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Three outbreaks of vitamin A deficiency in cattle: clinical and diagnostic characterization[1]

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ABSTRACT.- Micheloud JF, Medina-Vallejo DMN, Avellaneda-Cáceres A, Singh J, Sandoval GV, Aguirre LS, Barbeito CG, Uzal FA. **Three outbreaks of vitamin A deficiency in cattle: clinical and diagnostic characterization**. *Pesquisa Veterinária Brasileira 47:e07873, 2027*. Instituto Nacional de Tecnología Agropecuaria, Centro de Investigaciones Agropecuarias, Instituto de Investigación Animal del Chaco Semiárido, Área de Salud Animal "Dr. Bernardo J. Carrillo", Ruta Nacional 68 Km 172 (A4403), Cerrillos, Salta, Argentina. E-mail: micheloud.juan@inta.gob.ar

Three outbreaks of vitamin A deficiency were investigated in feedlot and dairy cattle in northwestern Argentina. Clinical signs included night blindness, corneal opacity, dilated pupils, and impaired menace and pupillary reflexes. In one dairy herd, newborn calves were weak and blind at birth, and their dams exhibited a high rate of retained placentas. Autopsy findings were unremarkable in animals of the three outbreaks. However, histologic examination revealed a marked reduction or absence of mucoid epithelial cells accompanied by expansion and multilayering of the ductal epithelium, which was composed predominantly of closely packed cuboidal to polygonal parotid cells. This finding is consistent with impaired differentiation associated with sustained vitamin A deficiency. Serum vitamin A concentrations were markedly decreased in affected animals, confirming herd-level deficiency. Outbreaks occurred in herds receiving cereal-based rations and/or having limited access to green forage. These findings underscore the continuing risk of vitamin A deficiency in intensive production systems. In addition to documenting the recurring occurrence of hypovitaminosis A in intensive production systems, this study provides a detailed characterization of parotid gland lesions, including marked

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reduction of mucoid epithelial cells and expansion and multilayering of the ductal epithelium, representing an underrecognized histopathological feature of naturally occurring hypovitaminosis A.

INDEX TERMS: Hypovitaminosis A, cattle, parotid gland, salivary gland, histopathology, blindness.

RESUMO.- [**Três surtos de deficiência de vitamina A em bovinos: caracterização clínica e diagnóstica**]. Três surtos de deficiência de vitamina A foram investigados em bovinos de confinamento e leiteiros no noroeste da Argentina. Os principais sinais clínicos incluíram cegueira noturna, opacidade corneana, midríase e comprometimento dos reflexos de ameaça e pupilar. Em um dos rebanhos leiteiros, bezerros recém-nascidos apresentavam fraqueza e cegueira ao nascimento, enquanto suas mães exibiam elevada frequência de retenção de placenta. Os achados de necropsia foram inespecíficos em todos os animais dos três surtos. Entretanto, o exame histopatológico revelou acentuada redução ou ausência de células epiteliais mucosas, acompanhada de expansão e multilayering do epitélio ductal, que era predominantemente constituído por células cuboides a poligonais estreitamente agrupadas da glândula parótida. Esse achado é compatível com comprometimento da diferenciação epitelial associado à deficiência prolongada de vitamina A. As concentrações séricas de vitamina A estavam marcadamente reduzidas nos animais afetados, confirmando a deficiência em nível de rebanho. Os surtos ocorreram em propriedades que utilizavam dietas à base de cereais ou com acesso limitado a forragem verde fresca. Esses resultados reforçam que a deficiência de vitamina A continua sendo um importante problema em sistemas intensivos de produção. Além de documentar a ocorrência recorrente da hipovitaminose A nesses sistemas, este estudo apresenta caracterização detalhada das lesões na glândula parótida, incluindo acentuada redução das células epiteliais mucosas e expansão e estratificação do epitélio ductal, representando uma característica histopatológica sub-reconhecida da hipovitaminose A de ocorrência natural.

