Congenital spinal stenosis and dam mortality associated with feeding moldy cereal straw

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Abstract

We describe herein an epidemiological investigation of the cause of a syndrome of congenital spinal stenosis (CSS) in four beef herds in western Canada. Three of the herds were affected with the syndrome in 1987, the fourth was affected in 1990. The prevalence of CSS in affected groups of calves varied from 29% (16/55) to 100% (14/14). All affected calves had congenital posterior paralysis or paresis; some calves also had one or more of the following: shortened limbs, forelimb varus deformities, superior brachygnathia, and a dome-shaped cranium. Affected calves showed focal premature closure of axial and appendicular metaphyseal growth plates. At three of the four farms most of the pregnant cows were affected with alopecia with or without pruritus in January, and 25% of the cows in one herd died during the winter. The investigation indicated that CSS was associated with feeding moldy cereal straw to pregnant beef cows during the winter. At all four farms, the cereal straw bales were thoroughly soaked by rain prior to stacking, and obvious mold was present when they were broken open for feeding. Species of both *Penicillium* and *Fusarium* were abundant within the bales. The most likely cause of the disease was a fungal mycotoxicosis, although the mycotoxin responsible was not isolated.

Résumé

Sténose congénitale du canal vertébral et mortalité chez des vaches associées à l'ingestion de foin moisi

Étude épidémiologique sur la cause du syndrome de sténose congénitale du canal vertébral dans quatre troupeaux de boeufs de boucherie de l'ouest canadien. Trois troupeaux ont présenté le syndrome en 1987 et le quatrième en 1990. La prévalence de la pathologie pour ces troupeaux variait de 29% (16/55) à 100% (14/14). Les veaux présentaient une parésie ou une paralysie du train postérieur. Certains avaient aussi l'une ou plusieurs de ces anomalies : membres plus courts, déformité des membres antérieurs en varus, brachygnatisme, crâne en forme de dôme. Les animaux démontraient des zones de fermeture prématurée des plaques de croissance de la métaphyse des os du squelette axial et appendiculaire. Dans trois troupeaux, la majorité des vaches gestantes ont présenté, au mois de janvier, de l'alopécie avec ou sans prurit. Vingt-cinq pourcent des vaches d'un troupeau sont mortes durant les mois d'hiver.

Dans les quatres fermes, les meules de foin avaient été détrempées par l'eau de pluie avant d'être entreposées, de sorte qu'elles présentaient des moisissures visibles lorsqu'elles ont été offertes en nourriture aux animaux. Des organismes du genre *Penicillium* et *Fusarium* ont été isolés en abondance. Une mycotoxicose fut édentifiée comme la cause la plus probable du syndrome. Toutefois, la mycotoxine n'a pu être détectée. *(Traduit par Dr Thérèse Lanthier)*

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Introduction

A syndrome of congenital spinal stenosis (CSS) affecting 7-96% of newborn beef calves in four herds in western Canada has recently been described by Doige *et al* (1). Affected calves were either unable to stand, or had posterior ataxia or posterior weakness at birth. Some calves had shortened limbs. None of the affected animals recovered, and two of the farms lost the majority of their calf crop. Focal premature closure of metaphyseal growth plates in the axial and appendicular skeleton had resulted in dorsoventral narrowing of the vertebral canal and shortening of the long bones (1). A very similar syndrome affecting most of the calves in a Saskatchewan herd was reported by Orr and McKenzie in 1981 (2). The cause of CSS was not determined in either of these reports.

We have recently identified a similar syndrome in three beef herds in eastern Alberta and one herd in western Saskatchewan. The entire calf crop was lost in two of these herds, as was most of the calf crop in the third herd. In addition, 25% of the cows in one of the herds died during the winter. The economic and psychological effects of losses of this magnitude were devastating to the cow-calf producers involved.

The purpose of this paper is to describe the results of our epidemiological investigation of these four cow-

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Table 1. Prevalence of congenital spinal stenosis (CSS) by farm and management group

Farm	Spring	Predominant overwinter feed	Total number calves	Calves with CSS	CSS prevalence (%)
Paynton	1990	straw	14	14	100
		hay	4	0	0
Chauvin S	1987	straw	50	48	96
Chauvin N	1987	straw	55	16	29
		hay	22	0	0
Vermilion	1987	straw	26	18	68
		hay	12	0	0

calf herds. Our objective was to determine the cause of CSS, or, at the very least, to discern methods for predicting and preventing the occurrence of the disease in the future. We show herein that the syndrome resulted from feeding moldy cereal straw to pregnant beef cows during the winter.

Materials and methods

Local veterinary practitioners alerted us to the appearance of a severe congenital skeletal anomaly problem at all four farms. Three of the investigations were carried out in the spring of 1987: two in the area of Chauvin, Alberta (Chauvin N and Chauvin S), and one close to Vermilion, Alberta. The fourth investigation took place during the spring of 1990 at a beef farm close to Paynton, Saskatchewan.

Our epidemiological investigation involved several visits to each farm and our techniques have been described (3,4). We examined the animals, feed, property, and calving records at each farm. We described the clinical appearance and prevalence of the congenital anomaly, examined the cows and their physical environment, and reviewed management practices. We also attempted to identify groups of cattle differing with respect to management, age, location, breeding, and CSS prevalence. This last procedure involved the attempt to identify what can be termed "natural experiments".

