REPEATED OCCURRENCE OF AKABANE AND CNS DEFECTS IN CALVES

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INTRODUCTION

Akabane virus was first confirmed in 1975 (2) as a causative agent of the big outbreak of abortion, arthrogryposis and hydranencephaly in 1972-73 in Japan. Akabane vaccine was developed soon after the virus discovery. However, Akabane disease has occurred several times thereafter at the interval of two to five years. No vaccination and early change of cattle generation will be the main reason.

In this report, the recent outbreak of calves with central nervous system (CNS) defects was investigated in comparison with the past Akabane and Chuzan (1) disease.

MATERIALS AND METHODS

Calves with congenital defects were collected throughout the year by Kagoshima University. For the defective calves, the breed, sex, date of birth, condition at birth, clinical findings and prognosis

were investigated. At necropsy, pathological examination was performed. For their dams, the parity and vaccination by Akabane virus were investigated. The serums obtained from dams and calves were freezed and later virologically analyzed.

RESULTS

During Dec. 1990 and May 1991, 114 calves with congenital defects were collected in Kagoshima prefecture. Among them, CNS defects were found in 74 cases (65%) as follows; hydranencephaly - 60 cases, cerebellar hypoplasia with internal hydrocephalus - 10 cases and only nervous signs without any macropathological findings - 4 cases.

As shown in Fig. 1, the distribution of birth place was found elsewhere for



Figure 1. Area and number of calves with CNS defect (hydranencephaly, cerebellar hypoplasia, nervous sign in order) in Kagoshima (n = 74).

	Breed		Sex				
	JB	Hol	JBxH	Male	Female	Total	%
Hydranencephaly	45	10	5	29	31	60	81
Cerebellar Hypoplasia	9	1	0	5	5	10	14
Nervous Sign	_3	1	0	4	0	4	5
Total	57	12	5	38	36	74	100
%	77	16	7	51	49		

hydranencephaly and sporadically for cerebellar hypoplasia and nervous sign. The breed of calves (Table 1) with hydranencephaly belonged to Japanese black cattle (45 cases, 75%), Holstein cattle (10 cases, 17%) and JB x Hol (dam is Hol) (5 cases, 8%). Most of the calves with cerebellar hypoplasia and nervous sign belonged to

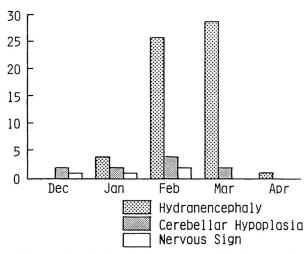


Figure 2. Number of cases and birth month.

Japanese black breed. There was no difference in the sex of the calves.

Seasonal incidence (Fig. 2) was characteristic. Calves with nervous sign were sporadically occurred during December to February. Calves with cerebellar hypoplasia were successively found during December to March. Calves with hydranencephaly were born between January and April, especially in February and March.

The parity of dams (Fig. 3) was also characteristic. In the calves with hydranencephaly, 59% (35 cases) of them was delivered in the first parity and 32% (19 cases) was delivered in the second parity. In contrast, calves with cerebellar hypoplasia were delivered widely between first and 9th parity. Calves with nervous sign were delivered between first and 4th parity.

In Akabane vaccination (Table 2), most of the dams which delivered the calves with hydranencephaly did not receive vaccination.

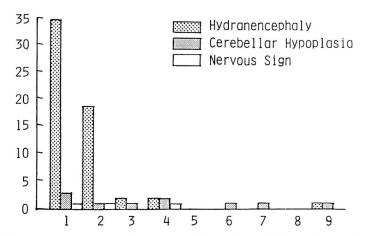


Figure 3. Number of cases and parity of dams (n = 72)

Table 2. Akabane vaccination to dam.

	Vaccinated	Unvaccinated	Unknown
Hydranencephaly	6	50	4
Cerebellar Hypoplasic	1 3	7	0
Nervous Sign	0	4	0
Total	9	61	4
%	12	83	5

Most of the calves with hydranencephaly were generally weak at birth and had temporary paresis and sucking difficulty. They were recovered after several days of nursing. However, they had the disturbance of vision and most of them experienced the left or right circular aimless walking. They can survive themselves, but the growth retarded markedly as the age increased.

Calves with cerebellar hypoplasia had no severe nervous signs like as opistotonus which was frequently found in Chuzan disease. They also walked aimlessly with the disturbance of vision. Most of them was found with complication of internal hydrocephalus. However, no hydranencephly was found at all.

Calves with nervous sign showed persistent paresis, opistotonus, spasms, nystagmus or swimming movement at birth. However, no macropathological change was found in CNS and other body system at necropsy.

Serologically, most of dams and calves showed the positive antibody titers against Akabane virus. However, a few colostrum-deprived calf serums from hydranencephaly cases were negative. Most of the calf serums from cerebellar hypoplasia cases showed the negative antibody titer against Chuzan and BVD-MD virus.

