CONGENITAL CEREBELLAR HYPOPLASIA IN JERSEY CALVES

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Introduction

Congenital cerebellar hypoplasia has been reported in Hereford, Guernsey, Friesian, Shorthorn, Australian Illawarra Shorthorn (AIS), Hereford — AIS, and Ayrshire cattle (Johnson et al 1962; Howell and Ritchie 1966; Blood and Henderson 1968). In Western Australia this condition has also been seen in Murray Grey — Friesian and Angus cattle (Allen and de Chaneet, unpublished).

This communication records the occurrence of congenital cerebellar hypoplasia in Jersey calves in Western Australia, and discusses possible aetiologies.

History

The property involved was in the extreme south-west of Western Australia. The herd consisted of high grade Jerseys, and no “outside blood” had been introduced into the herd for 23 years. In 1972 the breeding herd consisted of 44 mature cows and 30 heifers born in 1971, the latter being all sired by one registered pure bred Jersey bull (bull A).

Two other registered pure bred bulls, both descendants of bull A, were purchased in 1972 from a neighbouring Jersey stud. The older of these 2 bulls (bull B) was mated with the cows, and the other (bull C) with the heifers. Mating commenced in June 1972, and the cows and heifers were run separately from this time until calving.

The first heifer calved on March 5, 1973; the calf was premature and was destroyed. Another heifer gave birth to a normal calf on March 25. Thereafter, 8 ataxic calves (4 males, 4 females) and 1 dead calf were born to heifers between April 1 and April 9, followed by 18 normal calves over the next 3 weeks. One heifer was apparently sterile. All cows except 2 gave birth to normal calves during the same period. The 2 exceptions produced calves with gross abnormalities considered to be unrelated to cerebellar hypoplasia. One calf was a cyclops, while the other lacked a tail and the distal portion of one hindlimb.

No ataxic calf had been born on the property in question, or on the neighbouring stud, prior to 1973, and none has been born in subsequent years. Bull C was culled, and in 1973 and 1974 the heifers born in 1971 were mated with bulls A and B. In 1975 an Angus bull was introduced.

Clinical Signs

The calves were affected from birth. Six could stand, but only with their legs straddled. While standing their heads were held low, and moved up and down rhythmically. These calves were ataxic, and this was most pronounced and accompanied by loss of balance and falling, when they were forced to run. The 2 calves that could not stand lay in lateral recumbency, and intermittently threw their heads and necks across their bodies while making thrashing, paddling movements with their legs. All affected calves were able to suck and bellow, had normal temperatures, and all except one that could not stand had normal eye reflexes. This particular calf was apparently totally blind.

Figure 1. Brain of an affected calf showing marked cerebellar hypoplasia. The right ventricle has been opened to show the concurrent hydrocephalus and marked reduction in thickness of cerebral cortex.
Pathology

The blind, recumbent calf and 2 ataxic calves that could stand were necropsied within 1 week after birth. All had marked cerebellar hypoplasia and bilateral hydrocephalus of varying severity (Figure 1). The blind calf was the most severely affected; no cerebellar cortex and only remnants of its cerebellar peduncles were present and the cerebral cortex was reduced to a thickness of approximately 3 mm. In none of the cases was the calvarium domed.

The main microscopic lesions were in the cerebellum. The folia were all generally narrower than normal, with both the molecular and granular layers being abnormally thin. The granular layer, which was generally deficient in cells, was almost absent in some areas, and the border between it and the molecular layer was not distinct. There was also an irregular distribution, and a severe reduction in the numbers, of Purkinje cells.

Serology and Virology

Blood was collected from 2 affected calves before they were suckled by their dams, and from the 8 heifers that gave birth to affected calves. Antibodies to Bovine Virus Diarrhoea — Mucosal Disease (BVD-MD) virus were present in the serum of both calves and 7 of the heifers. The serum of the eighth heifer was toxic to tissue cultures and could not be examined. Isolation of BVD-MD and other viruses was attempted from the blood clots submitted, but none could be isolated.

Diagnosis

A diagnosis of congenital cerebellar hypoplasia and hydrocephalus was reached based on the clinical signs and gross and microscopic pathology.

Discussion

This appears to be the first report of congenital cerebellar hypoplasia in Jersey calves. Clinically the disease resembles congenital ataxia of Jersey (Saunders et al. 1952), Shorthorn and Hereford (Hulland 1957) and Angus-Shorthorn calves (Young 1969). These latter 2 conditions, however, are not characterised by any obvious gross pathology. Also, Jersey calves with congenital ataxia take 1 to 2 weeks to develop clinical signs (Saunders et al. 1952), whereas the affected calves reported here were ataxic from birth.

