Toxic Plants for Horses in Brazil

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Abstract

This review aims to update the toxic plants that affect horses in Brazil. Currently, it is known that there are at least 131 toxic plants in the country belonging to 79 genera. Pseudocalymma elegans, Baccharis coridifolia, Panicum maximum, Ricinus communis, Prosopis juliflora, Diospyros virginiana, Crotonalaria retusa, Senecio brasiliensis, Trema micrantha, Ateleia glazioviana, Bambusa vulgaris, Coffea canephora, Equisetum spp Hypochaeris radicata, Pteridium aquilinum, Sida carpinifolia, Turbina cordata, Senna occidentalis, Brachiaria spp., Froelichia humboldtiana, Brachiaria humidicola, Leucaena leucocephala, Allium cepa, Crotalaria juncea and Mimosa spp. are the main plants so far that cause poisoning in this species. The incidence of plant poisoning in horses is still low compared with other animals, due to their selective taste, except in cases where the animals are hungry in conditions of malnutrition or accidentally. Therefore, it is important to consider plant intoxication as the differential diagnosis of many other diseases in the country.

Subject Areas
Veterinary Medicine

Keywords
Plant Intoxication, Equines, Clinical Signs

1. Introduction

Toxic plants of interest to livestock have gained great economic importance in recent decades in Brazil. The equine industry has grown by almost 12% per year for the past 10 years. In 2006 it was R $7.5 billion in annual gross revenue and in
In 2015 we reached R $16 billion reais, according to FAO—International Agriculture Federation. Interest in poisoning has grown, mainly due to the economic losses caused by the death of animals, in addition to the losses associated with spending on the construction of fences to isolate areas infested by these plants, decreased weight gain or loss, abortions and lower reproductive rates [1] [2].

In a population of 218.23 million cattle [3], at least 5% (10.911 million) die annually from different causes. Considering data from diagnostic laboratories in different regions, it is estimated that 7.4% and 15.83% of these deaths are related to plant poisoning [1] [4] [5] [6] [7]. Taking these data as a basis, we can estimate that today the losses only with the deaths of cattle due to toxic plants would be approximately between 807,000 and 1,727,000 heads. The horse population in Brazil is 5.58 million heads [3]. Considering mortality similar to that of cattle (5%), it is estimated that 279,000 horses die annually. Considering that 14% of horses die from poisoning by plants [7], it is estimated that approximately 39,000 horses die annually from this cause.

The ingestion of the plants can occur due to several situations, such as thirst, hunger, palatability, sprouting after the first rains and when relocating the animals to unknown pastures. In addition, addiction and socialization among animals can favor ingestion [8]. Plant toxicity can vary between species according to age, race, toxic dose, ingestion period and susceptibility/resistance within the same species (Table 1). Unlike ruminants, horses select carefully what they are going to ingest, preferring initially grasses and legumes, and accidental ingestion of unknown plants may occur. In the cases that will be reported, most of the plants ingested are caused by starvation, due to the lack of forage.

In view of the economic importance of toxic plants in Brazil and the growth of equine culture in the country, this work aims to update data on the main epidemiological, clinical, pathological aspects and ways of controlling plant poisoning in this species.