DISCUSSION

In comparison between typical Akabane (2) and Chuzan (1) disease, and hydranencephaly and cerebellar hypoplasia in this report (Table 3), there are some differences. In the calves with hydranencephaly in this report, neonatal weakness is generally not so strong. The farmers, therefore, nursed them very eagerly. However, these calves did not always respond to farmer's expectation because of severe retarded growth and abnormal behavior.

Table 3. Comparison between typical Akabane and Chuzan disease, and hydranencephaly and cerebellar hypoplasia in this time. Underlined items are different from typical one.

	Тур	ical	This	Time
	Akabane	Chuzan	Hyd.enc.	Cere.Hyp.
Breed	JB,Hol	JB	JB, Hol	JB
Birth Place	widely	locally	widely	locally
Month of Birth	Jan-Jun	Nov-Mar	<u>Jan-Apr</u>	Dec-Mar
Parity	low	all	low	all
Neonatal Weakness	strong	strong	<u>weak</u>	strong
Growth	poor	poor	<u>mild</u>	poor
Nervous Sign	rare	strong	<u>weak</u>	<u>weak</u>
Arthrogryposis	have	none	<u>none</u>	none
Hydranencephaly	have	have	have	none
Cerebellum	intact	hypoplasi	a intact	hypoplasia
Akabane Antibody	positive	negative	positive	positive
Chuzan Antibody	negative	positive	negative	<u>negative</u>

In the calves with cerebellar hypoplasia in this report, neonatal nervous signs were not so strong as those found in Chuzan disease. Farmers also hoped their growth. However, they did not grow enough, either.

During past 20 years, the outbreaks of the calves with CNS defects occurred 7 times in Kagoshima (Table 4). Akabane disease was confirmed as a cause in 5 times and Chuzan disease, in one time. If the cause of hydranencephaly in this time is Akabane virus, it is characteristic that no previous prevalence of arthrogryposis. This is as same as in Akabane disease of 1988. The possibility may be considered that Akabane virus has been modified during these period.

Chuzan disease was first found in 1985. However, no clinical case has occurred therafter, though Chuzan virus itself has been recovered from cow serum every year.

In conclusion, the outbreak of calves with CNS defects in 1990-91 is not a simple origin, but the complicated causes will be related. The calves with hydranencephaly were very similar to Akabane disease, but with some difference. The calves with cerebellar hypoplasia were not to be Chuzan or BVD-MD.

Table 4. Outbreaks of calves with CNS defect in Kagoshima during 1972 to 1991.

Year	Cause	Aug Sep Oct Nov Dec Jan Feb Mar Apr May Jun
1972-73	Akabane	Abortion
		Arthro.
		Hydran,
1977-78	Akabane	Arthro.
		Hydran.
1979-80	Akabane	Arthro.
		Hydran.
1982-83	Akabane	Arthro.
		Hydran.
1985-86	Chuzan	Cerebe,
1988	Akabane	Hydran.
1990-91	unknown	Nervous Sigr
	unknown	Cerebe.
	mostly Akabane	Hydran.

Arthro:: Arthrogryposis, Hydran:: Hydranencephaly

Cerebellar Hypoplasia

REFFERENCES

- Hamana, K. and Taura, Y. (1988) Proc.15th World Buiatrics Congress, Vol.2, 886-889
- 2. Kurogi, H. (1975) Arch. Virol., 47, 71-83

SUMMARY

The outbreak of calves with central nervous system (CNS) defects was observed in Kagoshima prefecture during Dec. 1990 and Apr. 1991. In the investigation of 74 cases, 60 calves had severe hydranencephaly, 10 had cerebellar hypoplasia with internal hydrocephalus, and 4 had only nervous signs.

Calves with hydranencephaly were collected from all over the prefecture. 75% of them were found in Japanese black cattle and 25% were found in Holstein cattle. There was no sex difference. Seasonal incidence was the feature and they were born between January and

April, especially in February and March. 59% of them was delivered in the first parity and 32% was delivered in the second parity. 89% of their dams did not receive Akabane vaccination.

Most of the calves with hydranencephaly were generally weak at birth and had temporary paresis and sucking difficulty. They were recovered after several days of nursing. However, they had the disturbance of vision and most of them experienced the left or right circular aimless walking. They can survive themselves, but the growth retarded markedly as the age increased.

Calves with cerebellar hypoplasia had no difference in the seasonal incidence and the parity. They had no severe nervous signs like as opistotonus which was frequently found in Chuzan disease,.

They also walked aimlessly with the disturbance of vision.

Most of the calves with hydranencephaly were positive in the serum antibody for Akabane virus, but some were negative. Most of the calves with cerebellar hypoplasia were negative in the serum antibody for Chuzan virus and BVD-MD virus.

During past 20 years, the outbreaks of the calves with CNS defects occurred 7 times in this area and the last one occurred in the spring of 1988. In conclusion, the calves with hydranencephaly were very similar to Akabane disease, but with some difference. The calves with cerebellar hypoplasia were not to be Chuzan disease or BVD-MD.