MYELOPATHY IN HOLSTEIN X GIR CALVES IN BRAZIL

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The central nervous system (CNS) is susceptible to a variety of insults including infectious, toxicologic and genetic disorders. Inherited disorders may be evident at birth or may not be apparent until later in the first year of life or beyond. Most neurological disorders are recognized in the dog and cat with fewer examples in farm animals (SUMMERS and others 1995). This paper contains the first report of the clinical and pathological features of a degenerative myelopathy in Holstein X Gir crossbred calves in Brazil.

An abnormal pattern of locomotion progressing to tetraparesis was observed in calves of the Holstein X Gir breed, some of which were presented to São Paulo State University Veterinary Hospital (Araçatuba-Brazil). In the 1997 breeding season, the farm had 24 births of which 7 calves (3 males and 4 females) were normal until 3 months when they suddenly showed an altered pattern of locomotion in the thoracic limbs. Fifteen to 22 days later these animals were permanently recumbent and by 5 months of age had succumbed to complications of their recumbency (pneumonia, anaplasma or babesia).

All the affected calves were sired by a 4-year-old Holstein bull introduced in the 1996 breeding season at which time he had no prior progeny. This bull was mated to Gir cows, producing Holstein X Gir offspring. In the following year, this bull was used to breed 3 cows on a second farm 10 miles from the first property; one of these 3 cows had a calf with the same disorder. We examined 5 animals over a period of four months, 3 of whom could walk when first presented to us while 2 were recumbent. The 3 ambulatory calves were alert and responsive, could stand unaided and walked with a distinct
overreaching-floating action of the thoracic limbs. The pelvic limbs were slightly awkward and swayed slightly. All calves could see, had no head tremors and did not have a base-wide stance. Within days these calves were unable to rise even when helped; respiratory complications developed and they died after 1 to 3 months hospitalization. Two other animals arrived at our hospital in recumbency but the veterinarian who had examined them at the farm reported that the nature and evolution of neurological signs were the same. These animals became recumbent when 3 to 4 months old and died two to 10 weeks later of the same complications. These calves appeared bright and responsive but were recumbent and when lifted made no limb movements. The thoracic limbs appeared contracted with a fixed, flexed carpus bilaterally and were possibly atrophied.

No hematological or biochemical abnormalities were present in the ambulatory calves and analysis of CSF in 2 animals following cisternal puncture revealed no abnormalities in protein or nucleated cell numbers. No evidence of BVD infection or in utero exposure to toxic materials were recognized nor were poisonous plants or chemicals found on the farms.

Five calves were evaluated post mortem and macroscopic abnormalities were confined to areas of pulmonary consolidation due to pneumonia in four of them. The brains from 3 calves as well as the spinal cord from 2 were removed and fixed in 10\% buffered formalin. Gross brain lesions were lacking. Microscopically, there were minimal changes in the brain of only one of the calves which consisted of small glial nodules in the basal nuclei and medulla. The spinal cord sections showed a chronic, diffuse myelopathy in both animals. The degeneration was bilateral and fairly symmetrical and affected white matter, mainly superficially in the lateral and ventral
funiculi and did not relate to any particular tract except perhaps for involvement of spinocerebellar pathways. Lesions were diffuse in all segments of the spinal cord although milder at the terminal lumbar area.

The lesions were best demonstrated with the Bielschowski silver stains for axons, which identified areas where axons had degenerated and so were of reduced density (Figure 1A and 1B). Phagocytosis of myelin debris by macrophages (gitter cells) was lacking, suggesting that these were old, inert lesions. However, subpial astrocytic scarring in lateral and ventral funiculi was still present in H&E sections and shown to better effect with GFAP and vimentin stains. These bilateral and symmetrical spinal cord white matter lesions were interpreted as a primary axonopathy.

Over two breeding seasons, we examined several Holstein X Gir calves of both sexes which abruptly developed a gait disorder at 3 months of age. These calves progressed to recumbency and then death from secondary infections. In the first herd, calves from 7 of 24 cows were affected while at the second property 1 of 3 offspring developed the disorder. All affected calves were sired by the same bull, the disorder appeared in the first breeding season that the bull was used, and it was encountered on two separate farms: this suggests an inherited disorder and possibly an autosomal recessive trait.

The onset of a gait disturbance at 3 months of age denies a congenital spinal cord malformation such as diplomyelia or diastematomyelia (SUMMERS and others 1995). The calves in this report have a degenerative spinal cord disorder interpreted as an axonopathy which progresses to the point of clinical expression at 3 months of age. Whether spinal cord degeneration is present at birth in a subclinical form will require
further investigation. In cattle, there are only a few reports of primary, inherited axonopathies and these have resulted in clinical signs from birth. It is significant that the Holstein-Friesian breed has previously been incriminated as is the case in this report. HARPER and HEALY (1989) studied a diffuse axonopathy in 19 Holstein-Friesian calves. Affected animals had severe clinical deficits and most were recumbent from birth. Neuropathological studies showed a diffuse axonopathy, most pronounced in the spinal cord with degeneration extending into spinal roots. In the Netherlands, a congenital tremor syndrome in male Holstein Friesian calves was recorded in which it was suggested that myelin degeneration may have been secondary to an axonopathy (BETHLEHEM and others 1992). Congenital axonopathies have been also recorded in the Brown Swiss (KWIECIEN and others 1995) and probably occur in other bovine disorders. Acquired axonopathies in cattle could result from chemical intoxications such as chronic organophosphate poisoning (PERDRIZET and others 1985), plant poisoning (HALL 1987) and possibly from nutritional diseases such as copper deficiency although enzootic ataxia and swayback are generally only recognized in lambs and goats.

In conclusion, we have encountered a novel degenerative disorder of crossbred calves in Brazil in which a Holstein bull is incriminated. Calves show evidence of cervical spinal cord disease at 3 months which soon progresses to recumbency and death. Neuropathological studies show a central axonopathy which may be of the dying back type.

References


Figure 1A - Silver stain of the cervical spinal cord in transverse section. Bielschowski X
Figure 1B - Enlargement from figure 1A to show the loss of axons in the superficial part of the lateral funiculus. Bielschowski X 200
Table 1: Clinical data for 5 Holstein x Gir calves with myelopathy

<table>
<thead>
<tr>
<th>Animal number and sex</th>
<th>Onset of signs of spinal cord disease (weeks)</th>
<th>Onset of recumbency (weeks)</th>
<th>Post mortem examination (weeks)</th>
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<tbody>
<tr>
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