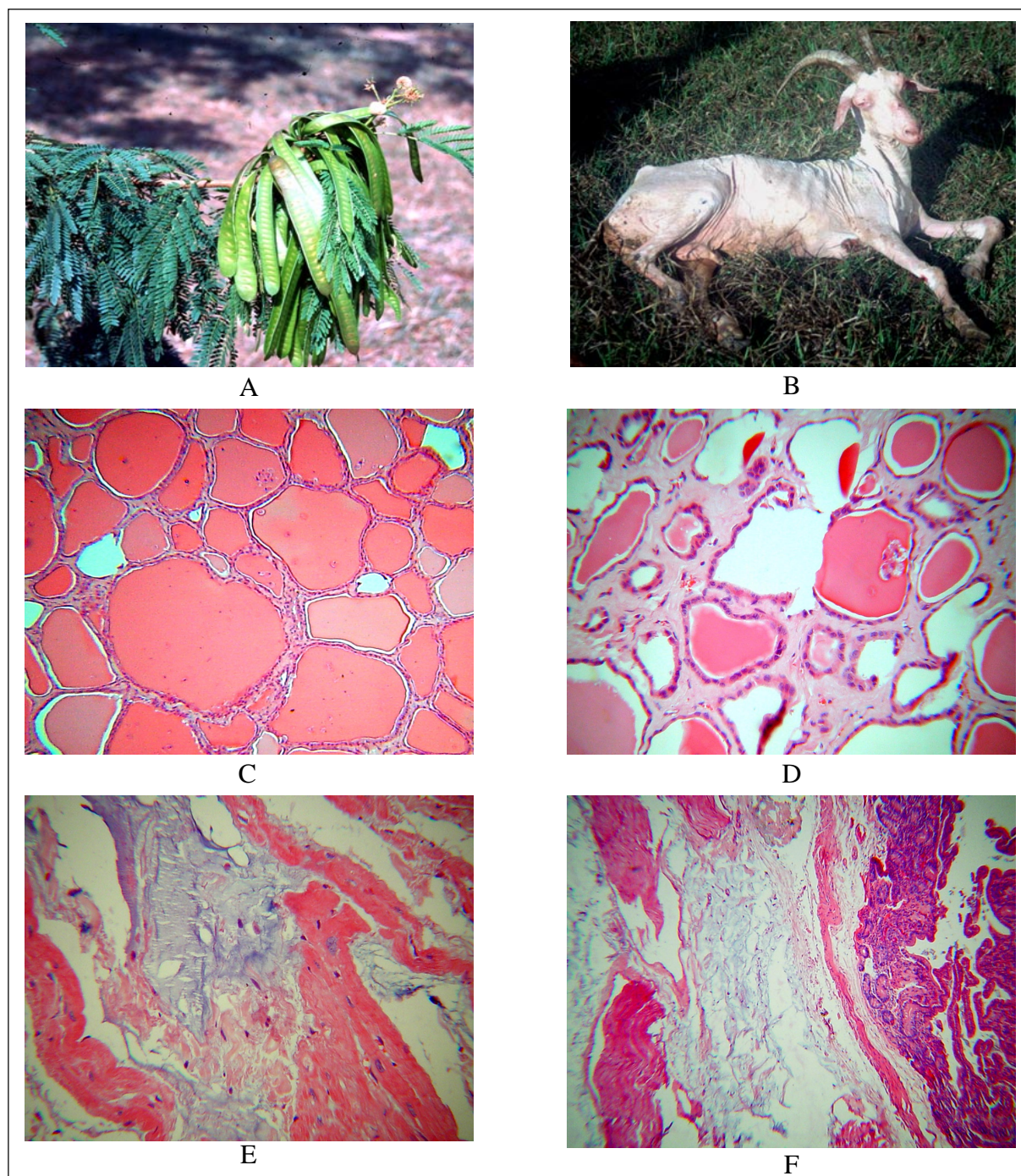


Figure 1 - Spontaneous poisoning in a goat by *Leucaena leucocephala* in Brazil. A. *Leucaena leucocephala* pods. B. Goat with emaciation and complete alopecia. C. Compressive atrophy of the follicular cells and increase of the colloid (colloidal goiter) of thyroid gland, HE, x10. D. Increased amounts of collagen between the follicles of the thyroid gland. HE, x250. E. Myxedema between myocytes of the heart. HE, x160. F. Myxedema between muscle fibers of the small intestine. HE, x160.