2. Revised Literature

2.1. Plants Affecting Heart’s Functioning

*Pseudocalymma elegans*

It is a vine plant of the Bignoniaceae family and was one of the first toxic plants studied in Brazil. It is found in the state of Rio de Janeiro and has a hillside natural habitat. Its toxic principle is not yet known; however, it is suspected that it is monofluoroacetic acid. The horses affected by the intoxication of the plant had rigid walking, instability, muscle tremors, sternal de cubitus, opisthotonus, nystagmus and tachycardia. Necropsy findings are practically negative, but jugular, vena cava and other veins in the thoracic and abdominal cavities may be markedly engorged, marked dilation of the cardiac chambers, pulmonary edema, mild hepatic and renal congestion. In the histopathological evaluation, mild alterations are observed, but it can be found edematous-vascular degeneration associated with nuclear pycnosis of the epithelial cells of the distal outlined...
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<td>Cardiac system</td>
<td><em>Pseudocalymma elegans</em></td>
<td>rigid walking, instability, muscle tremors, sternal decubitus, opisthotonus, nystagmus and tachycardia</td>
</tr>
<tr>
<td></td>
<td><em>Baccharis coridifolia</em></td>
<td>abdominal discomfort, increased heart rate and respiratory movements, anorexia, hypermotility of the small intestine and colon, presence of gas in the cecum and diarrhea</td>
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<tr>
<td></td>
<td><em>Panicum maximum</em></td>
<td>acute abdomen (colic), apathy, congested and in the final stages, cyanotic mucous membranes, increased capillary perfusion time, heart and respiratory rate enlarged, look at the flank, lie down and get up, roll on the floor, bilateral dilation of the abdomen, stop intestinal peristalsis, presence of large amounts of fluid in the small intestine and in the final stage, gastric reflux can be observed</td>
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<tr>
<td>Digestive system</td>
<td><em>Ricinus communis</em></td>
<td>inappetence/anorexia, fever, dyspnea, apathy, general malaise, tendency to keep the head supported, soft and dark feces, mucous-bloody diarrhea, abdominal pain, changes in the electrocardiogram, weak pulse and strong heartbeat, hydroelectrolytic disorders, oliguria, nephritis and renal failure, cyanosis and death from cardiorespiratory collapse</td>
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<tr>
<td></td>
<td><em>Prosopis juliflora</em></td>
<td>intestinal obstruction in horses</td>
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<td></td>
<td><em>Diospyros virginiana</em></td>
<td>damage the gastric mucosa causing ulcers that can lead to organ rupture</td>
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<td>Liver system</td>
<td><em>Crotalaria retusa</em></td>
<td>apathy or hyperexcitability, pressure of the head against objects, compulsive gait, uncontrolled and circular canter, decreased reflexes consistent with cranial nerves, ataxia, weakness, anorexia, weight loss, photosensitization and jaundice</td>
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<tr>
<td></td>
<td><em>Senecio brasiliensis</em></td>
<td>apathy, anorexia, weight loss, dehydration and other neurological signs, developing into a picture of hepatic encephalopathy, and there may also be edema in the subcutaneous tissue, in the ventral region of the abdomen, jaundice in the ocular, oral and vulvar mucous membranes</td>
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<td></td>
<td><em>Trema micrantha</em></td>
<td>muscle tremors, sternal and lateral decubitus, pedaling movements, dysphagia, sialorrhea, and subject their own heads against objects by pressing them</td>
</tr>
<tr>
<td>Reproductive system</td>
<td><em>Ateleia glazioviana</em></td>
<td>It causes abortion in cattle, sheep, goats and horses and is probably the main cause of abortions in cattle in these regions</td>
</tr>
<tr>
<td>Neurological system</td>
<td><em>Bambusa vulgaris</em></td>
<td>motor incoordination, limbs crossed during gait, drowsiness, difficulty in apprehension, chewing and swallowing of food with accumulation of grass in the oral cavity and instability and permanence in station with abducted limbs</td>
</tr>
<tr>
<td></td>
<td><em>Coffea canephora</em></td>
<td>cardiorespiratory and neurological changes (convulsions, tachycardia, tachypnea), which can progress to coma and death</td>
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<tr>
<td></td>
<td><em>Equisetum spp.</em></td>
<td>Muscle tremors, falls, weakness of the hind limbs, ataxia and muscle exhaustion</td>
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<td></td>
<td><em>Hypochaeris radicata</em></td>
<td>abnormal gait, with involuntary hyperflexion of the pelvic limbs, difficulty walking in circles and back, there is an association with left laryngeal hemiplegia, muscular atrophy of the pelvic limbs</td>
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<table>
<thead>
<tr>
<th>Plant</th>
<th>Symptoms</th>
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<tr>
<td><em>Pteridium aquilinum</em></td>
<td>anorexia, incoordination of pelvic limbs, staggering, distant or crossed</td>
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<td></td>
<td>limbs, muscle tremors, loss of balance, decubitus and death</td>
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<tr>
<td><em>Sida carpinifolia</em></td>
<td>progressive weight loss, incoordination, rigid walking and ambulation,</td>
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<td>in addition to difficulty in locomotion with exacerbated reactions after</td>
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<td>movement stimuli</td>
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<td><em>Turbina cordata</em></td>
<td>nervous origin, including tremors, severe ataxia, station with open limbs,</td>
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<td></td>
<td>changes in behavior and weight loss and death</td>
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</tbody>
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| Muscle disorders       | *Senna occidentalis*                                                      |
|                       | apathy, muscle tremors, motor incoordination, staggering walking,          |
|                       | tachycardia and dyspnea                                                   |