TERMOS DE INDEXAÇÃO: Hipovitaminose A, bovinos, glândula parótida, glândula salivar, histopatologia, cegueira.

INTRODUCTION

Vitamin A is an essential, fat-soluble nutrient that plays a role in many physiological processes, including vision, epithelial maintenance, immune function, skeletal development, and reproduction (Saari et al., 2016). Since ruminants cannot synthesize this vitamin, they must obtain it through dietary intake of provitamin A carotenoids. The most important carotenoid is β-carotene, which is abundant in fresh green forages. After ingestion, carotenoids are converted to vitamin A in the small intestine and are then stored in the liver as retinyl esters (Underwood & Suttle, 1999).

Hypovitaminosis A typically develops when animals consume diets low in β-carotene over an extended period. This can occur during droughts, seasonal forage shortages, or when feeding cereal-based rations or weathered hay (Underwood & Suttle, 1999). Clinical signs of vitamin A deficiency appear once hepatic reserves are depleted and vary with age. Calves may exhibit impaired growth, exophthalmos, diarrhea, convulsions, and blindness, while adult cattle may demonstrate ataxia, convulsions, and blindness (Donkersgood & Clark, 1988; Moore, 1961). Pregnant cows on vitamin A–deficient diets may give birth to calves with congenital hypovitaminosis A. This condition has been documented in several continents, including South America (Sosa et al., 2024). Newborn calves are particularly susceptible to hypovitaminosis A because their hepatic reserves are minimal, and they rely entirely on colostrum and milk for vitamin A intake (Zanker et al., 2000).

Extended drought, high temperatures, and nitrogen fertilization can significantly reduce β-carotene concentrations in pastures, and this predisposes cattle to vitamin A deficiency (Lindqvist et al., 2014; Underwood & Suttle, 1999). However, intensive confinement systems may pose an even greater risk if no vitamin A is supplemented to the diet, because it limits the use of fresh green forage (Sosa et al., 2024; Van Donkersgoed & Clark, 1988).

A presumptive diagnosis of hypovitaminosis A is based on epidemiology and clinical and post-mortem findings; confirmation relies on determination of vitamin A concentrations in serum and/or liver (Underwood & Suttle, 1999; Hill et al., 2009; Parker et al., 2017).

This report describes three outbreaks of vitamin A deficiency in cattle raised under intensive production conditions. It emphasizes the disease's diagnostic, epidemiological, and pathological features.

MATERIALS AND METHODS

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Ethical approval. The Animal Health Diagnostic Service of the Instituto Nacional de Tecnología Agropecuaria (INTA) performed clinical evaluations, animal handling, sample collection, euthanasia, necropsies, and tissue processing as part of their routine veterinary diagnostic activities. Euthanasia was only carried out when justified for diagnostic purposes and followed the laboratory's standard operating procedures. All procedures complied with Argentine Animal Protection Law (Law No. 14,346) and internationally recognized animal welfare standards. These standards include the principles established in European Union Directive 2010/63/EU on the protection of animals used for scientific purposes.

Three herd investigations were conducted following reports of clinical problems compatible with vitamin A deficiency (Table 1). Case 1 involved feedlot steers (10–12 months of age) presenting poor weight gain, visual impairment, and occasional neurological signs after prolonged confinement without vitamin–mineral supplementation. Case 2 corresponded to a dairy herd reporting weak, blind newborn calves, in which vitamin–mineral supplementation had been withdrawn during the dry and prepartum periods. Case 3 included Holstein replacement heifers (8–12 months of age) showing growth retardation and visual impairment despite receiving a vitamin–mineral premix. Detailed information on herd management and nutritional profiles is provided in Table 1.

Each investigation began with a field visit to the affected farms. During these visits, the herds were visually examined, and animals exhibiting neurological signs, visual impairments, or other abnormalities were identified, separated from the herd and examined clinically.