reported in sheep (ALMEIDA et al., 2006) and rabbits (MALAFAIA et al., 1994). Outside Brazil, experimental poisoning has been carried out in goats (JONES & MEGARRITY, 1986; MARTINEZ & SEIFERT, 1991;

MEGARRITY & JONES, 1983), cattle (ANDERSON et al., 2001; HOLMES et al., 1981), rabbits (MALINI et al., 1989), birds (JONES, 1985), rats, and mice (HEGARTY et al., 1979). In ruminants that ingest *L. leucocephala*,

the clinical signs can be acute, with loss of hair, particularly in the extremity of the tail, and loss of weight. When the plant is ingested for one to ten months, there are alopecia, loss of appetite and weight, excessive salivation, uncoordinated walk, hypertrophied thyroid, and low levels of circulating thyroid hormones (JONES, 1985; JONES et al., 1978). Hair-loss, reduced fodder uptake, salivation, excitation, ataxia of hindlimbs, vomit, uncoordinated chewing of fodder, swollen buccal papillae, ulceration and necrosis of the oesophagus, and hemorrhagic lesions in the reticulum and rumen have been observed in experimental poisoning in goats (MARTINEZ & SEIFERT, 1991). Swelling of the face in cattle (OTESILE & APPOKODJE, 1986) and mandibular osteodystrophy fibrosa in goats (YATES et al., 1987) were observed in experiments with leucaena. This communication reports an episode of *L. leucocephala* poisoning in a goat and describes its epidemiological, clinical, and pathological aspects.

In August 1997, a 4 year-old Saanen female goat was referred to Anatomic Pathology Section. After performing the routine clinical examination the animal died. The necropsy was carried out immediately after its death and fragments of several organs were collected, fixed in 10% formalin, processed by the usual methods and stained with hematoxylin-eosin (HE). The history and additional information regarding the clinical signs were obtained through a visit to the farm. The animal belonged to a farm located in Maricá County, State of Rio de Janeiro, where 200 goats were raised in extensive management. According to the owner, before the goat became ill with joint lesions, a significant part of its diet was based on leucaena. In May 1997, the animal felt ill, and the owner, aware of its food preference, began feeding it exclusively with this leguminous plant. The hair loss started in May 1997 and the general condition of the goat progressively worsened. Even though the animal was debilitated, it continued to eat leucaena. However, it became anorexic with progressive loss of weight and difficulty to stand up, and recumbent most of the time. At necropsy the goat was emaciated, completely alopecic (Figure 1B) and presented swollen radiocarpal-ulnar joints. The skin was thin, dry and hardened; some thicker and crusty areas were also observed, especially in the resting areas. Additionally, in the subcutaneous, there were gelatinous atrophy of the fat deposits and abscesses close to joints. The thyroid was firmer, somewhat whitish, with a moderate increase in volume and irregular surface. There were a small amount of brown liquid and some linear transversal erosions in the beginning of the ascending colon. The microscopic

changes included atrophy or moderate acanthosis of the epidermis, with formation of rete ridges, marked superficial and follicular hyperkeratosis. There was severe atrophy of the hair follicles, with swelling, vacuolization and lysis of keratinocytes, resting sometimes only the sebaceous glands. Most of the remaining follicles were in telogenic phase. Slight lymphoplasmocytic inflammatory infiltration around the vessels and dermal adnexa, areas of moderate dermal fibrosis, mild to moderate edema, and ectasia of the sudoriparous glands were verified. Discrete to moderate mononuclear inflammatory infiltration in the dermal-epidermal junction and vacuolation of keratinocytes were also observed in the periopic layer of the hoof wall. In the thyroid, there were compressive atrophy of the follicular cells due to increase of the colloid (colloidal goiter) and marked deposition of the collagen between the follicles in some areas (Figure 1C, D). Cardiac (Figure 1E), skeletal, tongue, intestinal wall (Figure 1F), and esophagus muscles had moderate amount of ground substance (myxedema) between muscular fibers. Mucosa of tongue, esophagus, rumen and omasum presented mild to moderate parakeratotic hyperkeratosis. Amyloidosis was seen in the liver, especially in the periportal region, and in splenic follicles. The lymph nodes showed mild to moderate hyperplasia and Russel bodies. There were small amounts of ground substance (myxedema) between the medullary tubules, proteinaceous fluid in the glomerular spaces, convoluted tubules filled with proteinaceous material, and hyaline droplets within tubular epithelial cells.

