ABSTRACT
The clinical and pathological aspects of a neurological disease observed in 16 horses in Pará, Amazonia, Brazil, are presented. The symptoms were mainly motor incoordination, paresis of the tongue, somnolence, difficulties in apprehension, chewing and swallowing of food, as well as instability and standing with abducted members. The clinical course was subacute or chronic and in most cases was not fatal. Postmortem examination performed in one already very sick, euthanized animal, did not show significant macroscopic lesions; histopathological examination revealed slight edema and degenerative alterations of a few axons, mainly in the medulla oblongata. In all pastures where horses were affected, plenty of bamboo had been eaten, probably because of scarcity of pasture. By feeding large amounts of fresh bamboo leaves of this region, in different growing stages, to three horses (horse 1, 47 g/kg/d for 30 days; horse 2, 10 g/kg/d for 60 days; horse 3, 18 g/kg on the first day, and 31 g/kg/d for 6 more days)—the animals ate the leaves unassisted—it was possible to reproduce nervous symptoms essentially identical to those observed in the natural disease 24 to 72 hours after the first feeding of the plant. In spite of continuous administration of the plant, intensity of the clinical signs did not increase. Based on field observations and comparison of the clinical and pathological pictures seen in the natural and experimental disease, the described illness can be concluded to be caused by the ingestion of large amounts of the leaves of Bambusa vulgaris f. vulgaris.

Key words: Encephalopathy; horses; bamboo; poisoning; Bambusa vulgaris f. vulgaris

INTRODUCTION
In the Amazon Region of northeastern Pará, a disease of horses, characterized by neurological manifestations of variable evolution, usually nonfatal, has been observed. Serological examinations for equine encephalomyelitis and equine protozoal myeloencephalopathy, and blood smears for hemoprotozoal research, performed in some animals, gave negative results. Poisoning by Sida carpinifolia, a plant that occurs practically all over the region, and which is capable of producing a nervous disease by storage of mannose in the central nervous system of horses, cattle and sheep, was initially suspected.1–4 However, an experimental feeding of approximately 10 kg of the fresh plant to a horse for 30 days did not produce any symptoms. A more detailed observation of all pastures where the disease occurred showed large amounts of bamboo being eaten by the horses. Three horses were fed bamboo leaves in an attempt to reproduce the disease. Additionally, several horses affected naturally by the illness were examined clinically, and one of them was killed for histopathological studies.

The objective of the current study was to demonstrate that ingestion of Bambusa vulgaris f. vulgaris is responsible for the neurological disease that occurs in the above-mentioned region.

MATERIALS AND METHODS

Natural Cases
In 16 cases of natural poisoning included in this study, clinical and other data were obtained in visits to five properties located in the counties of Castanhal and São Francisco do Pará, PA, made between 1998 and 2005 (Table 1). On those occasions, a general examination and specific examinations of the nervous system were made. One horse was killed and a postmortem examination was done (N8), with collection of fragments
of several organs and of the central nervous system for histopathological studies in the Pathology Section of the Embrapa/UFRRJ Animal Health Project. The material was collected in 10% formalin and processed by routine methods, set in paraffin, cut 5 μm thick, and stained by hematoxylin-eosin (HE).

Experimentation

The experiments were performed at Fazenda Curicaca, where fresh leaves of bamboo in various stages of maturation, collected in the county of Castanhal, were administered to three horses of undefined breed (E1, female, weighing 318 kg, 7 years old; E2, female, weighing 285 kg, 2 years old; E3, male, weighing 320 kg, 3 years old). The plant was identified as Bambusa vulgaris f. vulgaris, family Poaceae, subfamily Bambusoideae (Fig 1).

The plant was collected daily and offered to the animals, who ingested it spontaneously. Equine E1 received 15 kg (47 g/kg) of the mature leaves of bamboo daily for 30 days, equine E2 received 3 kg (10 g/kg) of the young leaves (sprouts) daily for 60 days, and equine E3 received 6 kg (18 g/kg) on the first day and 10 kg (31 g/kg)/d on 6 subsequent days of the mature leaves of bamboo.