| Photosentization       | *Brachiaria spp.*                                                          |
|                       | lesions on the parts with white fur, specifically on the nostrils, eyelids,|
|                       | extremities of the limbs and chamfer                                       |

| *Froelichia humboldtiana* | itching, weakness and death. In the clinical examination it is possible to |
|                         | observe extensive, ulcerated wounds, which drain abundant serous exudate  |

| Integumentary system   | *Leucaena leucocephala*                                                   |
|                       | alopecia in the tail and mane, anorexia, weight loss and apathy            |

| Hematological disorders| *Allium cepa*                                                              |
|                       | lack of appetite, wobbly walking, abortion, breath, feces and urine with   |
|                       | an onion odor, increased heart and respiratory rate, pale to jaundiced     |
|                       | mucous membranes and brown urine                                          |

| Respiratory system     | *Crotalaria juncea*                                                        |
|                       | ulcerative characteristics and irregular contours, hemorrhagic exudate,    |
|                       | are covered by crusts, located in the region of the rib cage, billet,      |
|                       | scapulohumeral joint, upper and lower lip, muzzle, nostrils, cheeks and    |
|                       | chamfer                                                                    |

| Traumatic injuries     | *Mimosa spp.*                                                              |
|                       | Traumatic angular cheilitis                                                |

| Traumatic injuries     | *Panicum maximum*                                                          |
|                       | Traumatic angular cheilitis                                                |

tubules, cytoplasmic vacuolization in the liver and myocardium. The diagnosis takes the most important aspect into consideration, which is “sudden death”. Microscopic damage to the kidney, when present, is of great diagnostic value. There is no known treatment for this intoxication. It is recommended to move the animals as little as possible when poisoning is suspected [8] [9].

2.2. Plants Affecting the Digestive System

*Baccharis coridifolia*

It is a sub-shrub of the Asteraceae family and is one of the most common and important plants in southern Brazil, popularly known as “mio-mio”. Its active ingredient is macrocyclic trichotheccenes. There are reports of an outbreak of natural intoxication in July 2008 in three of four PSI horses that had been transported from the state of Paraná to Rio Grande do Sul. The clinical signs were initially characterized by abdominal discomfort, increased heart rate and respiratory movements, anorexia, hypermotility of the small intestine and colon, presence of gas in the cecum and diarrhea. The evolution of the disease ranged from 18 - 36 hours, and one of the affected animals survived after symptomatic treat-
ment. Macroscopic lesions were characterized by marked congestion and hemorrhage from the glandular mucosa of the stomach and the presence of ulcers and marked edema of the wall. There was also congestion, edema, and hemorrhages in the mucous membranes of the ileum, cecum and larger colon. Histologically, degeneration and necrosis of the epithelium of the without glandula portion of the stomach, gastritis and enteritis with necrosis of the lining epithelium of the glandular stomach, ileum, cecum and colon were observed, with a marked infiltration of mononuclear and polymorphonuclear cells, swelling of the submucosa, and dilatation of lymphatic vessels [10]. An experimentally intoxicated horse with 1 g/kg body weight of green plant showed clinical signs, macroscopic and histological lesions similar to spontaneous cases. At the beginning of the clinical signs after the beginning of the experimental administration of the plant, it varied between 4 h 05 min and 12 h 50 min. The clinical evolution in these animals ranged between 11:45 am and 26:15 am. The period between ingestion of the plant and death was 15 h 50 min to 39 h 05 min [11]. According to experiments, the first clinical signs were observed 3 hrs after the plant administration, and the evolution was just over 15 hrs [10]. Necropsy findings are dilation with the presence of large amounts of fluid and mucus in the jejunum, cecum and colon, mucosal congestion, ulcers and edema in the glandular portion of the stomach [11]. It is important to determine the diagnosis to verify if the animal resides in regions where the plant does not vegetate. As a treatment, the use of activated carbon is recommended [8].

