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Cerebrocortical necrosis in small ruminants in Argentina: a case series

Necrosis cerebrocortical en pequeños rumiantes en Argentina: reporte de casos

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Summary: Several neurological diseases can affect small ruminants. Cerebrocortical necrosis (CCN) is a pathology associated with different etiologies. One of them, nutritional polioencephalomalacia (PEM) is a common metabolic disease associated to intensive production systems. We report eight outbreaks of CCN in small ruminants (goats and sheep) of different regions of Argentina, probably associated to ruminal acidosis, elevated levels of sulfur in the forage and water and/or suspected lead poisoning. Affected sheep and goats developed sudden neurological signs such as ataxia, amaurosis, opisthotonus, dissociation, lethargy, hypextension of the limb, lateral decubitus, and death. Postmortem examination of six animals was performed, and gross lesions were scarce, characterized as cerebrocortical malacia. Histologically, severe cerebrocortical laminar necrosis was observed. Based in these findings and/or response to thiamine and dexamethasone treatment in four outbreaks, a diagnosis of CCN was achieved, of different suspected origins. This disease is commonly associated with intensive production systems, and therefore, coud be underdiagnosed in extensive conditions, more frequently used for small ruminant rearing.

Keywords: sheep, goat, polioencephalomalacia, cerebrocortical necrosis, Argentina

Resumen: *Los pequeños rumiantes pueden sufrir diversas enfermedades neurológicas. La necrosis cerebrocortical (NCC) es una patología que puede tener diferentes etiologías. Una de ellas es la polioencefalomalacia nutricional (PEM), una enfermedad metabólica común asociada a sistemas de producción intensivos. En este trabajo se reportan ocho brotes de NCC en caprinos y ovinos de diferentes regiones de Argentina, posiblemente asociados a acidosis ruminal, elevados niveles de sulfatos dietarios o sospechas de intoxicación con plomo. Los caprinos y ovinos afectados desarrollaron enfermedad neurológica repentina con signos como ataxia, amaurosis, opistótonos, disociación del medio, letargia, hiperextensión de los miembros, y decúbito lateral seguido de muerte. El examen post mortem de seis animales, reveló lesiones macroscópicas escasas, caracterizadas como malacia cerebrocortical. En el análisis histopatológico, se observó severa necrosis cerebrocortical laminar. Basados en estos hallazgos clínico-patológicos y la respuesta favorable al tratamiento con tiamina y dexametasona en cuatro*

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brotes, se arribió al diagnóstico de NCC con diferentes posibles orígenes. Esta enfermedad se asocia comunmente con sistemas de producción intensivos, y por lo tanto, puede ser subdiagnosticada en sistemas extensivos, mas frecuentemente utilizados para la producción de pequeños rumiantes.

Palabras claves: *ovinos, caprinos, polioencefalomalacia, necrosis cerebrocortical, Argentina*

Introduction

Cerebrocortical necrosis (CCN) is a neurological disease affecting mainly ruminants: cattle (da Cunhal et al., 2010; Gould, 1998; Haydock, 2003; Sant'Ana and Barros, 2010), sheep (Low et al., 1996), goats (Lima et al., 2005; Maxwell, 1980; McSporran, 1988), deers (Smits & Wobeser, 1990), buffaloes (Barbosa et al., 2023) and camelids (Beck et al., 1996; Kiupel et al., 2003).

Various etiologies are associated with NCC. Nutritional polioencephalomalacia (PEM) is usually associated with thiamine deficiency (Sant'Ana and Barros, 2010; Thornber et al., 1979) or consumption of plants containing thiaminases (Ramos et al., 2005). Other etiologies have been reported as predisposing to CCN including salt intoxication or water deprivation (Duarte et al., 2014; Scarratt et al., 1985), lead intoxication (Baker, 1987), encephalitis caused by bovine herpesvirus type 5 infection (Cagnini et al., 2015; Riet-Correa et al., 2006), amprolium poisoning (Nogueira et al., 2010), and diets containing excessive sulfur (Gould, 2000; Haydock, 2003). In addition, PEM has been associated to ruminal thiamine metabolism alterations during ruminal acidosis (Karapinar et al., 2010; Owens et al., 1998; Sivaraman et al., 2016).

