BJVP
Brazilian Journal of Veterinary Pathology
ISSN 1983-0246

Case Report

Baccharis coridifolia Poisoning in Water Buffalo (*Bubalus bubalis*) in the North of the Province of Corrientes, Argentina

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Submitted November 11th 2014, Accepted January 1st 2015

Abstract

Eight of 57 water buffalo (*Bubalus bubalis*) died after grazing 30 days on 38-hectare pasture contaminated with *Baccharis coridifolia* in the north of the Province of Corrientes (northeastern Argentina). Most were found dead, others showed depression, anorexia, diarrhea, incoordination of movements and collision against fences. The conditions that caused the poisoning were overgrazing, poor pasture and lack of aversion to the consumption of *Baccharis coridifolia*. Gross findings included necrotic rumenitis, necrotic-hemorrhagic abomasitis, necrotic-hemorrhagic enteritis and marked brain edema. The histopathological findings were necrosis of the epithelium of the mucosa of the forestomachs, necrosis and hemorrhage in mucosa of the small intestine, necrosis of lymphocytes in lymph follicles in spleen and thymus and vasogenic brain edema. The lesions, except for brain edema, are typically reported in cattle intoxicated by *Baccharis coridifolia*. Vasogenic brain edema is not described in any animal species naturally or experimentally intoxicated with this plant; this change may be due to direct toxic effect of *Baccharis coridifolia* that cause vascular injury or was a secondary injury.

Key words: poisonous plants, water buffalo, necrotic rumenitis, lymphocyte necrosis, brain edema.

Introduction

Baccharis coridifolia (Asteraceae), popularly known as mio-mio, is an important toxic plant affecting ruminants and horses in southern Brazil, Uruguay and Argentina. It causes a severe and acute poisoning characterized by necrosis of gastrointestinal mucosal epithelial cells and lymphocytes in various lymphoid organs (4). In Argentina, this poisoning is considered very important and common in cattle. *Baccharis coridifolia* has a wide distribution covering the central, northern and northeastern regions, reaching the Rio Negro province in south Argentina (5, 9). Spontaneous poisoning occurs mainly in cattle (3, 5, 8) and is reported less frequently in sheep (10) and horses (1). To our knowledge, there are no documented cases of *B. coridifolia* poisoning in water buffalo (*Bubalus bubalis*) in Argentina. In southern Brazil natural poisonings are reported in buffalo with *Baccharis megapotamica* var. *weirii* with conditions of poisoning, clinical signs and lesions similar to those observed in cattle poisoned with *B. coridifolia* (6). Poisoning occurs when animals with no previous experience with this plant are introduced into pastures highly contaminated with *B. coridifolia*. The susceptibility increases when animals are subjected to stress conditions, such as fatigue, hunger and dehydration (3, 8). In cattle, the clinical course is acute and characterized by nervous and gastrointestinal signs. The clinical signs begin between 5-29 hours after ingestion of the plant; there is restlessness, the animal lies down and rises repeatedly, mydriasis, excessive tearing, incoordination of the hind limbs, hyperesthesia in the back skin, muscle tremors and, in the final stages of poisoning, depression with lateral recumbency until death. Gastrointestinal signs include anorexia, salivation, rumen stasis, mild abdominal distension and watery diarrhea. Other clinical sings are dyspnea, tachycardia and

polydipsia. In fatal cases, death occurs within 4-34 hours after onset of clinical signs (3, 4, 8). The aim of this report is to describe the anamnesis, necropsy and histopathology findings of an outbreak of *B. coridifolia* spontaneous poisoning in water buffalo which occurred in the northern of the province of Corrientes, Argentina.

Case report

In June 2012, 57 Mediterranean buffalo, between 12 and 18 month-old males, were transferred 43 km from lands without *B. coridifolia* in Empedrado Township to a field located in the Riachuelo Township, in the north of Corrientes, Argentina. The animals were housed in a paddock of 38-hectare of native pasture. On day 30 the first dead buffalo is recorded, the deaths continued until day 50, recording a total of eight buffalo dead. The owner reports that some buffalo were found dead without previous clinical signs of illness, others expressed anorexia, diarrhea and depression and a buffalo showed incoordination of movements and collision against the fences, the day before dying.

