



Hypovitaminosis A in two beef calves: case report in Buenos Aires province

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Abstract

Hypovitaminosis A is an uncommon disease in grazing livestock systems, as the primary source of precursors (carotenes) is fresh forage. However, under environmental circumstances predisposing to the development of poor-quality forage, vitamin A deficiency and associated clinical signs can occur. This article describes the clinical findings of hypovitaminosis A in two 2-months-old calves in beef herd in Ayacucho, Buenos Aires Province, in November 2023. Blindness was the only clinical sign observed in a beef herd. Low vitamin A concentration was detected in blood of affected and non-affected calves and their mothers ranging from 7.8 to 24.8 $\mu\text{g dL}^{-1}$ in calves and from 15.5 to 28.9 $\mu\text{g dL}^{-1}$ in cows (reference 25-35 $\mu\text{g dL}^{-1}$ in calves and 40-50 $\mu\text{g dL}^{-1}$ in adults), confirming the deficiency status of the herd. During gestation, dams were fed corn-harvested hay. This diet may have been a poor source of carotene, leading to reduced hepatic reserves of vitamin A in the calves at birth. Consequently, clinical signs may have occurred during the first few weeks when these reserves were depleted.

Key words: vitamin A, deficiency, blindness, cattle.

Hipovitaminosis A en terneros de cría: reporte de caso en la provincia de Buenos Aires

Resumen. La hipovitaminosis A no es común en sistemas de producción pastoriles ya que sus principales precursores (carotenos) abundan en el forraje verde. Sin embargo, si se dan condiciones que favorezcan el desarrollo de forraje de poca calidad nutricional, se puede favorecer a la aparición de casos de deficiencia de vitamina A y la aparición de signos clínicos asociados. Este artículo describe los hallazgos clínicos de un caso de hipovitaminosis A en dos terneros de 2 meses de vida, en un rodeo para carne bovina en el partido de Ayacucho, en la provincia de Buenos Aires, en noviembre de 2023. Estos aparecieron con ceguera como único signo clínico. Se constataron bajos niveles de vitamina A en muestras de sangre de los terneros afectados como de sus compañeros (entre 7,8-24,8 $\mu\text{g dL}^{-1}$; valores de referencia 25-35 $\mu\text{g dL}^{-1}$), y sus madres (entre 15,5 y 28,9 $\mu\text{g dL}^{-1}$; valores de referencia 40-50 $\mu\text{g dL}^{-1}$), confirmando el estatus de deficiencia generalizada en el rodeo. Las madres durante la gestación estuvieron consumiendo rollos confeccionados con restos de cosecha de maíz. Esto puede haber hecho que reciban una escasa oferta de carotenos durante la gestación, y consecuentemente, los terneros nacieron con escasas reservas hepáticas, llevando a la aparición de los primeros signos clínicos cuando estas se agotaron, durante las primeras semanas de vida.

Palabras clave: vitamina A, deficiencia, ceguera, bovinos.

INTRODUCTION

Vitamin A is an essential fat-soluble molecule with different forms including retinol, retinal, and retinol esters (McGill et al. 2019). It plays a crucial role in physiological processes such as growth, development, reproduction, bone remodeling, and maintenance of epithelial tissues. Unlike water-soluble vitamins, fat-soluble vitamins cannot be synthesized by ruminants and must be ingested through

the diet. Although preformed vitamin A is not found in plant species, its most important precursor, beta-carotene, is abundant. After ingestion, this provitamin A molecule is converted to vitamin A in the small intestine, absorbed, and stored in the liver in the form of retinol ester. Vitamin A deficiency is frequently diagnosed in animals fed cereals and hay for prolonged periods (Craig et al. 2016). It's well known that clinical manifestations of hypovitaminosis A in cattle occur after several months of deprivation such

as weight gain, convulsions, diarrhea, exophthalmos, and blindness in calves and convulsions, ataxia, and blindness in adult cattle (Donkersgoed and Clark 1988, Van der Lugt and Prozesky 1989, Mason et al. 2003, Millemann et al. 2007). Congenital hypovitaminosis A can also occur when pregnant cattle is exposed to vitamin A deficient diets (Mason et al. 2003, Sosa et al. 2024). Newborn calves obtain vitamin A through colostrum and milk supply (Swanson et al. 2000) and are more susceptible to vitamin A deprivation due to their rapid growth, higher requirements, and lower reserves than adult cattle (Donkersgoed and Clark 1988, Kang et al. 2017).

Different factors, such as environmental and seasonal conditions (e.g. long-term drought), and nitrogen fertilization, cause decrease in beta-carotene concentration in plants and therefore can affect the vitamin A equivalents (Pickworth et al. 2012). Diagnosis of hypovitaminosis A is based on anamnesis and clinical information, in conjunction with the confirmation of low levels of serum or hepatic vitamin A (Donkersgoed and Clark 1988, Hill et al. 2009, Kang et al. 2017, Parker et al. 2017).

This work describes a case of clinical hypovitaminosis A in beef herds under grazing conditions in Buenos Aires province.