Nutritional polioencephalomalacia is clinically similar to other neurological disease. Although in the initial stages, PEM is characterized by clinical signs of aggression and excitement, the main features are depression, central blindness (amaurosis), muscular tremors, nystagmus, opisthotonus, circling, ataxia, lateral or sternal recumbency and death (Gould, 1998; Low et al., 1996; Maxwell, 1980; Sant'Ana and Barros, 2010; Sivaraman et al., 2016). Upon prompt treatment with thiamine and dexamethasone clinically affected animals usually respond favorably (Rammell and Hill, 1986; Sivaraman et al., 2016) and this response could be used as therapeutic diagnosis. Clinical and pathological characteristics of PEM are similar in different domestic ruminants, however, in small ruminants, the clinical course is typically shorter (Koestner and Jones, 1997).

Histopathological diagnosis is key to confirm CCN. Laminar neuronal degeneration with individual neuronal necrosis in the cerebral cortex, neuronophagia, endothelial cell hyperplasia, and gliosis are characteristic lesions associated with PEM (Câmara et al., 2018; Kiupel et al., 2003).

This paper reports eight outbreaks of CCN in goats and sheep, registered at three Veterinary Diagnostic Services of the National Institute of Agricultural Technology (INTA) in Buenos Aires, Salta and Rio Negro provinces, Argentina.

Material and methods

The database from three different Veterinary Diagnostic Services of INTA was revised: INTA Balcarce (2000-2023), INTA Bariloche (2010-2023) and INTA Salta (2013-2023). Eight outbreaks of PEM in small ruminants were included in this study. Diagnosis criteria was based on anamnesis, clinical signs, response to treatment, gross and/or histological findings.

One necropsy was performed in outbreaks #1, #2, #3, #5, #6 and #7. on animals spontaneously dying after clinical neurological course. Samples of central nervous system, lung, liver, heart, kidney, lymph nodes, prestomachs, abomasum, small and large intestine were collected and fixed in 10% buffered formalin for 48h and embedded in paraffin-embedded blocks. Four micrometer sections were prepared routinely and stained with H&E for histological study.

When available, fresh or formalin fixed cerebral cortex samples were observed under ultraviolet light according to Little (1978) for the presence of autofluorescence compatible with cerebrocortical necrosis.

Results

The information of the eitght affected flocks is summarized in Table 1. Briefly, the eight outbreaks occurred in different production systems affecting goats (n = 5), sheep (n = 2) and both species simultaneously (n = 1). Disease was detected in different ecoregions of Argentina: Salta (n = 2), Jujuy (n = 1), Buenos Aires (n = 2) and Rio Negro (n = 3)". The affected animals were from beef (n = 4), dairy (n = 2) and Mohair fiber (n = 2) systems. Three out of eight outbreaks affected young animals (kids or lambs) and five of them occurred in adult sheep or goats.

Table 1. Epidemiological data of eighth outbreaks of nutritional polioencephalomalacia in sheep and goats of Argentina.

Outbreak	Province / Department	Specie / Age / Breed	Date	Nutrition	System	Morbidity	Mortality
1	Buenos Aires / General Pueyrredón	Goat Saanen kids	November 2019	corn silage, chopped grass, complete meal and alfalfa pellet	Intensive / Dairy	4.3% (3/70)	1.4% (1/70)
2	Buenos Aires / Olavarría	Sheep / Texel and Dorper lambs	October 2023	complete meal, corn and mineral/vitamin supplement	Intensive / Beef	4.2% (4/96)	4.2% (4/96)
3	Salta / Cerrillos	Goat / kid	May 2015	corn, alfalfa hay and stale bread	Intensive / Beef	Individual (pet)	100.0% (1/1)
4	Salta / Cerrillos	Goat and sheep / adult	December 2020	cattle feedlot leftovers	Intensive / Beef	4.0% (1/25)	0.0% (0/25)
5	Jujuy / Tilcara	Goat / adults	December 2023	alfalfa hay and corn	Intensive / Dairy	7.1% (3/42)	4.8% (2/42)
6	Rio Negro / Veinticinco de Mayo	Goat /Angora adult	April 2014	native grasslands	Extensive / Mohair fiber	4.7% (7/150)	4.7% (7/150)
7	Rio Negro / Pilcaniyeu	Goat / Angora adult	June 2021	native grasslands / alfalfa hay	Semi-intensive / Mohair fiber	0.8% (1/123)	0.8% (1/123)
8	Rio Negro / General Conesa	Sheep / 2 years / Corriedale	February 2024	native grasslands / alfalfa hay / corn	Intensive / Beef	2.0% (1/50)	0.0% (0/50)