**Panicum maximum**

This grass used for forage is found mainly in the Amazon region, but it is widespread throughout Brazil. Its toxic principle is still unknown, and the horses that undergo its ingestion end up presenting a clinical picture of acute abdomen (colic), apathy, congested and in the final stages, cyanotic mucous membranes, increased capillary perfusion time, heart and respiratory rate enlarged, look at the flank, lie down and get up, roll on the floor, bilateral dilation of the abdomen, stop intestinal peristalsis, presence of large amounts of fluid in the small intestine and in the final stage, gastric reflux can be observed [12]. The necropsy findings are the dilated stomach with a large amount of fluid, diffuse and reddish glandular mucosa of the stomach with the presence of ulcers, dilated small intestine with the presence of liquid and reddish mucosa, large intestine with dry feces and soaked blood, mucous with areas necrotic, and liver with orange areas and noticeable lobular pattern. In histopathology there is a marked swelling of hepatocytes with a vitreous aspect, groups of macrophages distributed by the parenchyma, binucleated hepatocytes, megalocytosis and hepatocyte necrosis, bile duct hyperplasia, portal space edema, inflammatory cell infiltration, in the intestine it is noticeable inflammation predominantly eosinophilic in mucosa and submucosa, congestion and hemorrhage, with necrosis points. A mild tubular nephrosis and coagulative necrosis in epithelial cells in some tubules can be seen at the renal level [12]. It was also reported that horses in pastures that con-
tained this forage developed fibrous osteodystrophy, due to the high levels of oxalate in the plant [13] [14]. It is noticeable at necropsy the presence of kidney stones, urethral stones and also bladder stones [15] [16].

*Ricinus communis*

It is popularly known as “castor bean”. They are seeds easily found throughout the Brazilian territory, and their known toxic principle is Ricina [8]. It is important to note that the shrub of the Euphorbiaceae family has toxic principles that act differently according to the digested location. The seeds contain ricin and the substance in this part, mainly affects the digestive tract. The ricin present in the leaves and in the pericarp, on the other hand, can cause clinical signs of neurological disturbance (this has not yet been reported in horses). In experimental studies it has been shown to be toxic to horses at a dose of 0.1 g/kg [8]. In histopathological changes, a picture of wide cell disintegration is described, particularly in lymphoid cells [17].

The animals can present inappetence/anorexia, fever, dyspnea, apathy, general malaise [18] [20] [21], tendency to keep the head supported, soft and dark feces [18], mucous-bloodly diarrhea [19] [20] [21] [22] [23], abdominal pain, changes in the electrocardiogram, weak pulse and strong heartbeat, hydroelectrolytic disorders, oliguria, nephritis and renal failure, cyanosis and death from cardiorespiratory collapse. Prolonged contact can cause conjunctivitis, runny nose, dermatitis, eczema, and inhalation can cause asthma [22] [23]. In laboratory tests, there may be the presence of proteins, cylinders and hemoglobin in the urine, in addition to an increase in urea and non-protein nitrogen in the serum [22]. At necropsy and histopathological examination, hemorrhagic gastroenteritis can be found with edema, congestion and coagulation necrosis of the mucosal epithelium of the gastrointestinal tract, mainly of the small intestine and cecum [18] [19] [21]; presence of fibrin in flakes or filaments in the intestinal contents and/or covering the mucosa (pseudomembranes); congested mesenteric lymph nodes [18]; inflammation and edema of ileum and Peyer’s plaques [21]; pulmonary edema [18]; congestion and atrophy of lymphoid follicles in the spleen [19] and degenerative changes in the liver and kidney [23]. At necropsy, the mucosa of the small and large intestine is reddish and rectum full of mucous and hemorrhagic content [24].

**2.3. Plants That Cause Mechanical Changes in the Digestive System**

*Prosopis juliflora*

Ingestion of large quantities of pods of *Prosopis glandulosa* causes intestinal obstruction in horses [25]. Ruminal compaction has not been observed in ruminants consuming pods of *Prosopis juliflora* (mesquite) in the Northeast, however, horses fed in mesquite invaded areas may develop intestinal obstruction caused by the presence of indigestible masses formed by the pods of the plant (phytobezoaries) [7].
The ingestion of *Diospyros virginiana* (persimmon), in horses, forms phyto-o-benzoaries in the stomach that damage the gastric mucosa causing ulcers that can lead to organ rupture [25]. In Brazil, although this plant is cultivated, there is no description of similar cases. Ingestion of *Pennisetum purpureum* (napier grass, elephant grass) can cause greater colon compaction in horses [25] [26].

