Akabane Disease of Cattle in Australia

W. J. Hartley
Wallaceville Animal Research Centre
Upper Hutt
New Zealand

Introduction

Akabane disease before the recognition of its cause was locally called in Australia, Curly Calf Disease, or arthrogryposis and hydranencephaly (AG/HE) (1) (18) (4).

The AG/HE syndrome has been recognized also in cattle, sheep and goats in Israel (11) (14) and also in Japan (15).

Japanese workers (13) (8) later showed that serums from unsuckled calves with the AG/HE syndrome contained neutralizing antibodies to the Akabane virus (originally isolated from mosquitoes in Japan). This association was confirmed in Australia (5) (3) and in Israel (6). Japanese workers have also isolated Akabane virus from an aborted calf and also from the foetus of a cow that had recently seroconverted (9). These workers have also produced the clinical disease in some calves whose dams were inoculated with the virus between 76 and 96 days of pregnancy (10).

The state of knowledge of Akabane disease and the virus has been reviewed recently (12) and the detailed pathology of the naturally occurring outbreak seen in Japan has been reported (7). My contribution will be to review the epidemiology, clinical signs and pathology of the disease as seen in Australia.

Epidemiology

In Australia Akabane disease is largely confined to south eastern New South Wales (N.S.W.) where it has been seen over at least the last 40 years as a cause of periodic severe outbreaks of perinatal calf mortality. However, the same entity does occur sporadically in intervening years. Akabane virus has been isolated from the biting insect *Culcoides brevitarsis* on a number of occasions. It has also been isolated from the blood of sentinel cattle in areas where at the same time it has been isolated from *Culicoides* (17). It is considered that this species of *Culicoides* is responsible for the spread of Akabane virus amongst ruminants in Australia.

Outbreaks of Akabane disease are likely to occur in Australia when seasonal conditions favour insect multiplication and allow southerly spread of virus-infected *Culicoides* which feed on susceptible pregnant cows. This is what apparently happened in the outbreaks of 1964 and 1974. Sporadic cases or small outbreaks occur when non-immune pregnant cows are introduced into areas where the carrier insects are normally present, which is all of northern and north-eastern Australia, and in pockets in those areas where the insects have not penetrated for several years. The clinical disease is rarely seen in the northern areas because most of the cattle are exposed to the infection before reaching breeding age.

Clinical signs and pathology

This description is based on the many affected calves examined from N.S.W. in the 1964 and 1974 outbreaks and from sporadic cases obtained in the intervening years (3). In retrospect it is possible that some of the cases may have been caused by one or more other insect-borne viruses e.g. Aino (q.v.).

In general the clinical signs and pathology of Akabane disease depend on the gestational age of the foetus when exposed to the Akabane virus. For ease of description the syndrome has been split up into several distinct groups.

A. This part of the Akabane syndrome was seen only in a few calves and occurred in late summer/early autumn in calves whose mothers presumably became infected by the virus in late pregnancy. These cases were invariably followed by cases of AG/HE in later calving cows on the same property or in the same district. Affected calves were born alive, some were incoordinate, some were unable to stand. Pathologically there was a moderately severe acute non suppurative encephalomyelitis.

B. The few calves in this category were born later and showed either incoordination, flaccid paralysis or mild rigid fixation of limbs. Microscopically there was active Wallerian degeneration of all white fibre tracts of the spinal cord except the dorsal columns. There was also Wallerian degeneration of the ventral spinal nerves. These lesions were associated with loss of ventral horn motor neurones and a mild encephalomyelitis.

C. This group and group D were the most commonly seen manifestations of Akabane disease. In outbreaks cases were seen over a 4 to 5 month period during the winter months. Characteristically affected calves has arthrogryposis or rigid fixation of limbs and this frequently resulted in dystocia. Sometimes all four limbs were involved with fixed flexion, sometimes only the front limbs. Occasionally one or more limbs were fixed in extension. Several calves had fixed lateral flexion of the head and neck and there were a few cases of scoliosis. There was also atrophy of the musculature of affected limbs. Some calves with AG also had HE (q.v.). Microscopically in the cord there was severe loss of myelinated fibres in all tracts but the dorsal columns,

together with loss of most ventral horn neurones, marked loss of nerve fibres in spinal motor nerves and neurogenic muscle atrophy.