The animals were examined daily for the following parameters: behavior, appetite, posture, appearance of the feces and urine, coloration of the mucous membranes, intestinal peristalsis, heart and respiratory rates, cutaneous turgor, and rectal temperature. In addition, the three experimental horses were submitted to specific examinations of the nervous system.

Equines E1 and E2 were killed and a postmortem examination performed at the end of the experimental period of 30 and 60 days, respectively. Fragments of sev-
eral organs and of the central nervous system were treated similarly to methods described for the cases of natural poisoning.

Detection of Cyanogenic Glycosides

The young leaves (sprouts) and mature leaves of Bambusa vulgaris f. vulgaris of three farms of the county of Castanhal, PA, were tested for the presence of hydrocyanic acid by the sodium picrate test.\(^5\)

RESULTS

Natural Cases

Natural cases occurred in properties in the counties of Castanhal and San Francisco, both located in the northeastern region of the State of Pará. In the five properties where horses affected by the disease were examined, the pastures were composed mainly of Brachiaria brizantha and Brachiaria humidicola. In the

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**Table 1 (continued)** Poisoning of horses by *Bambusa vulgaris f. vulgaris*: central nervous symptoms

<table>
<thead>
<tr>
<th>Equine</th>
<th>Decrease of upper lip reflex</th>
<th>Decrease of palatal reflex</th>
<th>Difficulties in retracting tongue after traction</th>
<th>Scaring reflex diminished</th>
<th>Flexor reflex diminished</th>
<th>Difficulties in circling in a small radius</th>
<th>Anal reflex diminished</th>
<th>Anal sphincter relaxed</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1 O O</td>
<td>O O ++</td>
<td>O O</td>
<td>O O</td>
<td>O O</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>N2 O O</td>
<td>O O ++</td>
<td>O O</td>
<td>O O</td>
<td>O O</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>N3 O O</td>
<td>O O ++</td>
<td>O O</td>
<td>O ++</td>
<td>O ++</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>N4 O O</td>
<td>O O ++</td>
<td>O O</td>
<td>O N</td>
<td>O N</td>
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</tr>
<tr>
<td>N5 O O</td>
<td>O O ++</td>
<td>O O</td>
<td>O O</td>
<td>O O</td>
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</tr>
<tr>
<td>N6 + +</td>
<td>++ +</td>
<td>N O</td>
<td>+ N</td>
<td>+ N</td>
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<tr>
<td>N7 O O</td>
<td>O O ++</td>
<td>O O</td>
<td>O O</td>
<td>O O</td>
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<td></td>
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<tr>
<td>N8 ++ +</td>
<td>++ +</td>
<td>N O</td>
<td>O N</td>
<td>O N</td>
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<td></td>
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</tr>
<tr>
<td>N9 N N</td>
<td>N N ++</td>
<td>N O</td>
<td>O N</td>
<td>O N</td>
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<tr>
<td>N10 N N</td>
<td>N N ++</td>
<td>N O</td>
<td>O N</td>
<td>O N</td>
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</tr>
<tr>
<td>N11 + +</td>
<td>++ +</td>
<td>N O</td>
<td>+ N</td>
<td>+ N</td>
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<tr>
<td>N12 O O</td>
<td>O O ++</td>
<td>N O</td>
<td>O N</td>
<td>O N</td>
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<td>N13 O O</td>
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<td>N O</td>
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<tr>
<td>N14 O O</td>
<td>O O ++</td>
<td>N O</td>
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<tr>
<td>N15 O O</td>
<td>O O ++</td>
<td>N O</td>
<td>O N</td>
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<tr>
<td>N16 O O</td>
<td>O O ++</td>
<td>N O</td>
<td>O N</td>
<td>O N</td>
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</tbody>
</table>

**Experimental cases**

| E1 + + | N + | + N | + N |
| E2 + + | N + | + N | + N |
| E3 + + | N + | + N | + N |

\(^1\)11 severe, 11 moderate, 1 slight, – no symptoms.
\(^2\)O, symptoms not alleviated.
\(^3\)N, normal.