Blood samples were collected from a total of 33 clinically affected animals across the three outbreaks using tubes wrapped in aluminum foil to prevent photodegradation of retinol; samples from clinically healthy control animals were not analyzed for serum vitamin A. Samples were maintained at 4°C during transport and centrifuged upon arrival to obtain serum (Sosa et al., 2024). Serum vitamin A concentrations were quantified by liquid chromatography–tandem mass spectrometry (LC-MS/MS) in a private diagnostic laboratory (AZAR S.A., Argentina), using an internally validated method with external calibration curves and certified quality control materials included in each analytical batch, following the general principles of isotope dilution LC-MS/MS methods previously validated for serum retinol quantification (Yu et al. 2019). Animals with serum vitamin A concentrations <25 µg/dL were classified as having low vitamin A status, using 25 µg/dL as the reference threshold based on previously reported serum vitamin A concentrations in cattle (Frye et al., 1991).

One clinically affected animal from each outbreak was euthanized and a necropsy was performed. Samples of liver, brain, lungs, optic nerves, eyes and parotid salivary glands, were collected and fixed in 10% neutral buffered formalin before being routinely processed for the production of 4 µm thick, hematoxylin and eosin-stained sections.

Selected sections of parotid gland from necropsied animals were evaluated for cellular proliferation by immunohistochemistry targeting proliferating cell nuclear antigen (PCNA). Parallel samples from the same tissue of clinically healthy animals, collected during the same period as the last outbreak and submitted to the laboratory from pasture-based systems, were processed simultaneously as controls. Four micrometer sections were mounted on positively charged slides, deparaffinized, rehydrated, and treated to block endogenous peroxidase activity and nonspecific binding. Antigen retrieval was performed in citrate buffer (pH 6.0). Slides were incubated overnight at 4 °C with a rabbit monoclonal anti-PCNA antibody (1:1000), followed by a biotin–streptavidin–peroxidase system with DAB and hematoxylin counterstaining. Negative controls omitted the primary antibody, and small intestine served as an external positive control. Histological controls consisted of parotid glands from four animals (two calves and two heifers) submitted to the diagnostic service for reasons unrelated to vitamin A deficiency, originating from pasture-based herds with no history of the disease. Serum vitamin A concentrations were not available for these animals.

RESULTS

Clinical findings and epidemiology

In Case 1, ten finishing steers exhibited blindness and transient neurologic episodes, characterized by sudden collapse with marked hyperextension of the limbs and neck, brief apnea, and spontaneous recovery within seconds; these signs were more severe when the animals were moved. In Case 2, the clinical evaluation focused on newborn calves. At the time of the visit, approximately 7 calves were present in the rearing unit, of which a subset of five calves showing clinical signs was examined and sampled in detail (Table 2). Affected calves exhibited blindness, absent bilateral pupillary light reflex, and lack of menace response. Based on farm records, the outbreak was associated with significant reproductive problems, with an estimated 20% of calves showing congenital blindness and 80% of affected dams experiencing placental retention. In Case 3, 17 of 100

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replacement heifers examined showed impaired vision under low-light conditions (night blindness), accompanied by an unsteady gait and disorientation within the pen. Ocular examination revealed mild exophthalmos, abnormal pupillary reflexes, absence of menace response, and a greenish to bluish tinge to the globe due to changes in corneal transparency. Some animals also displayed mild corneal opacities and conjunctival dryness.

Gross and histopathological findings

All three necropsies revealed no significant macroscopic lesions. Histopathological examination consistently identified alterations in the parotid salivary glands of all animals. The ductal epithelium showed a marked reduction or complete absence of mucoid cells. In contrast, the ducts were lined by a multilayered epithelium composed of closely packed cuboidal to polygonal cells, with round to oval nuclei, indistinct cell borders, and eosinophilic cytoplasm. The epithelium showed increased cellularity and loss of normal polarity, without evidence of keratinization. Histopathological examination of the ocular globes was unremarkable, with no evidence of retinal degeneration. However, the optic nerves exhibited mild, diffuse rarefaction, with subtle loss of nerve fibers and mild gliosis. Occasional features suggestive of axonal degeneration were observed, whereas gitter cells and overt myelin loss were not conspicuous. Overall, the lesions were considered mild in severity. The severity of these lesions varied among cases, being more pronounced in necropsied animals from Cases 1 and 3, whereas only mild changes were observed in Case 2.