It is important to note that there are factors that can favor equine colic or acute abdomen syndrome. The decrease or variations in the level of physical activity, sudden changes in diet, changes in housing conditions, a diet rich in concentrates, a roughage or poor quality food, excessively fast consumption of the food, water deprivation and even transportation when traveling. The horse is more demanding and sensitive to changes in food and environmental management than other species. Therefore, sudden changes in food, with any type of roughage or concentrate, may favor gastrointestinal disorders [27].

### 2.4. Plants Affecting Liver

**Crotalaria retusa**

It is a legume, found in the Northeast region of Brazil, popularly known as “rattle-rattle”. It has good palatability for horses, and cases of spontaneous intoxication, mainly in chronic form, have been reported in horses. From 2000 to 2003, eight cases of poisoning by *Crotalaria retusa* were observed in horses from 8 farms in the semi-arid region of Paraiba and Ceará, and *C. retusa* was found in pasture in all properties. Its toxic principle is monocrotaline, and it can lead the animal to present as clinical signs apathy or hyperexcitability, pressure of the head against objects, compulsive gait, uncontrolled and circular canter, decreased reflexes consistent with cranial nerves, ataxia, weakness, anorexia, weight loss, photosensitization and jaundice. The necropsy findings were liver with an irregular and hard surface, white and red areas, with marked lobular pattern, jaundice, ascites, hydropericardium, hydrothorax, edema and pulmonary congestion, in histopathological findings the liver presented with areas of fibrosis in the periportal region, megalocytosis and proliferation of cells in the bile ducts, multifocal hemorrhagic areas in the center of the lobes, hemorrhagic centrilobular necrosis, and type 2 Alzheimer astrocytes can be found in the caudate nucleus and cortex [28] [29].

**Senecio brasiliensis**

It is a plant characterized by its yellow flower found in the field next to the pastures, popularly known as “maria-mole”. Its toxic principle is the pyrrolizidine alkaloid and there are reports of spontaneous intoxication in horses in the cities of São Paulo [30] and Paraná [31], for example, in which the clinical signs are apathy, anorexia, weight loss, dehydration and other neurological signs, developing into a picture of hepatic encephalopathy, and there may also be edema in the subcutaneous tissue, in the ventral region of the abdomen, jaundice in the ocular, oral and vulvar mucous membranes. Necropsy findings are enlarged, dark and firm liver. Histologically, there is coagulative necrosis, hemorrhage and
fibrosis, proliferation of bile ducts, hepatomegaly, and bile stasis. In the brain, degenerated astrocytes are noted [32] [33].

**Trema micranta**

Arboreal plant was found in southern Brazil. Its toxic principle has not yet been elucidated in horses. However, after ingestion, it is possible to notice that they develop neurological clinical signs such as muscle tremors, sternal and lateral decubitus, pedaling movements, dysphagia, salivation, and subject their own heads against objects by pressing them. The signs usually appeared three to four days after the consumption of the plant, with a clinical course of two to four days (abbreviated by euthanasia). The main necropsy findings were observed in the liver, which was slightly enlarged and with an evident lobular pattern, and in the central nervous system (CNS), flattening of the telencephalic girdles was observed, multiple yellow areas in the white matter of the cerebellum and brain stem, foci of malacia and hemorrhage, mainly in the brainstem and cerebellum [34]. There are also reports of bruises on the face [34]. Histologically, there was acute liver necrosis, marked edema in the CNS with fibrinoid degeneration of the vessel wall associated with hemorrhage and thrombosis and, frequently, with neutrophil infiltration. Other changes observed in the brain were: large amounts of Alzheimer’s type II astrocytes in the gray matter, accumulations of Gitter cells and Wallerian degeneration close to foci of vascular lesions [34]. Very similar to the report by Pavarini et al. 2013, histopathological changes are centrilobular hepatic necrosis, perivascular edema, perivascular hemorrhage, fibrinoid degeneration, characterizing encephalopathy [34].

2.5. Plants That Cause Abortion

**Ateleia glazioviana**

The Ateleia glazioviana tree is frequently found in the western region of Santa Catarina and northwest of Rio Grande do Sul. It causes abortion in cattle, sheep, goats and horses and is probably the main cause of abortions in cattle in these regions [35]. Abortions occur at any stage of pregnancy and there are no specific injuries to the fetuses [36]. Intoxication is diagnosed through the presence of the plant and the negative results of other causes of abortion, mainly equine Herpesvirus-1. An outbreak of this intoxication, causing abortions in horses, occurred in the city of São José do Ouro, Rio Grande do Sul, in May 1994 [37].

