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< Case Report >

Encephalopathy caused by maternal deficiency of vitamin A in a calf

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Abstract

Blindness was observed in five calves born from cattle fed only a commercial feed of growing stage and dried rice straws for about two years in a farm in Gyeongsangbuk-do province. Three of them died within a month after birth, and a body and sera of his mother and other 19 cattle were submitted for diagnosis. At necropsy, the calf was very weak and filled with cerebrospinal fluid in the cerebrum. Any histopathological lesion including atrophy of death of optic nerve cells was not observed, but the irregular proliferation such as lace pattern of choroidal cells and lymphocytic infiltration just below choroid was observed. No pathogen was detected as a result of the etiological tests on the internal organs of calves and bloods. In addition, the levels of serum vitamin A in different affected and his mother cattle were all lower than normal. Finally, we determined this case as an encephalopathy caused by maternal vitamin A deficiency in a calf. This report is an extreme example of how important it is to supply adequate s diets and a good quality of hay for each stage of growth in cattle.

Key words : Blindness, Cattle, Encephalopathy, Serum vitamin A, Vitamin A deficiency

INTRODUCTION

Vitamin A is not synthesized by vertebrates and depends essentially on beta-carotene of green plants in diets (He et al, 2012). A deficiency of vitamin A can be caused by dietary deficiency or decreased intestinal absorption, and it has been frequently reported in animals (Radostits et al, 2007; He et al, 2012; Parker et al, 2017). The clinical signs of this deficiency include weakness, ataxia, blindness, xerophthalmia, convusions, and syncope in calves and blindness and production of dead or weak calves in adult cattle (Radostits et al, 2007; Maxie, 2016).

The gross pathologic findings at necropsy are rarely observed, so the final diagnosis of this disease is made by taking history and clinical sings into consideration and verifying low levels of serum vitamin A. The normal levels of serum vitamin A in calves and adult cattle were reported to be 25～35 μg/dL and 40～50 μg/dL, respectively, with a level of 20 μg/dL or less usually considered a vitamin A deficiency (Millemann et al, 2007; Kang et al, 2017). This disease should be differentiated from diseases that can show both neurological signs and visual loss, especially polioencephalomalacia and lead poisoning (Yoon et al, 2003).

In calves, congenital defects have been known to be common in the offspring of deficient mother-animals and limited to blindness due to optic nerve constriction and encephalopathy (Mason et al, 2003; Radostits et al, 2007). However, the encephalopathy associated with blindness caused by vitamin A deficiency has been uncommonly reported in Korea (Yoon et al, 2003; Kang et

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al, 2017). This report describes a case of congenital encephalopathy in a calf born from a mother provided with vitamin A deficient diets for a long period of time.

CASE

In the spring of 2018, five calves with signs of blindness were born on a farm in Gyeongsangbuk-do province. Three of them died within a month. A body and sera of his mother and other 19 cows were submitted for diagnosis. In this farm, 71 adults, 5 growers, and 18 calves were raised and sign of blindness was observed only in the calf stage. Grossly, no specific lesions were observed in the eyeballs and the internal organs when the abdominal and thoracic cavity opened. However, the surface of the cerebrum was weak and depressed, and large spaces filled with cerebrospinal fluid was observed when the brain was incised in half (Fig. 1). After necropsy, the tissues were fixed and stained with hematoxylin and eosin staining (Kim et al, 2017). Histopathologically, cell damages of the internal organs and optic nerve were not observed. However, severe atrophy of cerebral parenchymal cells, irregular proliferation such as lace pattern of choroidal cells, and lymphocytic infiltration just below choroid were observed (Fig. 2). Bacteria and viruses were also checked for tissues and blood samples according to standard diagnostic guidelines (APQA, 2017). Viruses that have been tested include bovine viral diarrhea virus, bovine herpesvirus-1, bovine herpesvirus-5, Aino virus, Akabane virus, Chuzan virus, bovine ephemeral fever virus, and Ibaraki virus. As a result, no causative agent was isolated and detected. Levels of vitamin A in sera of the mother cow and affected cattle were measured to be 2.0 μg/dL to 17 μg/dL, all lower than the reference value (EONE Laboratories, Korea) (Table 1).

DISCUSSION

Newborn calves are forced to receive vitamin A only through colostrum and milk. This calf submitted before being weaned relied entirely on colostrum and milk for vitamin A. In history-taking, this farmer stated that all cattle, including pregnant cows, had been fed only a commercial feed of growing stage and dried rice straws without supplements for about two years. Epidemiologic

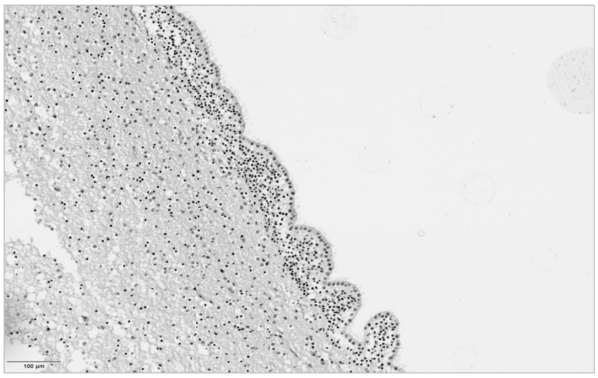

Fig. 2. Histopathological findings of brain. Meningitis infiltrated lymphocytes in cerebrum and lacework-shape of meninge under ependymal cell lining. H&E. Bar=100 μm.