D. This important group was seen towards the end of an outbreak in late winter and spring. Affected calves were born alive, were dejected, walked poorly, were blind and some had a poor sucking reflex. Some had a slightly domed cranium. If able to suckle some survived for several months. Grossly most calves had cerebral hemispheres which were virtually completely replaced by a fluid filled sac—hydranencephaly. In some cases there were varying amounts of surviving subleptomeningeal brain parenchyma. The brain stem structures were usually present, sometimes in reduced amount and showed only focal cavitation of cerebro cortical white matter. The cerebellum was usually unaffected.

E. At the end of the outbreak in October/November—Spring— a variety of neurology abnormalities were seen in a few affected herds which included micrecephaly, cerebellar hypoplasia and arthrogryposis.

F. Many abortions and still births were reported during the outbreak on affected properties. Most were not examined pathologically, but some that were had AG/HE (16).

Discussion

Epidemiological, pathological and serological findings suggest that most of the perinatal losses seen in the 1974 outbreak of AG/HE in N.S.W. were due to infection by the Akabane virus. However, no serums were available for testing from groups A and B. Further several of the group E calves and their dams did not have antibodies to Akabane virus.

It is of interest that in a later small outbreak of AG/HE investigated in N.S.W. there were no antibodies to Akabane virus in affected calves but there were antibodies to Aino virus (2).

References

1. Blood, D.C.: 1956 Aust. Vet. J., 32:125. 2. Coverdale, O.R., D.H. Cybinski and T.D. St. George: 1978 Aust. Vet. J., 54:152. 3. Hartley, W.J., W.G. De Saram, A.J. Della Porta, W.A. Snowden and N.C. Shepherd: 1977 Aust. Vet. J., 53:319. 4. Hartley, W.J. and R.A. Wanner: 1974 Aust. Vet. J., 50:185. 5. Hartley, W.J., R.A. Wanner, A.J. Della Porta and W.A. Snowden: 1975 Aust. Vet. J., 51:103. 6. Kalmar, E., B.A. Peleg and D. Savir: 1975 Refuah Vet., 32:47. 7. Konno, S., M. Moriwaki and M. Nakagawa: 1982 Vet. Pathol. 19: 246. 8. Kurogi, H., Y. Inaba, Y. Goto, Y. Miura, H. Takahashi, K. Sato, T. Omori and M. Matumato: 1975 Arch;. Virol., 47: 71. 9. Kurogi, H., Y. Inaba, E. Takahashi, K. Sato, T. Omori, Y. Miura, Y. Goto, Y. Fujiwara, Y. Hatano, K. Kodama, S. Fukuyama, N. Sasaki and M. Matumoto: 1976 Arch. Virol., 51: 67. 10. Kurogi, H., Y. Inaba, E. Takahashi, K. Sato, K. Satoda, T. Goto, T. Omori and M. Matumoto: 1977 Infect. Immun. 17:338. 11. Markusfeld, O. and E. Mayer: 1971 Refuah Vet., 28: 51. 12. Matumoto, M. and Y. Inaba: 1980 Kitasato Arch. Exp. Med., 53:1. 13. Miura, Y., S. Hayashi, T. Ishihara, Y. Inaba, T. Omori and M. Matumoto: 1974 Arch. ges. Virsforsch., 46:377. 14. Nobel, T.A., U. Klopfer and F. Neumann: 1971 Refuah Vet., 28:144. 15. Omori, T., Y. Inaba, H. Kurogi, Y. Miura, K. Nobuto, Y. Ohashi and M. Matumoto: 1974 Bull. Off. Int. Epizoot., 81:447. 16. Shepherd, N.C., C.D. Gee, T. Jessep, G. Timmins, S.N. Carroll and R.B. Bonner: 1978 Aust. Vet. J., 54:171. 17. St. George, T.D., H.A. Standfast and D.H. Cybinski: 1978 Aust. Vet. J., 54:558. 18. Whittem, J.H.: 1957 J. Path. Bact., 73:375.

Paper presented at the XIIIth World Congress on Cattle Diseases, Durban, S. Africa, Sept. 17-21, 1984.