The best form of prophylaxis for intoxication is not to place pregnant mares in potters where the plant is. It is not recommended to cut the trees, because sprouts are more easily ingested by animals. You can eradicate the plant using herbicides or uprooting trees with roots [36].

2.6. Plants Affecting Neurological System

**Bambusa vulgaris**

Bamboo species found in the northeastern region of Pará, and spontaneous intoxication in horses is reported. Its toxic principle is still unknown. Clinical signs are motor incoordination, limbs crossed during gait, drowsiness, difficulty
in apprehension, chewing and swallowing of food with accumulation of grass in
the oral cavity and instability and permanence in station with abducted limbs.
The clinical course varied from subacute to chronic, with no lethal success in
most cases. Of 16 horses observed, only two died. The animals show clinical
signs while ingesting it, after ingestion, they recover in two to three days [8]. The
necropsy findings were not conclusive, and in the histopathological evaluation,
degenerative changes of axons were observed with formation of digestion
chambers in the oblong medulla [38].

**Coffea canephora**

Better known in Brazil as coffee, *Coffea canephora* is a plant widely used for
commercialization. Coffee production in Brazil is responsible for about one
third of the world’s coffee production [39]. Coffee poisoning can occur by acci-
dental ingestion of products containing caffeine, and also by direct ingestion of
grains. There are some reports of dogs that have cardiorespiratory and neuro-
logical changes (convulsions, tachycardia, tachypnea), which can progress to
coma and death. In horses, some cases of accidental poisoning have been re-
ported in Espirito Santo, in the municipality of São Mateus. In some properties
in Espirito Santo, it is common to use coffee by-products as husks, as bedding
material for horses, in order to replace sand or wood in stalls, especially in places
where there is cultivation of the plant and associated horses. The diagnosis was
based on the epidemiological and clinical characteristics of the disease. The main
clinical signs in horses are excitability, agitation, involuntary muscle tremors,
chewing movements, constant tremors on the lips and tongue, sweating, ta-
chypnea and tachycardia due to the effects of caffeine on the circulation, which
can progress to death [40].

**Equisetum spp.**

Poisoning by *Equisetum spp.* (horsetail) was diagnosed in the municipality of
Catanduva, São Paulo, and in the Mineiro Triangle; in the latter associated with
the intake of *E. giganteum*. Ingestion occurs during the dry season, due to the
plant remaining green, or due to the ingestion of contaminated hay [41]. Its tox-
ic principle is thiaminase, and how much consumption by horses can cause cli-
nical signs such as: Muscle tremors, falls, weakness of the hind limbs, ataxia and
muscle exhaustion [42].

Some reports mention that after a few weeks of ingesting the plant, the first
clinical signs can be observed, characterized by weight loss and nervous signs.
Depending on the animal’s age and the amount of plant ingested, in approx-
imately 30 days, there is loss of muscle control and staggering walking. Subse-
sequently, the horse is unable to stand, and death occurs as a result of weight loss
[41]. No specific macroscopic and histological lesions were found to be de-
scribed for this disease.

**Hypochaeris radicata**

It is a plant found in southern Brazil, in native pastures. Its toxic principle is
not known, it is called as spontaneous intoxication, and its consumption is re-
lated to the harp. The clinical signs are abnormal gait, with involuntary hyper-
flexion of the pelvic limbs, difficulty walking in circles and back, there is an association with left laryngeal hemiplegia, muscular atrophy of the pelvic limbs. It is called an axoniopathy, not showing macroscopic lesions at necropsy. In histopathological findings there is axonal degeneration in peripheral nerves and congenital muscular atrophy, demyelination, regeneration and remyelination of peripheral nerves is also found [43] [44].

*Pteridium aquilinum*

It is an ornamental plant, popularly known as “fern”, found throughout Brazil. Its toxic principle is thiaminase type I. Spontaneous poisoning in horses has been reported. It appears to be like intoxication by *Equisetum* spp.