Since cerebellar hypoplasia of calves was described in England by Innes et al. in 1940, it has been generally considered to be of genetic origin (Innes et al. 1940; Finnie and Leaver 1965; O'Sullivan and McPhee 1975). However, recent work has shown that a similar condition may result from infection of pregnant cows with the BVD-MD virus (Kahrs et al. 1970).

In the cases reported here, in which all the affected calves were the progeny of one bull and there was a history of in-breeding, the possibility of a genetic aetiology should be considered. Unfortunately, a thorough genetic analysis was not possible, and the farmer was not willing to re-mate the same heifers with Bull C.

If it is assumed that the condition reported here was hereditary, then the fact that approximately one-quarter of the heifers produced ataxic calves would suggest that a single autosomal recessive gene was responsible. As the affected calves were of both sexes, the gene would presumably be non-sex linked. The high incidence of affected calves would also suggest that the "mutant" gene had a high frequency in the affected herd, and was also present in the neighbouring stud. It would therefore be expected that the condition should have occurred as more than a single outbreak. As this was not the case, there must be some considerable doubt that this condition was of genetic origin.

The fact that the 8 affected calves were born within a short period of 9 days, while all normal calves were born before and after this period, casts further doubt on a genetic aetiology. On the other hand, it has been shown that the BVD-MD virus causes cerebellar hypoplasia only when it infects foetuses within a critical period of their gestation (Kahrs et al. 1970). Therefore, the birth of the affected calves over a short period of time would be expected if BVD-MD virus was the cause of the cerebellar hypoplasia reported here.

Infection of the bovine foetus by BVD-MD virus has been shown to cause foetal mummification (Kendrick 1971), ocular defects (Bistner et al. 1970), abortion (Kahrs and Ward 1967), partial alopecia (Kendrick 1971), premature births (Kahrs et al. 1970), stillbirths (Kendrick 1971), and cerebellar hypoplasia (Kahrs et al. 1970). The last 3 manifestations were all present in the herd described here.

The detection of neutralising antibody to BVD-MD virus in the precolostrum serum of 2 affected calves indicates that these animals, at least, were infected in utero by this virus (Kahrs et al. 1970; Horner et al. 1973). However, BVD-MD is considered to be enzootic throughout the
world (Kahrs et al 1970) and there is serological evidence that the virus is widespread in the cattle population of Australia (St George et al 1967). It is likely therefore, that a significant number of normal calves will have precolostrum serum neutralising antibodies to this virus, and this has been shown to be the case in Victoria (Horner et al 1973). For this reason the concurrent finding of precolostrum serum BVD-MD virus neutralising antibody and cerebellar hypoplasia does not necessarily indicate a cause-effect relationship. Nevertheless, of the 2 possible aetiologies considered in this report, foetal infection by BVD-MD virus seems the more likely.

Summary

Congenital cerebellar hypoplasia and hydrocephalus is reported in 8 Jersey calves. The possible relationship between this entity and BVD-MD virus is discussed.

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References


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BOOK REVIEW

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senting a static series of facts and directions and questions, but as a representation of the dynamic processes of the epidemiology and economics of disease.

The first chapter is a brief perspective on disease monitoring, the second (from J. H. Steele) deals with the development of disease surveillance, including its use in disease control that relates to human and animal health. It is an interesting review, spiced with some history. Some pragmatic aspects (Hugh-Jones) covers definitions, objectives, scales of project, information flow, evolution of monitoring schemes, duration, and disease recording. Monitoring for disease eradication deals chiefly with bovine brucellosis and tuberculosis. Gold puts the veterinary practitioner into the picture in a most interesting and useful account. From Denmark, Willenberg presents concepts and fundamentals of an integrated University system from animal disease date. Wildlife considerations note Newcastle Disease and Duck Virus Enteritis.

Cost-benefit aspects note that detecting foci of infection is a complex problem, and that "surveillance cannot be relaxed, in fact, it will probably have to be intensified until eradication is achieved." The Australian contribution is from Roger Morris and Dick Roe — it is one of the longest sections and deals with the use of computer simulation and monitoring in control programs (with particular reference to brucellosis and mastitis). A chapter from Hugh-Jones looks at brucellosis in some depth via a computer model. Calf mortality is a serious, and frustrating disease — Martin looks at a model of economic costs.

Most chapters have brief and useful bibliographies: but one misses some useful books, Uses of Epidemiology (J. N. Morris), Principles of Epidemiology (Taylor and Knowelden), Epidemiology (Fox, Hall and Elveback), Patterns of Animal Disease (Halpin) and Epidemiology: A Guide to Teaching Methods (Ed. Lowe and Kostrewski, from the International Epidemiological Association and World Health Organisation).

The book will interest most veterinarians, and is essential reading for those concerned with herd health programs — whether teacher, planner, field veterinary officer or practitioner.

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