Cerebrocortical necrosis episodes were registered mostly in intensive or semi-intensive production systems and associated with the inclusion of chopped grass or hay (4/8) and corn (4/8) among other diet components. Morbidity and mortality rates ranged from 0.8 to 7.1% with high lethality rates. According to the anamnesis information some of the outbreaks were suspected to be secondary to ruminal acidosis (outbreaks #1 to #5 and #8). Outbreak #6 occurred in adult goats grazing native grasslands. This later episode was suspected to be related to the consumption of grasslands or water with excessive sulphur. Unfortunately, the content of sulphur in the diet was not evaluated. Case #7 affected only one adult goat, where the presence of an old battery near to the shelter was suspected to be associated with CCN-lead poisoning.

Clinical and/or pathological findings registered in the eight outbreaks included in this work are summarized in Table 2, and shown in Figure 1. Briefly, sudden clinical signs were observed in all eight outbreaks, characterized by ataxia and opistotonus (7/8), dissociation, lethargy, and bruxism (2/8), amaurosis and muscular tremors (1/8). In four ouf of six outbreaks (where treatment was applied in affected animals), a positive clinical response to prompt treatment with thiamine and dexamethasone was registered; in two outbreaks, no response was observed after treatment.

Table 2. Clinical and gross pathological information of eight outbreaks of cerebrocortical necrosis in sheep and goats of Argentina. ND: not determined. NA: not available (no necropsy performed).

Outbreak	Clinical signs	Treatment response	Gross findings	UV	Microscopical findings
1	Ataxia, opisthotonus, dissociation, lethargy.	Yes	No lesions.	Yes	Cerebrocortical laminar spongiosis, hyperchromatic neurons (neuronal death) in the deepest layer of gray matter. Hydropic degeneration in the stratified epithelium of rumen; hyperkeratinization of the mucosa of ruminal pillars.
2	Ataxia, opisthotonus, dissociation, lethargy.	No	Flattening of convolutions, yellow staining of the gray matter in occipital and parietal cerebrocortical areas, ocasionally with a marked pale line between gray and white matter, rarely in depression.	Yes	Cerebrocortical necrosis with loss of neuropil architecture and infiltration with Gitter cells. Endothelial hypertrophy, perivascular edema, and mononuclear infiltration. Vacuolization of superficial ruminal epithelium (balloon degeneration) and hyperkerathosis.
3	Ataxia, opisthotonus.	No	Bilateral frontal cerebrocortical edema.	No	Laminar and segmental cortical necrosis, with neuronal death, spongiosis, and occasional presence of Gitter cells.
4	Ataxia, opisthotonus.	Yes	NA	NA	NA
5	Ataxia, opisthotonus.	Yes	Assymetrical bilateral frontotemporal cerebrocortical malacia.	No	Cerebrocortical neuronal necrosis, perivascular mononuclear inflammatory cells and spongiosis.
6	Ataxia, opisthotonus, blindness, bruxism, muscular tremors	ND	Bilateral frontotemporal cerebrocortical malacia (cavitation).	No	Neuronal necrosis and hyperplasia of glial cells.
7	Opisthotonus, bruxism, recumbency.	ND	No lesions.	No	Moderate vacuolization of cerebrocortical neuropil, neuronal necrosis, and infiltration of glial cells.
8	Anorexia bruxism dissociation lethargy	Yes	NA	NA	NA

Gross pathological findings were scarce and only reported in four out of the six outbreaks where necropsies were performed. These gross findings were characterized by cerebrocortical malacia/edema affecting the occipital, frontal, temporal and parietal areas and in one necropsy, with cavitation of cerebral cortex (Figure 1C). Malacia was confirmed by laminar cerebrocortical fluorescence observed under UV light, in two out of six brain samples collected during postmortem examination (Figure 1D).

The main microscopic lesions were observed in the brain and consisted of moderate to severe cerebrocortical laminar necrosis characterized by severe vacuolation (spongiosis) and neuronal necrosis ("red neurons") of the gray matter (6/6), and the presence of glial cell hyperplasia and Gitter cells (4/6) (Figure 2). In two animals (2/6), ruminal mucosa lesions commonly associated with ruminal acidosis, including hydropic degeneration, vaucuolization and hyperkerathosis of superficial ruminal epithelium, were observed (Table 2).