Immunohistochemical findings

Immunohistochemical staining for proliferating cell nuclear antigen (PCNA) revealed marked variation in nuclear labeling intensity across affected areas of the parotid salivary glands. Lesions were diffuse, although with varying degrees of severity between regions. Areas with milder changes exhibited scattered or absent PCNA-positive nuclei, consistent with low proliferative activity. In contrast, regions showing more pronounced ductal epithelial hyperplasia displayed numerous strongly immunoreactive nuclei, predominantly within the ductal epithelium and in areas of squamous metaplasia. Overall, a high proportion of epithelial cells showed nuclear positivity, indicating increased cellular proliferation. These observations are qualitative; no labeling index was calculated and no statistical comparison with control tissue was performed, and results should be interpreted accordingly.

Serum Vit. A level

Serum vitamin A concentrations were below the reference threshold in all animals evaluated at the time of sampling in Cases 1 and 2, whereas Case 3 showed greater variability, although most samples were also below the reference threshold (see Table 2 for details).

DISCUSSION

The outbreaks described in this report highlight the clinical, epidemiological, and pathological variability associated with vitamin A deficiency in cattle. Hypovitaminosis A is a well-recognized nutritional disorder, particularly under conditions of prolonged reliance on low β-carotene feeds (Underwood & Suttle, 1999). However, the present cases demonstrate that it remains relevant in modern production systems and can cause significant morbidity in animals of different ages and physiological stages. This is especially important in intensive systems, where limited access to fresh, carotene-rich forage increases the risk of deficiency (Sosa et al., 2024; Van Donkersgoed & Clark, 1988). These findings emphasize the importance of consistent vitamin A supplementation in both dairy and feedlot systems.

The clinical manifestations observed across outbreaks were consistent with previously described signs of hypovitaminosis A, including visual impairment in calves and adult cattle (Hill et al., 2009; Van Donkersgoed & Clark, 1988; Moore, 1961). The spectrum ranged from night blindness and corneal changes to complete bilateral blindness. In one outbreak, transient neurological episodes were also recorded. Neurological signs are rarely reported in cattle with vitamin A deficiency, with only a few documented cases in the literature (Cagnotti et al., 2025). These findings further support neurological involvement in bovine hypovitaminosis A. This condition has been associated with increased intracranial pressure, resulting from impaired cerebrospinal fluid circulation or stenosis of the optic canal in chronic deficiency (Moore, 1961). Optic nerve degeneration in hypovitaminosis A has been attributed to increased cerebrospinal fluid pressure and compression within the optic canal secondary to abnormal skull remodeling during growth. The occurrence of congenital blindness and a high rate of placental retention in Case 2 further highlights the well-recognized susceptibility of pregnant cows and neonates, likely

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related to the low hepatic reserves of newborn calves and their dependence on adequate maternal vitamin A intake during gestation (Swanson et al., 2000; Sosa et al., 2024). Vitamin A plays an important role in reproductive function, including maintenance of the uterine epithelium and normal placental development (Underwood & Suttle, 1999). Placental retention has previously been reported in vitamin A-deficient cattle, likely reflecting impaired epithelial integrity at the maternofetal interface (Frye et al., 1991). The high proportion of dams with retained placentas in Case 2 may therefore represent an additional manifestation of the herd-level deficiency documented in this outbreak.