The necropsy of the three buffalo was performed. The small intestine had severe hyperemia and was distended with fluid, the abomasum was distended with fluid and showed petechiae and ecchymosis in serous the greater curvature. The mucosa of the rumen, reticulum and omasum were observed reddened (diffuse hyperemia) and with erosion foci (Fig. 1). The mucosa from the abomasum showed diffuse edema and hemorrhage (Fig. 2). The mucosa from the small intestine, particularly the duodenum and jejunum, were with petechiae, ecchymoses and some areas with diffuse hemorrhage. The liver was paler with fibrinous exudate on the capsules and distended gallbladder. The lung was with congestion and edema. The heart showed hemorrhages in the pericardium and in the sub-endocardium. The brain showed severe swelling, softening and congestion (Fig. 3). Samples of rumen, reticulum, omasum, abomasum, small intestine, myocardium, liver, lung, lymph nodes, spleen, thymus, kidney and whole brain were fixed in 10% formalin.

Tissue samples were embedded in paraffin, sectioned at 4 μm and stained with hematoxylin and eosin (HE) for histopathological analysis. The main histopathological findings were necrosis of mucosal epithelial cells in forestomachs, necrosis of lymphocytes and perivascular edema in the brain. The mucosal of the rumen, reticulum and omasum were with degeneration and necrosis of the stratum spinosum epithelial cells, causing the formation of the vesicles and fissures filled with cellular detritus (Fig. 4). Multiple foci of hemorrhage and necrosis were found in intestinal mucosa. Furthermore, the forestomachs, abomasum and small intestine showed diffuse vascular changes characterized by hyperemia, edema and swelling of capillary endothelial cells of the lamina propria mucosae and submucosa. Some arterioles of the submucosa showed necrosis of the tunica media associated with thrombosis (Fig. 5). The liver showed diffuse cell swelling of hepatocytes. The spleen and thymus showed necrosis of lymphocytes in germinal centers of lymphoid follicles (Fig. 6). The cerebral cortex, cerebellum and brainstem (cross sections through cerebellar peduncles, cross sections of the midbrain through the anterior colliculi and posterior colliculi) showed swelling of endothelial cells and perivascular edema. Perivascular edema was most severe in the brainstem and was characterized by eosinophilic globular drops into the Virchow-Robin spaces (Fig. 7). The glial cells around the blood vessels showed swollen eosinophilic cytoplasm. The neurons showed chromatolysis and multifocal ischemic necrosis in cerebral cortex.

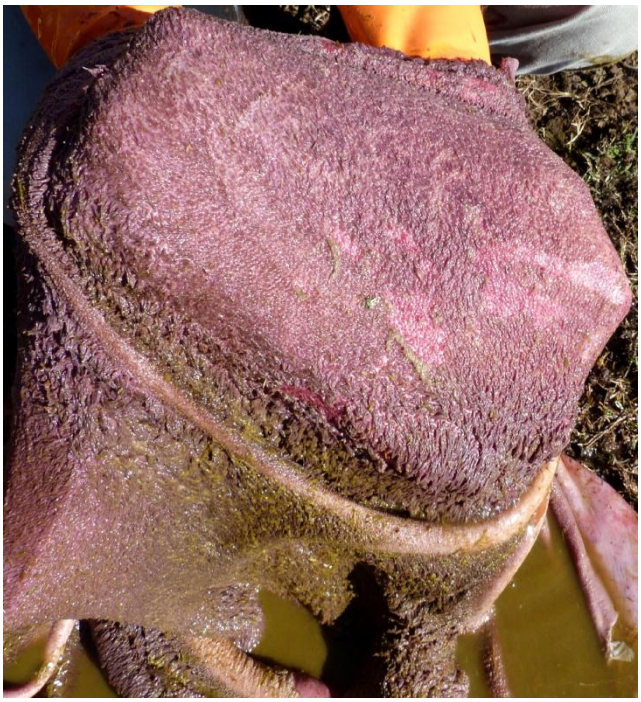

Figure 1. *Baccharis coridifolia* poisoning in water buffalo. Necrotic rumenitis. Ruminal mucosa with severe diffuse hyperemia and erosion areas.