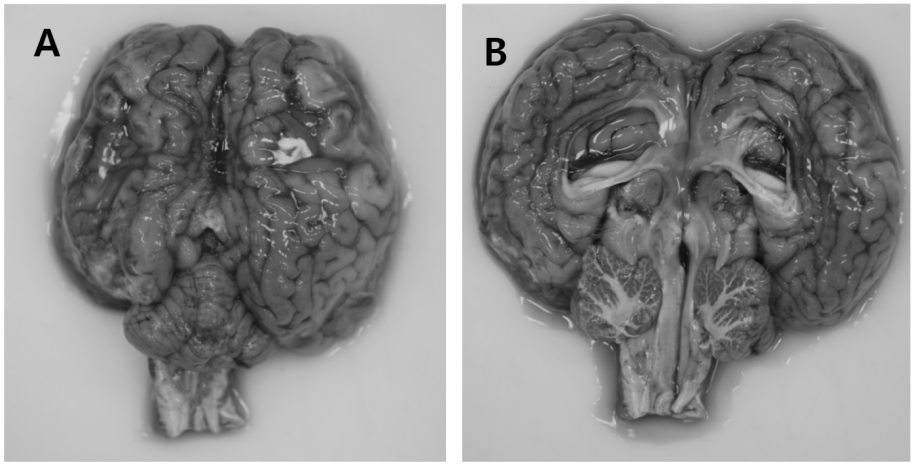

Fig. 1. Macroscopic findings of brain. (A) Weak and depressed surface of the cerebrum. (B) Space filled with cerebrospinal fluid in the cut surface of cerebrum.

Table 1. The serum levels of vitamin A in mother cow and affected cattle

Group	Serial No.	Serum vitamin A (μg/dL)*
Mother cow		8
Affected cattle	1	6
	2	10
	3	4
	4	9
	5	6
	6	14
	7	3
	8	2
	9	5
	10	6
	11	17
	12	7
	13	2
	14	8
	15	5
	16	4
	17	7
	18	2
	19	5

*The reference range of vitamin A levels in serum is 25～60 μg/dL.

surveys on this farm also revealed that frequent diarrhea in young and adult cattle, loss of sight in calves, and bone fractures in two cattle have occurred for a long time. It has been reported that signs of severe deficiency may appear in the suckling calves within 2～4 weeks of birth if given a ration low in carotene or vitamin A during pregnancy (MSD, 2019). The signs such as diarrhea and bone fracture were consistent with reports of vitamin A deficiency indicating incoordination of bone growth and atrophy of all epithelial cells (Radostits et al, 2007). It has also been reported that maternal deficiencies of vitamin A are characterized by herd outbreaks in calves (Van der Lugt and Prozesky, 1989; Radostits et al, 2007). They are mainly born dead, born alive but blind and weak and died within 1～3 days after birth, and had ocular abnormalities.

In the case of vitamin A deficiency reported in Korea, optic atrophy or retinal degeneration associated with loss of sight was observed (Yoon et al, 2003; Kang et al, 2017). Because this case was submitted dead, it was difficult to observe those lesions, but gross hydrocephalus and histopathologic encephalopathy were observed. The characteristic pathologic findings of polioencephalomalacia were not observed. Infectious diseases were also excluded because no causative agents for diarrhea and neurologic signs were isolated or detected. We finally measured the vitamin A in sera of the mother and other affected cattle to demonstrate the deficiency. The serum levels of vitamin A were 8 μg/dL in a mother cow and 2 μg/dL to 17 μg/dL in the affected cattle, indicating severe deficiency. Unfortunately, in this case, we could not measure the vitamin A content in the feed.

Limiting vitamin A intake in cattle has been reported to contribute to muscle fat accumulation, or marbling (Kruk et al, 2018). So, there have been farmers intentionally lowering or limiting vitamin A feeding in order to produce marbled beef cattle in Korea. However, the farmer of this case did not know that he had to deliberately lower it, but that he would have to feed the appropriate ration and hay for each stage of breeding. Cattle that are fed a diet containing little or no green forage are at risk for developing signs of vitamin A deficiency (Yoon et al, 2003). Furthermore, vitamin A needs during pregnancy in cows are more than twice as much as calves (Radostits et al, 2007). As a result, the pregnant cows in this farm, fed only the commercial feed of growing stage and dried rice would have been insufficient vitamin A.

To date, there have been few reports of vitamin A deficiency in calves born to dams fed a deficient ration in Korea. This report presents the pathological features of encephalopathy caused by maternal vitamin A deficiency in a calf and low levels of serum vitamin A in cows raised on the same farm. In addition, this case suggests that proper diet ration should be fed at the stage of growth and vitamin A deficiency should be considered if there are clinical signs such as blindness and diarrhea in cattle farm.

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