The clinical signs are anorexia, incoordination of pelvic limbs, staggering, distant or crossed limbs, muscle tremors, loss of balance, decubitus and death. Evolution can take up to 48 hours [45]. The necropsy findings are not conclusive, and do not reveal changes consistent with the disease. There are no reported reports of macroscopic or histological changes of significance. The indicated treatment is the daily administration of 100 mg of thiamine, however, when the animal is in the final stage of the disease, this treatment may not be efficient [41] [45].

*Sida carpinifolia*

It is a shrub plant that, when ingested, causes lysosomal accumulation because it contains an indolizidine alkaloid called swainsonine. The plant can be found in Rio Grande do Sul, and when an horse ingests large quantities, it can present a clinical picture of progressive weight loss, incoordination, rigid walking and ambulation, in addition to difficulty in locomotion with exacerbated reactions after movement stimuli, with evolution 90 days and death. At necropsy, no macroscopic lesions were found. Histological findings in the central nervous system were an increase in the size of neurons, with a cytoplasm containing microvacuoles, these changes were observed in the region of the thalamus, hippocampus, cerebellum and bridge [46].

*Turbina cordata*

It is a plant of Brazilian origin, until then with few reported cases of natural intoxication in horses in Brazil. In Bahia, two outbreaks of *Turbine cordata* poisoning occurred in goats and one in horses. Clinical signs were predominantly of nervous origin, including tremors, severe ataxia, station with open limbs, changes in behavior and weight loss and death. There was a large amount of the plant on the farm. The diagnosis was made through clinical and epidemiological data [47].

2.7. Plants That Cause Muscle Disorders

*Senna occidentalis*

Distributed in tropical regions, intoxication occurs by accidental ingestion of seeds in the feed. Its toxic principle has not yet been found. Clinical signs were apathy, muscle tremors, motor incoordination, staggering walking, tachycardia and dyspnea [48]. Necropsy findings were jaundice, nutmeg-like liver, renal
cortex congestion and there were no changes in the muscles [49]. In histopathological findings, there are liver and muscle lesions in the form of degeneration and tissue death, in the muscles there are tumefied, hyalinized, tortuous and fragmented fibers, with segmental hypercontraction [48].

2.8. Plants That Cause Photosensitization

**Brachiaria spp.**

African grass introduced in Brazil, with the purpose of pasturing. Its toxic principle is saponin and sapogenins. Clinical signs are lesions on the parts with white fur, specifically on the nostrils, eyelids, extremities of the limbs and chamfer. The lesions have exudative characteristics and with crusts, there may be pigmented areas also affected, such as rump, neck and nostrils, and necrosis and separation cracks between the skin and the horny layer of the hooves may occur, the lesions may have an ulcerated character, with irregular edges and with granulating tissue. Progressive weight loss and death are noted in horses [50] [51]. Necropsy findings are mainly generalized jaundice, and liver with a greenish surface. Histopathological findings indicate a swelling of hepatocytes, which look like plant cells, in the liver there are changes in their cell morphology, such as megalocytosis, and several multinucleate hepatocytes with anisocariosis. There is coagulative necrosis of isolated hepatocytes giving rise to the digestion chambers, with spaces filled by fibroblasts and collagen deposition, it is possible to observe different degrees of biliary retention. In the skin, the formation of acanthosis and multiplication of fibroblasts and collagen deposits in the dermis are notable. In more advanced cases, edema and fibrosis of the dermis can be seen, with dilated sweat glands proliferating blood vessels [51].

**Froelichia humboldtiana**

It is a plant that causes primary photosensitization and can be found in the Northeast of Brazil. Primary photosensitization occurs when the pigment of the ingested plant ends up passing through after intestinal absorption and reaches the skin that ends up becoming more sensitive when it encounters solar radiation. In secondary photosensitization, the plant ends up damaging the liver, which consequently fails to eliminate phylloerythrin, which ends up passing into the bloodstream and deposits on the skin, which is sensitive to sunlight. In the case of *Froelichia humboldtiana*, its toxic principle is naphtodiantrona [1].

In cases of ingestion of the plant, horses may present as clinical signs, itching, weakness and death. In the clinical examination it is possible to observe extensive, ulcerated wounds, which drain abundant serous exudate. All wounds are due to self-mutilation secondary to intense itching. Photodermatitis lesions have only been reported in depigmented skin areas. In histopathological findings, perivascular inflammation can be seen in the superficial dermis, and in the epidermis there were extensive ulcers covered by fibrin associated with the superficial neutrophilic infiltrate [52].