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*Figure 1.* Bamboo from a farm where natural cases occurred and which was used for the experimental poisoning.
fields where the animals became sick, bamboo had been planted to supply shade to the animals during the hottest hours of the day. Of the 16 examined horses affected naturally by the disease, nine were females, of which four had recently given birth and two were pregnant; the age of the animals varied from 11/2 to 19 years. The cases of poisoning usually were associated with shortage of forage caused by the smaller rainfall from July to November or because the animals were on pastures of *B. brizantha*, which horses do not like to eat. Most of the animals recovered after their removal to pastures that did not contain bamboo. Of the 16 diseased horses, only two died spontaneously (N3 and N5), but no necropsy could be done on these. One animal (N8), affected for 3 days by the disease, was killed for a postmortem examination.

Clinical picture

The clinical picture (Table 1) was characterized by disturbances of the nervous system such as motor incoordination, crossing of the legs during walking, somnolence, difficulty in apprehension, mastication, and swallowing of food with accumulation of grass in the oral cavity. Other clinical signs included instability and standing with abducted legs (Figs 2 and 3) and occasional head pressing (Fig 4). There was a decrease or absence of the tongue reflex, as observed by difficulty or incapacity to retract the tongue after it had been pulled out (Fig 3). Also observed was a decrease of both the palatal reflex and that of the upper lip. Animals had difficulty walking in a tight circle and correcting the position of the legs after abduction or crossing. In addition, affected animals lost weight, had little or no local cutaneous sensitivity, sialorrhea, and, in some cases, edema in the ventral area of the abdomen. In one animal (N16), temporary blindness also was observed.

The course of the disease varied from subacute to chronic, according to the time the animals spent in the pastures that contained bamboo.

Postmortem findings

In the euthanized animal, no significant alterations were observed, except for slight ascites.

Histopathology

In the single natural case examined (N8), a few random lesions were verified in the central nervous system in the form of slight edema of the white matter. Degeneration of some axons (digestion chambers containing a few macrophages) was observed in the medulla oblongata.
EXPERIMENTAL CASES

Clinical picture

Clinical alterations consisted of motor incoordination with crossing of the legs during walking, somnolence, and difficulties in mastication and swallowing of the food with accumulation of grass in the oral cavity. Other clinical signs included abduction of the legs (Fig 5), decrease of the lingual reflex, evidenced by difficulty of retracting the tongue after it had been pulled out, decrease of the palatal reflex and that of the upper lip, difficulty walking in a tight circle and correcting the position of the legs after abduction and crossing. Weight loss, decrease of cutaneous sensitivity, and, in two animals (E1 and E2), slight edema in the ventral area of the abdomen also were observed.

Symptoms were first seen at 24 hours after initial feeding of the plant in equines E1 and E3 and at 72 hours in equine E2. In spite of continuing consumption, the symptoms did not increase in intensity, but varied slightly in degree during the experiment. Equines E1 and E2 were killed on the day of the last administration, the 31st and 61st days, respectively.

Postmortem findings

No significant alterations were observed in the two euthanized experimental horses (E1 and E2).

Histopathology

No significant lesions were present in the two euthanized experimental horses (E1 and E2).

Tests for cyanogenic glycosides

All tests performed on the sprouts and mature leaves of Bambusa vulgaris f. vulgaris were negative for hydrocyanic acid.

DISCUSSION

Feeding bamboo leaves to three horses produced an identical clinical picture to one observed in naturally affected animals occurring in northeastern Pará, Amazonia, Brazil.