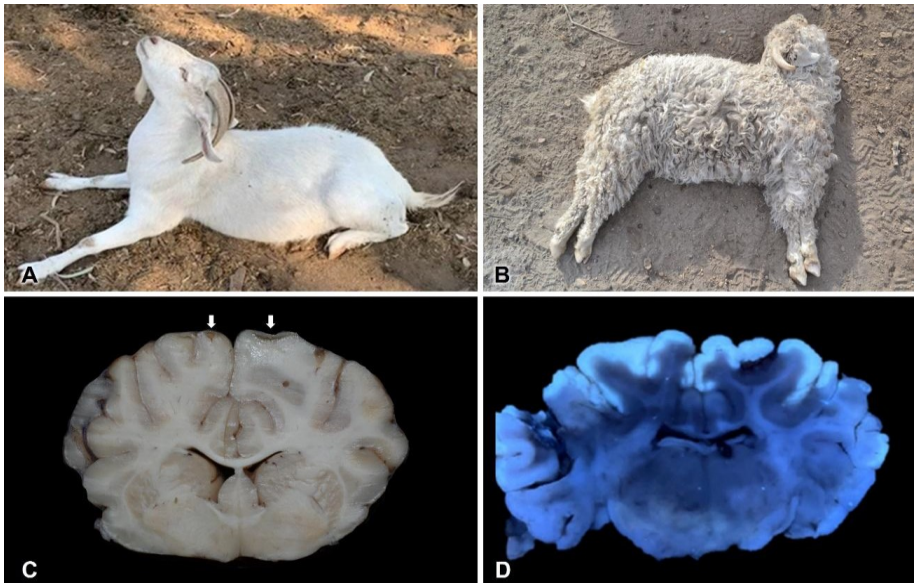

Figure 1. Clinical and gross pathological findings in cases of cerebrocortical necrosis in small ruminants from Argentina. (A) Saanen and (B) Angora adult goats with neurological signs: decubitus and opistotonus (outbreak #4 and #6, respectively). (C) Cut surface of partially formalin fixed brain with loss of cortical tissue seen as cavitations (arrows) in the cortical gray matter (outbreak #6). (D) Laminar fluorescence in the cerebral cortex of an affected sheep, under ultraviolet light (case #2).