**Brachiaria humidicola**

Ingestion of *Brachiaria* harms the liver as there are changes in nuclear mor-
phology, with megalocytosis, numerous bi- or trinucleated hepatocytes or with evident anisokaryosis (bizarre or sometimes wrinkled nuclei, with indented, irregular, hyperchromatic and evident nucleoli) in the parenchyma there was coagulative necrosis or lysis of isolated hepatocytes or small groups, whose remains, in other areas, were phagocyted by macrophages, giving rise to the formation of small “digestion chambers” filled, in addition, by brown pigment. In the skin of a horse that developed subacute disease, there was marked acanthosis with formation of “rete ridges”, accentuated multiplication of fibroblasts with collagen deposition in the dermis, accompanied by proliferation of small vessels, incontinence of pigment in areas of necrosis and ulceration of the skin. In one of the chronic cases, edema and fibrosis of the dermis, dilatation of sweat glands and exuberant vascular proliferation were observed [51].

### 2.9. Plants Affecting Skin

**Leucaena leucocephala**

It is a tree of good palatability, found in São Paulo and Goiás. Its toxic principle is mimosina. Clinical signs are alopecia in the tail and mane, anorexia, weight loss and apathy. In the skin biopsy, it was possible to notice a marked telogenization of the hair follicles, and in the serum dosage of triiodothyronine (t3) and thyroxine (t4), a decrease in hormone levels was found, suggesting a bociogenic effect of mimosina and its derivative compounds [53].

### 2.10. Plants That Cause Hematological Disorders

**Allium cepa**

Popularly known as onion, it is widespread throughout the world. In experimental studies it is toxic to horses. Its toxic principle is n-propyl disulfides which cause an oxidation in the erythrocytes. Clinical signs are lack of appetite, wobbly walking, abortion, breath, feces and urine with an onion odor, increased heart and respiratory rate, pale to jaundiced mucous membranes and brown urine. Necropsy findings were jaundiced cadaver, brown urine. Histopathological findings show degeneration in hyaline drops in the kidneys located in the tubular epithelium, and moderate to multifocal centrilobular coagulation necrosis in the liver [54].

### 2.11. Plants Affecting the Respiratory System

**Crotalaria juncea**

It is a legume used as fertilizer in Minas Gerais. Its toxic principle has yet to be elucidated. The clinical signs are trembling, dyspnoea, fever and death. The necropsy findings were edema, pulmonary congestion and consolidation of diffuse areas of the lung parenchyma, and the liver is enlarged and congested. Histopathological findings prove a diffuse fibrous alveolitis in the lung, alveolar septa thickened by interstitial edema, infiltration of inflammatory cells, congested liver and hepatic trabeculae with compression [55].
2.12. Plants That Cause Traumatic Injuries

*Mimosa spp.*

It is a plant with a thorny character, found in Rio de Janeiro. The lesions present ulcerative characteristics and irregular contours, hemorrhagic exudate, are covered by crusts, located in the region of the rib cage, billet, scapulohumeral joint, upper and lower lip, muzzle, nostrils, cheeks and chamfer. In the skin biopsy, the histopathology showed ulceration of the epidermis and inflammatory infiltrate, consisting of macrophages and neutrophils, delimited by underlying granulation tissue, also microspheres of Hirsute Trichomes in the inflammatory area [56].

*Panicum maximum*

Acute intestinal bloat in horses and mules introduced to *Panicum maximum* pastures has been more frequent in rainy seasons, it has been suggested that they are associated with a greater storage of non-fibrous carbohydrates in the grass [57]. Traumatic angular cheilitis in horses has been reported to be associated with the ingestion of Panicum Maximum in the states of Maranhão and Pará [12].

3. Conclusion

The incidence of plant poisoning in horses is still low compared with other animals of interest in production. Horses are more selective animals and with a more demanding taste, intoxication ends up not being recurrent, except in cases where the animals are hungry in conditions of malnutrition or accidentally. It is noticed that some plants are directly connected with some clinical syndromes in horses, such as acute abdominal disease. Therefore, it is important to consider plant intoxication as the differential diagnosis of many other diseases in the country.

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Conflicts of Interest

The authors declare no conflicts of interest.

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