In the present investigation, no significant gross lesions were observed in any of the three necropsied animals, underscoring the importance of histopathology in the diagnosis of vitamin A deficiency. The parotid salivary glands consistently exhibited a marked reduction in mucoid epithelial cells accompanied by expansion of the ductal epithelium, which became multilayered and composed of closely packed cuboidal to polygonal cells with eosinophilic cytoplasm and loss of normal epithelial polarity. No keratinization was observed. This pattern likely reflects impaired differentiation of mucus-producing epithelial cells under sustained vitamin A deficiency. Parotid duct metaplasia in bovine vitamin A deficiency was first described by Nielsen et al. (1966) and further characterized ultrastructurally by Hayes et al. (1970), who demonstrated defective differentiation of mucus-producing epithelial cells in the absence of keratinization. The parotid lesion itself is therefore not a novel finding; rather, the present report contributes a detailed characterization of this lesion in naturally occurring, spontaneous cases, with emphasis on the marked reduction in mucoid epithelial cells and the expansion and multilayering of the ductal epithelium. This pattern may represent a useful and underrecognized histological feature of naturally occurring bovine hypovitaminosis A. The absence of keratinization in these lesions may further distinguish them from the more advanced squamous metaplasia described in severe or prolonged vitamin A deficiency.

In contrast, more advanced stages of hypovitaminosis A have been associated with squamous metaplasia, epithelial stratification, and keratin accumulation within salivary ducts (Moore, 1961; McDowell, 2000; Baldwin et al., 2012). The absence of overt keratinization in the present cases, together with the multilayering and increased cellularity of the ductal epithelium, suggests active epithelial remodeling rather than fully developed squamous metaplasia. The limited number of reports describing salivary gland lesions may be related to the fact that this tissue is not routinely collected during necropsy.

Visual deficits were the most frequent clinical finding in the present study. Peripheral blindness and papilledema are recognized as early manifestations of vitamin A deficiency in young, growing animals and may be reversible if appropriate supplementation is instituted promptly (Cagnotti et al., 2025; Cantile and Youssef, 2016). A similar pattern was observed in the present study. Although funduscopic examination was not performed in all animals, those evaluated showed variable degrees of papilledema and recovered vision following treatment. In contrast, optic nerve lesions identified histologically, particularly in animals from Cases 1 and 3, were associated with persistent visual impairment, supporting the concept that optic nerve atrophy represents a more advanced and irreversible stage of the disease. Notably, the severity of the visual impairment observed clinically exceeded what would be expected from the relatively limited ocular lesions found on histopathology; this discrepancy is consistent with reports indicating that blindness in hypovitaminosis A is primarily attributable to increased intracranial pressure and optic nerve compression rather than to primary retinal or corneal disease, which may explain why gross ocular changes were mild despite profound clinical blindness. In agreement with previous reports (Mayhew and MacKay, 2022), neurologic manifestations such as episodic seizures were also observed and resolved after vitamin A supplementation, further reinforcing the link between hypovitaminosis A and reversible neurologic dysfunction.

Immunohistochemical labeling for proliferating cell nuclear antigen (PCNA) further supported these findings, demonstrating increased nuclear immunoreactivity within hyperplastic ductal epithelium compared with less affected areas, consistent with enhanced epithelial proliferative activity. This proliferative pattern corroborates the histologic interpretation of active ductal remodeling. Because quantitative assessment of proliferative activity was not performed, PCNA immunolabeling should be interpreted as supportive evidence only. Epithelial hyperplasia affecting multiple tissues, including salivary glands, is a well-recognized lesion associated with vitamin A deficiency (McDowell, 2000; Baldwin et al., 2012; Sosa et al., 2024).