The diagnosis was based on the diffuse alopecia and on the histopathological alterations, typical for an animal that, for at least 4 months, has been ingesting exclusively *Leucaena leucocephala*. We believe that the colloidal goiter in this goat is due to the action of 3,4-DHP that interferes in the intracellular process of iodine oxidation of the thyroid gland, which results in accumulation of colloid poor in thyroglobulines, decrease of thyroxin (JONES, 1985), myxedema, skin alterations and follicular atrophy. In this case, the hair loss in the initial phase could be associated with the mimosine action. Its depilatory action during the anagenic phase is due to interference in the cysteine synthesis from methionine, by this way reducing the hair protein synthesis that could delay or even interrupt its growth (HYLIN, 1969). The total alopecia observed at the end of the clinical picture may have been determined by the action of 3,4-DHP in the thyroid hormone production.

The differential diagnosis must include mainly other agents capable of inducing diffuse

alopecia and/or hair loss. In Brazil, *Ramaria flavo-brunnescens*, a well known poisonous mushroom that occurs in eucalyptus forests and its ingestion may induce also hair loss, esophageic ulcers, and hoof alterations (TOKARNIA et al., 2000). Given the fact that the studied goat did not have access to eucalyptus forests, and that no evidence of the mushroom was found on the farm, this hypothesis can be disregarded. Similar alterations have been described in chronic selenium poisoning in cattle (O'TOLLE & RAISBECK, 1995). However, the alopecia found in this goat was complete, the animal showed colloidal goiter and myxedema in several tissues, and these alterations do not occur in animals poisoned by selenium. Anyway, the owner was not applying or using selenium based products, and also there were no seleniferous plants at the place. Parakeratosis was found in some areas of the epidermis and digestive tract (tongue, esophagus, rumen and omasum). It is known that hypothyroidism induces to vitamin A deficiency (RIVLIN, 1996). It could be the cause of the parakeratosis verified in this goat. On the other hand, 3,4-DHP could promote zinc deficiency (MEGARRITY & JONES, 1983), disturbance that also causes parakeratosis. However, vitamin A and zinc deficiencies do not cause diffuse alopecia, myxedema and colloidal goiter. Probably, hepatic and splenic amyloidosis, and the lymphoid hyperplasia are associated with chronic infection in joints.

It is believed that the increase in face volume (OTESILE & APPOKODJE, 1986) and fibrous osteodystrophy (YATES et al., 1987), reported by some authors in chronic intoxication by leucaena, are due to undernourishment caused by hyporexia or inadequate supplies of calcium and phosphorus contained in some varieties of leucaena. In the aforementioned case, we did not observe those changes.

The severity of poisoning by *L. leucocephala* is generally associated with the amount of the plant in the diet (JONES et al., 1976). Possibly, if the owner would not have interfered with the feeding of the goat, the clinical signs would have not been so severe. It is important to remember that in Australia diets with less than 30% of leucaena are considered to be safe for ruminants, at least for non pregnant animals (JONES & MEGARRITY, 1986). Prophylactic measures are the destruction of mimosine by heat (above 70°C), the use of chemical additives such as ferrous sulfate, ruminal colonization with the bacteria *Synergistis jonesii*, or utilization of leucaena in smaller proportion (<40%) for the animal diet (FRANZOLIN-NETO & VELLOSO, 1987).

This communication reports the first case of *L. leucocephala* poisoning in goats in Brazil. As the

goat showed colloid goiter, it is possible that the bacteria *Synergistis jonesii* was not present in its rumen.

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