The clinical manifestation, in both the natural and experimental cases, was the motor incoordination associated with the paresis of the tongue, although this was slightly less severe in the experimental cases. Minor histological lesions were seen in the central nervous system in the natural cases but not in the experimental animals. The more severe symptoms and the histological alterations observed in the natural cases could possibly be explained by the fact that the animals ingested larger amounts of the plant over a longer period. However, we have no data to confirm that the histological brain lesions were caused by ingestion of bamboo.

Part of the clinical picture is very similar to that seen in the pontino-bulbar syndrome, so this poisoning should be differentiated from equine diseases that affect the brain stem. These include amitraz poisoning, rabies, myeloencephalitis by equine herpesvirus I, and equine encephalomyelitis.

In the clinical picture of poisoning by amitraz, apart from the nervous signs, digestive symptoms originate from the impaction of the large intestine that are usually progressive. In rabies, other herbivores also are affected, the symptoms are progressive, and the lethality is 100%. This contrasts with the full recovery of the horses after bamboo ingestion was interrupted. The nonsuppurative encephalomyelitis and ganglioneuritis with or without Negri bodies also are not part of the histological picture of poisoning by bamboo.

In cases of myeloencephalitis by the equine herpesvirus I, the neurological symptoms usually begin suddenly, vary from light ataxia to severe paralysis, and can accompany outbreaks of abortion and respiratory disease. An important clinical manifestation of this disease is paralysis of the bladder, which commonly occurs in initial phases. Postmortem findings are hemorrhagic focuses in the brain and in the spinal cord, whereas histological examination shows vasculitis with secondary lesions in the neuropil.

Equine encephalomyelitis is mainly characterized by symptoms of hyperexcitability, severe depression, blindness, and pressing of the head against firm objects. The mortality rate varies from 20–90% accordingly to the virus strain. Histological lesions, predominantly in the gray matter and especially in the cerebral cortex, thalamus, and hypothalamus, are characterized by neuronal necrosis with neuronophagia, severe perivascular cuffing (mono and polymorphonuclear leukocytes), and focal and diffuse microgliosis. Consequently, the symp-
tomatology, mortality rate, and histological lesions of this disease are very different from those found in poisoning by bamboo.

Poisoning by bamboo also can be differentiated from leukoencephalomalacia and from equine protozoal myeloencephalitis. In cases of leukoencephalomalacia, the symptoms are mainly attributable to disturbances of cortical origin but also of the brain stem. The diagnosis is confirmed by malacia cavitation in the white matter of the brain, by information about moldy maize in the ration, and that the symptoms last from a few hours to 3 or 4 days\(^\text{10}\) with a lethality of 100%. Equine protozoal myeloencephalitis can cause disturbances related to lesions of any part of the central nervous system, but most frequently to the encephalic brain stem and the spinal cord, with asymmetrical ataxia and tetra or paraparesis.\(^\text{12}\) Asymmetrical clinical signs are highly suggestive of this disease. The start of symptoms can be insidious and gradual or acute with rapid progress.\(^\text{7}\) Histologically, malacia and nonsuppurative inflammatory reactions are seen in the central nervous system with intraneuronal schizonts in approximately 50% of cases.\(^\text{13}\)

Diagnosis of bamboo poisoning in horses should take into account the following: pastures that contain bamboo with evidence that large amounts have been ingested; occurrence of a nervous disease characterized by lack of motor coordination and paresis of the tongue; low lethality and fast recovery after removal to a pasture free of the plant.

From the epidemiological data and by comparison of the clinical and pathological pictures seen in both the natural and experimentally produced disease, it can be concluded that the neurological disease that occurs in horses in the Northeast of Pará is caused by the ingestion of leaves of *Bambusa vulgaris* f. *vulgaris*.

The disease has no relationship to poisoning by hydrocyanic acid, because of the negative results for hydrocyanic acid and also because the clinical picture is not similar to that caused by this acid.

**ACKNOWLEDGMENTS**

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**REFERENCES**