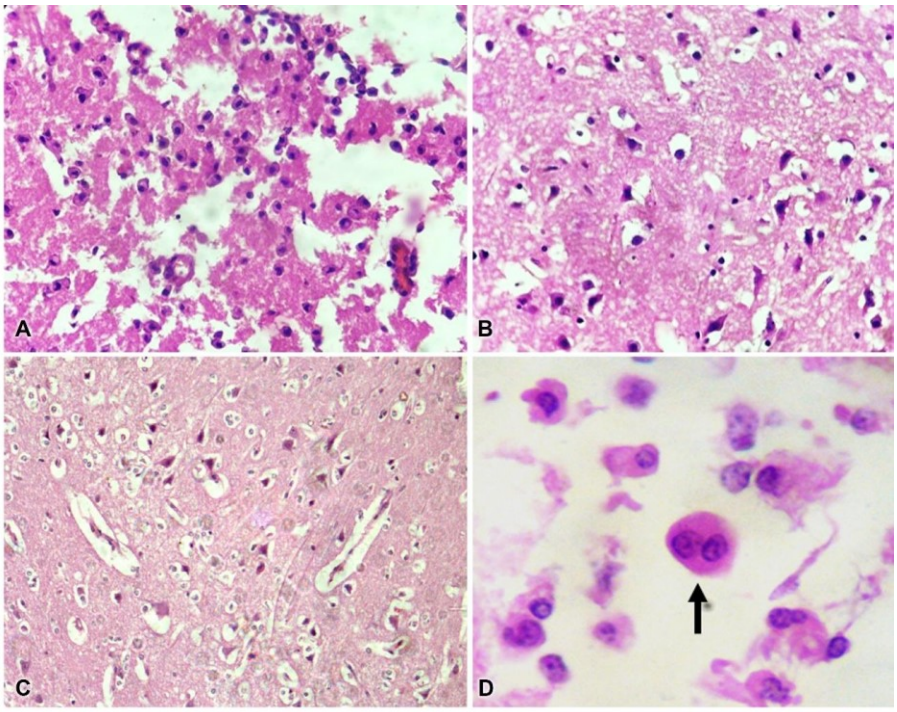

Figure 2. Histopathological findings in cases of cerebrocortical necrosis in small ruminants from Argentina. (A) Texel lamb (case #2). Brain cortex. Cerebrocortical necrosis with loss of neuropil architecture and infiltration with Gitter cells, and endothelial hypertrophy (H&E, 100×). (B) Texel lamb (case #2) and (C) Angora goat (case #7), respectively. Brain cortex. Cerebrocortical neuronal necrosis ("red neurone"), perineuronal retraction spaces and dilatation of the Virchow-Robin space with eosinophilic material, suggesting edema (H&E, 100×). (D) Goat kid (case #3). Brain cortex. Cerebrocortical spongiosis with occasional Gitter cells (arrow) (H&E, 400×).

Discussion

Clinical diagnosis of neurological diseases in ruminants is difficult, and submission of several samples are needed to confirm the etiology. The inclusion of anamnesis, epidemiological data, characterization of clinical signs, and gross and microscopical pathological findings, as well as other ancillary techniques, are needed to achieve a pathological diagnosis.

Nutritional PEM is a frequent neurological disease in youg cattle reared under intensive systems in Argentina (Castro et al., 2019). However, PEM in small ruminants is not commonly diagnosed probably related with their more extensive rearing systems. All these eight outbreaks affecting small ruminants were confirmed after analyzing the epidemiological and anamnesis information, compatible neurological signs, macroscopic and microscopic lesions, positive fluorescence under UV light, and/or positive response to recommended treatment, as it has been previously reported in affected animals (Rammell & Hill, 1986; Sivaraman et al., 2016).

The descriptive term CCN usually refered grossly to softening of gray matter and histopathologically to laminar cerebrocortical necrosis. Among different diseases characterized by similar neurophatologic changes, it usually referred to different etiologies linked to thiamine (vitamin B1) deficiency (da Cunhal et al., 2010; Evans et al., 1975; Thornber et al., 1979). We support that four out of five PEM outbreaks included in this report (outbreaks #1, #4, #5 and #8) were related to thiamine deficiency because some affected animals responded favorably to treatment with dexamethasone and thiamine.

Moreover, the occurrence of PEM in outbreaks #1 to #5 and #8 were suspected to be associated to ruminal acidosis, as it has been previously described in different ruminant species due to changes on ruminal microbiota and the consequent alteration of thiamine ruminal syntesis (Karapinar et al., 2010; Owens et al., 1998; Sivaraman et al., 2016). The nutritional information of these outbreaks, as well as the presence of ruminal pathological findings, such as hydropic degeneration in the ruminal mucose in small ruminants (Bonadiman et al., 2018) supported our diagnosis. Case #3 was suspected to be related to ruminal acidosis due to the nutritional information, but no lesions were observed during postmortem and histopathological analysis. These clinical-pathologic findings are similar to those reported in small ruminants and cattle (Câmara et al., 2018, da Cunhal et al., 2010; Ramos et al., 2005, Sant'Ana & Barros, 2010).

Excess sulphur in the diet was suspected to be the cause of PEM in goats of Patagonia grazing native grasslands, as it was previously described in lambs (Low et al., 1996). Sulphur poisoning can be diagnosed by measuring urine sulphur in affected animals (Gould, 2000) or the detection of excess sulphur in the diet (feedstuff and water). Unfortunately, sulphur content of the diet and urine of affected animals was not analyzed in case #6.

Other reported cause of CCN is lead poisoning (Baker, 1987), which cause ischemia-anoxia leading to laminar cortical necrosis. In spite of lead content in fluid/tissues was not performed to confirm the etiology, we think that the presence of one battery in the pen where animals stayed in outbreak #7 could be associated with the occurrence of CCN.

Nutritional PEM has been previously described as common cause of neurological disease of young cattle of intensive production systems in Argentina (Castro et al., 2019). Considering that small ruminants are extensively pastoral in our country (Devendra, 2010), it is probable that the occurrence of this disease is rare or underdiagnosed. Nevertheless, PEM should be included as differential diagnosis of neurological disease in such species, particularly when intensive husbandry or nutritional conditions are applied, such as dairy farms, feedlot systems or the inclusion of supplements in the diet.

Declaration of Competing Interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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