Collectively, these findings indicate that the parotid gland represents a sensitive histological indicator of vitamin A depletion. The selective loss of mucoid cells and expansion of closely packed cuboidal to polygonal ductal epithelial cells reinforce the diagnosis and highlight the diagnostic value of salivary gland histopathology. Serum vitamin A concentrations were markedly reduced in most sampled animals, supporting the clinical and pathological findings. In Cases 1 and 2, all samples fell below the reference range (25–60 µg/dL) (Frye et al.,

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1991; Sosa et al., 2024), indicating a sustained herd-level deficiency. Case 3 showed greater variability, suggesting a mixed population with different exposure levels or dietary histories, despite the reported use of a vitamin-mineral premix, whose formulation, intake, and storage conditions could not be verified and may have been inadequate or improperly stored; however, more than half of the animals (9/16 = 56.25%) remained below the reference limit. Despite variability in serum retinol concentrations, the combination of compatible clinical signs, histopathological lesions, and the occurrence of multiple animals below the deficiency threshold supported a diagnosis of herd-level vitamin A insufficiency. Variable serum concentrations in Case 3 may reflect differences in feed access, duration of deficiency, or prior supplementation among animals. Although hepatic vitamin A concentrations were not determined, the combination of compatible epidemiology, clinical findings, histopathology, and markedly reduced serum vitamin A concentrations supported the diagnosis. Given the marked heterogeneity among outbreaks, case-specific values are presented rather than a pooled mean; means, standard deviations, and the percentage of samples below the reference limit for each case are reported in Table 2 (12 ± 10 μg/dL in Case 1, 13 ± 18 μg/dL in Case 2, and 23 ± 14 μg/dL in Case 3), all substantially below expected physiological values.

Overall, these outbreaks emphasize that vitamin A deficiency can occur in both feedlot and dairy systems, that clinical signs may be variable, and that gross lesions may be absent. Under these conditions, histopathology—particularly of the parotid gland—together with serum retinol determination, represents a valuable diagnostic approach. Preventive strategies should include regular monitoring of vitamin A status and appropriate dietary supplementation, especially in systems relying on conserved forages or cereal-based diets.

Limitations of this study include the small number of necropsied animals, the lack of serum vitamin A determinations in unaffected herd mates, the absence of hepatic vitamin A measurements, and the qualitative nature of the immunohistochemical evaluation. Despite these limitations, the combination of epidemiological, clinical, pathological, and biochemical findings strongly supported the diagnosis of hypovitaminosis A.

CONCLUSIONS

Vitamin A deficiency is still a significant nutritional issue in intensive cattle production systems in northwestern Argentina, affecting both feedlot and dairy cattle. The three outbreaks described here demonstrated that clinical presentation can vary according to the animal's age and physiological state. Symptoms can range from night blindness and corneal opacity to congenital blindness, weakness in newborn calves, reproductive problems and transient neurological signs.

Gross lesions were either absent or minimal, emphasizing the need for additional diagnostic tools. Consistent histological alterations were observed in the parotid salivary gland. These were characterized by the depletion of mucoid epithelial cells and the predominance of ductal epithelial metaplasia, with qualitatively increased PCNA immunoreactivity in affected ducts. This suggests active epithelial remodeling associated with vitamin A deficiency.

These findings support the value of including the parotid gland in necropsy protocols when vitamin A deficiency is suspected. Serum retinol determination remains essential for confirmation at herd level. Regular monitoring of vitamin A status and adequate supplementation are critical preventive measures, particularly for herds on cereal-based diets or with limited access to fresh green forage.

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Figures



Fig. 1. Calves showing corneal thickening, opacity, and mild exophthalmos (a), together with a close-up view of an affected eye displaying a blue-iridescent tapetal reflection (b) associated with reduced corneal transparency.



Fig. 2. Neurological signs observed in an affected steer during a spontaneous convulsive episode. (a) Animal walking prior to the episode. (b) Same animal in lateral recumbency during the convulsive episode. (c) Animal recovering in sternal recumbency. (d) Animal walking without evidence of neurologic signs shortly after recovery.