Suspecting poisoning, the pasture was inspected and large amounts of a plant later identified as *B. coridifolia* with seeds were observed (Figure 8). There was evidence that buffalo had been consuming these plants. Samples of the plant were sent to the Institute of Botany Northeast (IBONE), they were identified as *Baccharis coridifolia*.

Discussion

These buffalo were born in lands without *B. coridifolia*, this lack of previous contact with this plant could promote the poisoning. Additionally, overgrazing also contributed to this poisoning, since the first cases

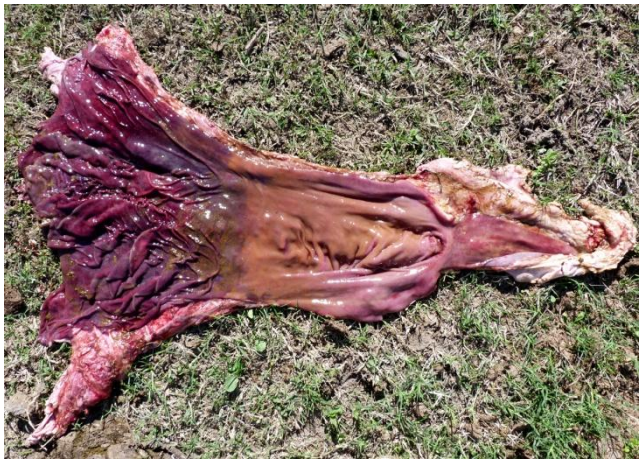

Figure 2. *Baccharis coridifolia* poisoning in water buffalo. Necrotic-hemorragic abomasitis.

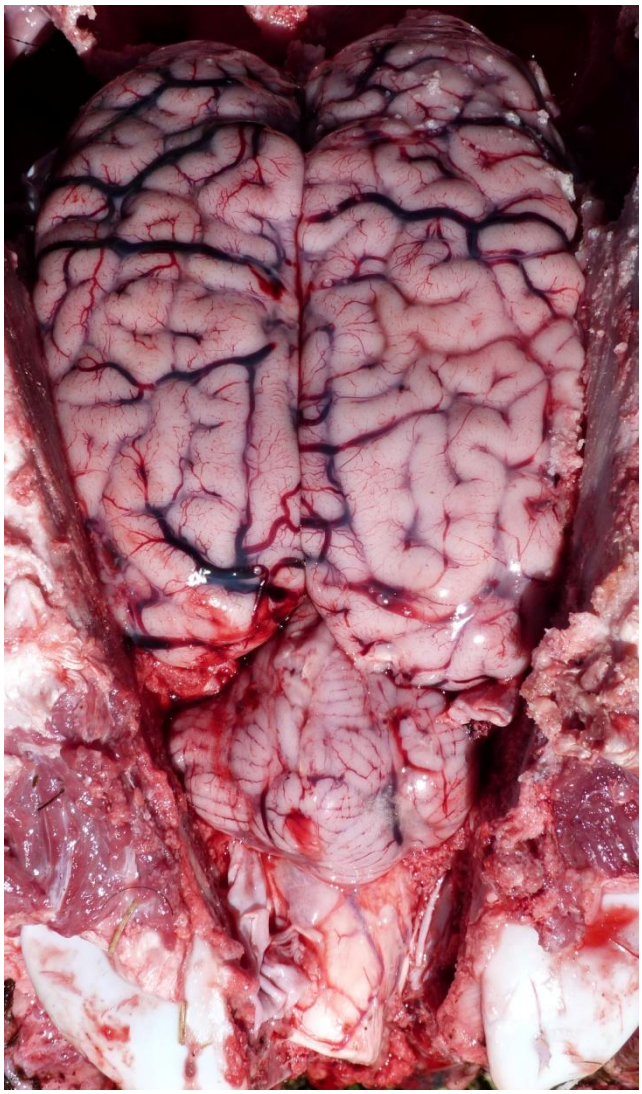

Figure 3. *Baccharis coridifolia* poisoning in water buffalo. Brain edema. The brain circumvolutions are swollen, flattened and soft. Meningeal vessels are hyperemic.