MATERIALS AND METHODS

The case occurred in a beef farm located in Ayacucho (37°07'10"S 58°34'29"W), Buenos Aires province, during November 2023. Affected calves were detected and clinically assessed. Blood samples were collected from clinically affected and non-affected and their mothers, by jugular puncture into a sterile collection tube, covered with metal paper to avoid contact with ultraviolet (UV) rays. Blood samples were kept at 4°C and later centrifuged for serum extraction. Vitamin A concentration in the serum was determined by liquid chromatography-tandem mass spectrometry (LC-MS/MS).

RESULTS AND DISCUSSION

The affected herd was composed by 60 multiparous cows with 51 calves (1 to 3 months-old). During the second half of their pregnancy, the cows were fed exclusively on maize harvest hay. At the beginning of the calving season (end of winter and middle of spring) the pregnant cows start grazing a native grassland. At the end of the calving season, two 2-months-old calves (out of 51) were identified with clinical signs (morbidity rate 3.92%; 2/51). They were clinically assessed showing partial blindness since they were able to distinguish some shadows when moving around. Nevertheless, they frequently collided with various obstacles in the corral where they were examined. They also showed an erratic gait with mild hypermetria and little or no response to threatening stimuli. In addition, an iridescent blue discoloration of the *tapetum lucidum* was observed (Figure 1). Papilledema, bleached *tapetum lucidum*, and dilated pupils are frequent changes of vitamin A deficiency in calves. The relationship between vitamin A deficiency and the *tapetum lucidum* bleaching has been

previously investigated and not fully understood (Takahashi et al. 2011).

Beside classical ocular lesions and clinical signs that could be attributed to vitamin A deficiency, further ophthalmic examination is necessary in the future to confirm these cases. Reports of ocular changes in yearling cattle due to vitamin A deficiency describe retinal degeneration, papilledema, corneal thickening and opacity and exophthalmos.



Figure 1. Blue iridescent coloration of the *tapetum lucidum*.

Low vitamin A concentration was detected in all sampled animals, including those with or without clinical signs and their mothers, ranging from 7.8 to 24.8 $\mu\text{g dL}^{-1}$ in calves and from 15.5 to 28.9 $\mu\text{g dL}^{-1}$ in cows (Table 1). Results showed that the average vitamin A content in serum was below the normal value (25–35 $\mu\text{g dL}^{-1}$ in calves and 40–50 $\mu\text{g dL}^{-1}$ in adults; Millemann et al. 2007). Nevertheless, most animals in the herd did not show clinical signs. Serum vitamin A levels represent a small proportion of the total body vitamin A reserve but also may be affected by many other factors such as the physiological estate of the animals, among other (Ghaffari et al. 2019, McGill et al. 2019).

After sampling, affected and non-affected calves were treated with a commercially available injectable solution containing vitamins A, D and E. According to the veterinary practitioner, the clinical signs in the affected calves were not reversed after treatment, but no new cases appeared in the herd. This could be related to an advanced stage of hypovitaminosis A. Hypovitaminosis A usually occurs in three phases: 1) nyctalopia or “night blindness”, a reversible stage and the first clinical sign, related to the deficiency of the visual pigment rhodopsin; 2) degenerative changes in the retina associated with prolonged vitamin A deficiency; and 3) stenosis of the optic canal, which induces an irreversible loss of vision (Van der Lugt and Prozesky 1989). Earliest signs of night blindness are generally unnoticed in extensive farmed animals (Donkersgoed and Clark 1988). More severe cases with degenerative changes of the retina are no longer reversible, which could explain no reversion of the treated affected calves in this case.

Although we cannot confirm that vitamin A was the ultimately cause of the blindness in these calves, the clinical sign observed in the deficient calves is characteristic of

hypovitaminosis A. However, this clinical presentation is uncommon in grazing livestock systems, where the intake of vitamin A precursors is usually covered by the forage consumed. In this case, the presumptive limited dietary intake during the second half of gestation may have resulted in calves being born with low hepatic reserves and clinical signs appearing in the first weeks of life. Therefore, hypovitaminosis A should be included as differential diagnosis under similar husbandry or environmental conditions. Regular monitoring of dam health and nutrition should be carried out during gestation to avoid deficiencies. It is important to provide information to beef farmers on feeding management practices to prevent the occurrence of such clinical signs and probable productive effect of this nutritional deficiency.

Table 1. Average and standard deviation of serum vitamin A concentration in calves with or without clinical signs and their dams.

Identification	Vitamin A ($\mu\text{g dL}^{-1}$)
Calves with clinical signs ($n = 2$)	13.8 ± 4.4
Calves with no clinical signs ($n = 7$)	17.1 ± 6.5
Dams of calves with clinical signs ($n = 2$)	17.3 ± 1.1
Dams of calves without clinical signs ($n = 4$)	22.0 ± 5.5
Reference values: 25 to 35 $\mu\text{g dL}^{-1}$ in calves. 40 to 50 $\mu\text{g dL}^{-1}$ in adult cattle (Millemann et al. 2007).	

CONCLUSIONS

Blindness could occur in young calves with low concentration of vitamin A in the serum. This presentation could occur when beef cows are fed with diets with low concentration of vitamin A during the last half gestation period.

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