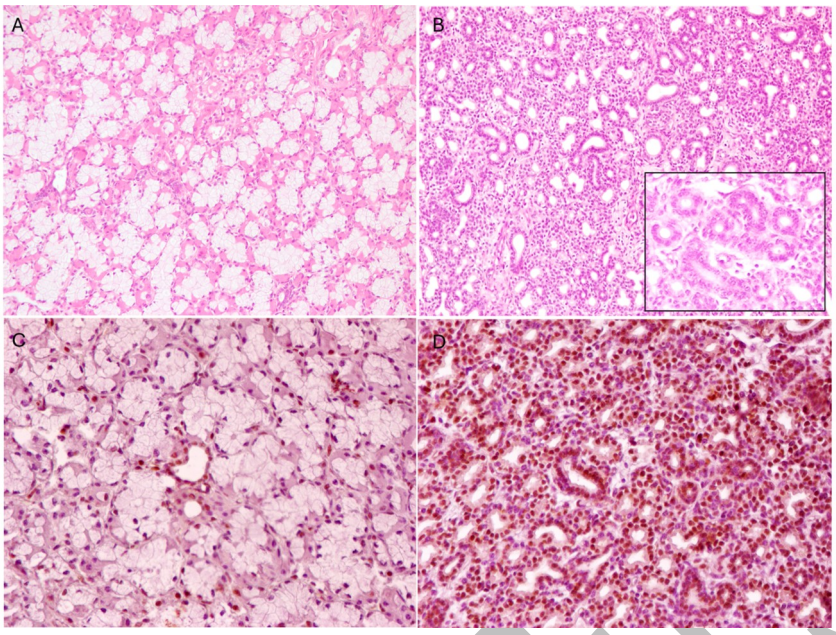


Fig. 3. Salivary gland. (a–b) Hematoxylin and eosin. (a) Control animal with preserved acinar architecture. (b) Calf with congenital vitamin A deficiency showing metaplasia of the ductal epithelium (inset) with loss of mucoid cells, together with disruption of glandular parenchyma and acinar disorganization consistent with epithelial injury. (c–d) Immunohistochemistry for PCNA (proliferating cell nuclear antigen). (c) Control animal with low nuclear immunoreactivity, restricted to scattered basal/proliferative cells. (d) Congenitally affected calf (necropsy case 2) with a marked increase in PCNA-positive nuclei within the glandular epithelium, indicating increased proliferative activity associated with tissue regeneration and remodeling. (20×).

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Table 1. Epidemiological and management characteristics of the three investigated outbreaks, detailing animal category, location, date, and herd management and feeding practices, including dietary composition and vitamin–mineral supplementation

Case	Animal category, location, and date	Herd management and nutritional profile
1	Feedlot steers (10–12 months of age), Rosario de la Frontera, Salta (6 November 2020).	The affected group consisted of 120 finishing steers that had been confined for 10 months. During the first month they were fed maize, followed by sorghum silage and grain for the remainder of the feeding period. No vitamin–mineral premix was included in the ration.
2	Newborn dairy calf, Trancas, Tucumán (19 August 2025).	The farm reported the birth of weak, blind calves. At the time of the investigation, seven calves were housed in the calf-rearing unit. The dairy farm milked approximately 120 cows. The herd was fed a ration based on maize silage, sugarcane bagasse hay, soybean expeller, and a vitamin–mineral premix. However, supplementation was discontinued during the dry and prepartum periods.
3	Dairy replacement heifers (Holstein, 8–12 months), Campo Quijano, Salta (3 September 2025).	The affected group included 100 replacement heifers fed maize silage, ground maize, soybean expeller, and hay, together with a vitamin–mineral premix.

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Table 2. Serum vitamin A concentrations by outbreak, expressed as number of animals sampled (N), mean ± standard deviation (SD), percentage of animals with concentrations below the reference limit (<25 µg/dL), and minimum–maximum values (min–max). Given the heterogeneity among outbreaks, values are presented by case rather than pooled

Case	N	Mean ± SD	% Below reference limit (<25 MG/DL)	Min-Max
1	12	12 ± 10 µg/dL	100 % (12/12)	3 – 23 µg/dL
2	5	13 ±18 µg/dL	100% (5/5)	0-21 µg/dL
3	16	23 ±14 µg/dL	56.25% (9/16)	11-59 µg/dL

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