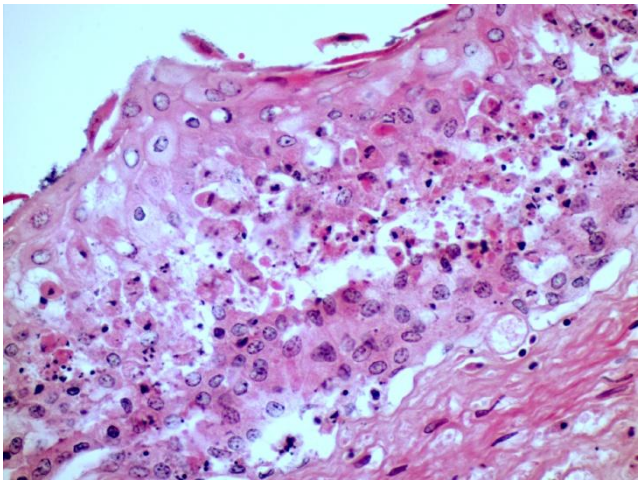

Figure 4. *Baccharis coridifolia* poisoning in water buffalo. Ruminal mucosa with necrosis of the epithelial cells of the stratum spinosum (HE 400x).

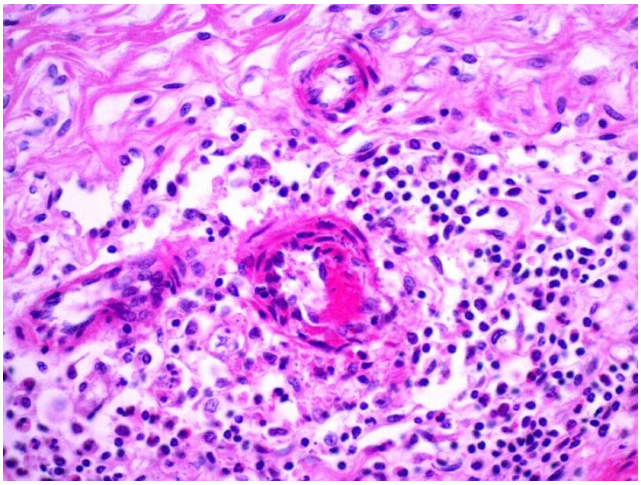

Figure 5. *Baccharis coridifolia* poisoning in water buffalo. Jejunal submucosa. Arteriole with focus of necrosis of the tunica media and perivascular inflammatory infiltrate (HE 400x).

occurred 30 days after grazing a poor pasture. The conditions which favor the poisoning with *B. coridifolia* reported more frequently were the introduction of animals from lands without *B. coridifolia* into contaminated lands with the plant, after spending several hours of fasting, thirst, fatigue and stress (transportation or confinement in paddocks without food). In these cases, the poisoning appears within hours or days after introduction of the animals into infected lands with *B. coridifolia* (1, 8, 10). Animals raised in areas where there is *B. coridifolia* are not poisoned because they develop a strong aversion to the consumption of this plant, after ingesting small amounts of it (2).

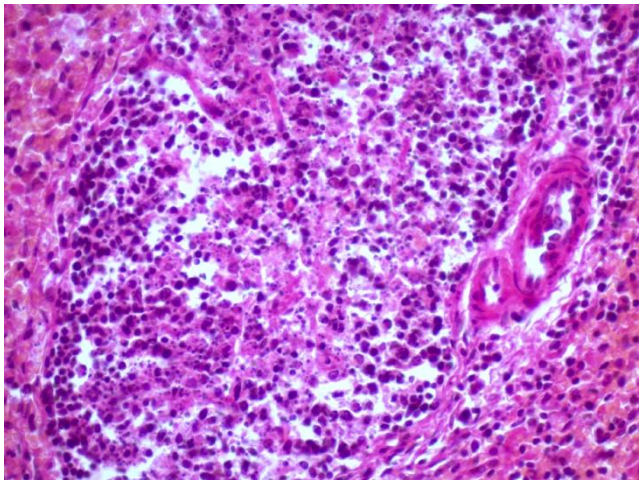

Figure 6. *Baccharis coridifolia* poisoning in water buffalo. Lymphoid follicles of the spleen with pyknosis and karyorrhexis of the lymphocytes (HE 400x).

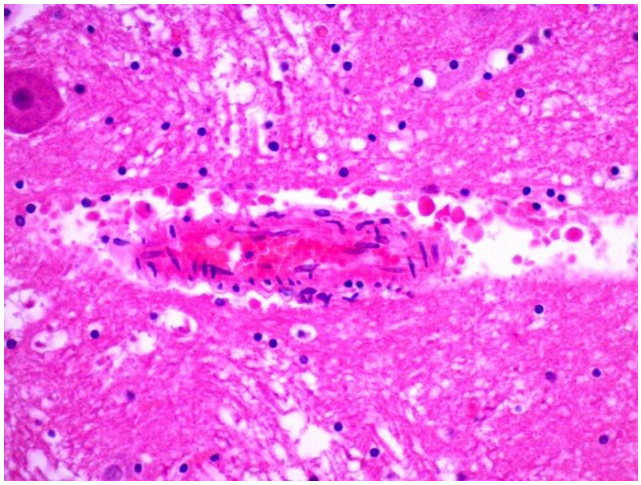

Figure 7. *Baccharis coridifolia* poisoning in water buffalo. Vasogenic brain edema. Acidofilic drops of extravascular plasma proteins into the Virchow-Robin spaces of the midbrain (cross sections through the anterior colliculi). Some perivascular glial cells have swollen eosinophilic cytoplasm (HE 400x).

The lesions described in cattle (3, 4, 8), sheep (10) and horses (1) poisoned by *B. coridifolia* were lymphoid tissue necrosis and necrotic gastroenteritis characterized by different degrees of hyperemia, edema, hemorrhage and necrosis of the mucosa of forestomachs, glandular stomach and intestines. Such lesions were found in this outbreak; however, cases of natural and experimental poisoning with *B. coridifolia* do not report vasogenic brain edema or vasculitis, as observed in this case. In some cases, are reported neurological signs (instability of the hind limbs, muscle tremors and depression) when cattle consume *B. coridifolia* in flowering and fruiting, when the plant is more toxic (4). Numerous authors have reported vascular injuries related to poisoning with *Baccharis* spp. Barros et al. (1993) describe neutrophilic infiltrate and perivascular brain hemorrhage in cattle poisoned with *B. coridifolia*, although they were not considered significant results of the poisoning. Buffalo poisoned with *B. megapotamica* var. *weirii* had fibrin thrombi in vessels in the submucosa of forestomach and in the liver sinusoids, possibly in response to necrosis of endothelial cells and hepatocytes (7). Hamsters treated with high doses of *Baccharis pteronioides* developed severe necrotizing vasculitis with thrombosis of the hepatic, kidney, spleen and mesenteric vessels. *B. pteronioides* could have a novel vascular toxin. It is also possible that the vasculitis was a secondary lesion to endothelial damage caused by bacterial endotoxins (11).

Figure 8. *Baccharis coridifolia* on native pasture of the farm where the buffaloes become intoxicated, June 2012, Riachuelo Township, Corrientes, Argentina.

The most important differential diagnosis includes poisoning with *Baccharis megapotamica* var. *megapotamica* and *B. megapotamica* var. *weirii*. Histological examination of liver contributes in this respect (4). Liver lesions reported in buffalo poisoned with *B. megapotamica* var. *weirii* (6, 7) and in bovine poisoned with *B. megapotamica* var. *megapotamica* (4) are hepatocelular necrosis and intense hepatocellular vacuolization with intracytoplasmic eosinophilic globules (Councilman bodies). Liver lesions reported in *B. coridifolia* poisoning are vacuolization of hepatocytes, edema in the space of Disse and polymorphonuclear infiltrate into sinusoids (3, 4).

The pathogenesis of this poisoning is not fully established. It is suggested that dehydration, electrolyte imbalance, septicemia derived from bacterial invasion from the digestive tract through the epithelial damage and disseminated intravascular coagulation may have played an important role (3, 8, 7). Our observations also suggest that vasogenic brain edema and vasculitis could be involved in the pathogenesis and cause of death in this poisoning.

The diagnosis of poisoning *B. coridifolia* was based on anamnesis, the lesions and the identification of the plant in the pasture. The circumstances that led to poisoning were overgrazing and lack of aversion to *B.*

coridifolia consumption. The lesions observed, with the exception of brain edema, are similar to those reported in cattle. The vasogenic brain edema and gastrointestinal vasculitis observed in the present case were not described in naturally or experimental poisoned animals.

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