In outbreak investigations like the one described herein, natural experiments can sometimes be identified and analyzed (5,6). With a natural experiment, the investigator has not actually performed a true experiment where all risk factors (or variables) but one are controlled; instead, two (or more) subgroups within a population can be identified which were treated identically, except for one or two risk factors, by circumstance. A farmer might, for example, regularly separate a herd into two groups (of first-calf heifers and older cows) and feed them differently during the winter. Although the farmer did not intentionally plan this as an experiment, an investigator can take advantage of the situation and look for differences in disease prevalence between the two groups, carefully determining whether other important differences also existed between the groups. John Snow's investigation of a cholera epidemic in London in 1854 was a classic example of the usefulness of identifying a natural experiment (7).

Some affected calves from each of the Alberta farms were brought to the Western College of Veterinary Medicine (WCVM) for further clinical evaluation; necropsies were performed on all of these calves after euthanasia. Necropsies were also performed at the WCVM on the affected calves, and one unaffected calf (a dystocia-related death), from the Paynton herd.

Samples of winter feed were collected and submitted to the Saskatchewan Feed Testing Laboratory at the University of Saskatchewan for analysis. Blood samples were obtained from groups of affected and nonaffected calves and their dams for serum mineral analyses.

All cow deaths in the Chauvin S herd occurred during the winter prior to our investigation. The farmer did not notify the local practitioner of these mortalities until spring; as a result, no necropsies were performed on these animals. Skin scrapings were taken from representative cows with alopecia.

Results

Three natural experiments were identified, where the farmers separated their pregnant herd into two groups. In each case, the winter diet of one group was predominantly straw (barley, wheat, or oat straw), while the diet of the other group was predominantly grass hay. The prevalence of CSS in calves coming from each of these feeding groups is presented (Table 1). All of the CSS calves came from the groups that were fed predominantly straw during the winter.

Straw bales at all four farms, when broken open, were very moldy. The insides of many of the bales were wet. Species of both *Penicillium* and *Fusarium* were isolated from samples taken at each farm; *Acremonium* spp. were also isolated from samples of wheat and barley straw taken from the Paynton farm. At the three Alberta farms, the farmers' descriptions of the previous harvest were similar. In each case, it began raining the day after the straw was baled. The rain continued for several days. Then the bales were left in the fields for two to three weeks in an attempt to dry them out before they were collected and stacked. Many of the bales had not dried completely by winter.

In Paynton, 88 mm of rain fell over nine days on recently baled barley straw. The bales were left on the pasture for two weeks prior to stacking. The owner had also just finished combining the wheat two days prior to the rains, so the wheat lay in swath for three weeks before being baled and brought home.

The two farms feeding the largest volume of moldy straw on a per head basis, Chauvin S and Paynton, also had the highest prevalence of CSS. At Vermilion, the least affected calf came from a dam that was shifted on a regular basis from the hay-fed group to

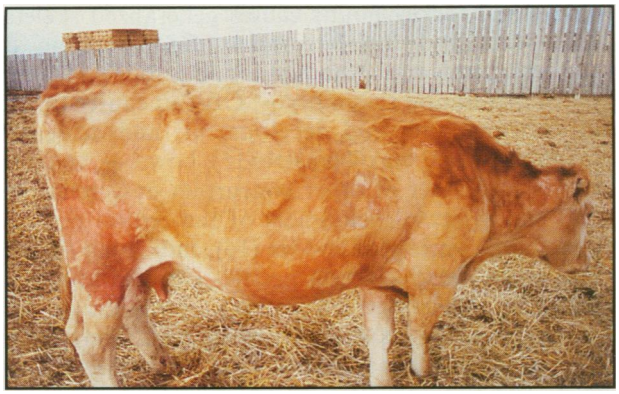

Figure 1. Marked alopecia in a cow, coinciding with congenital spinal stenosis in beef calves. Both conditions were associated with feeding moldy straw during the winter. The exposed epidermis is pink (most notable in this particular cow along the lower edge of the ribs and on the hindlimb above the hock), smooth, and thin with no evidence of bacterial or parasitic infection.

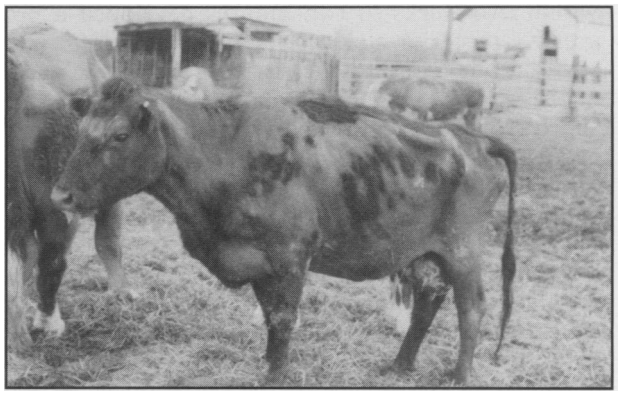

Figure 2. A cow in very poor body condition on the farm experiencing the highest prevalence of congenital spinal stenosis. Alopecia is marked and there are only a few patches of normal hair remaining. Mortality in cows at this farm during the winter was 25%.

the straw-fed group and back again. The affected group at Chauvin N, where CSS prevalence was lowest, received proportionately less straw than cows at the other three farms.

All farmers also fed various quantities of grain, hay, and mineral mixes. Feed analyses showed that the total winter diet at all the farms exceeded NRC requirements for crude protein, TDN, Ca, and P. Selenium levels in the feed and in serum samples were within normal ranges. Water quality varied from excellent to poor, but did not appear to correlate with CSS prevalence.

By late January, the owners of the three most severely affected farms reported noticing severe alopecia with or without pruritus in the pregnant cows being fed straw. The hay-fed group at Vermilion, which commingled with the straw-fed group all day except for feeding time, showed no hair loss. Likewise, the four hay-fed cows at Paynton, and a group of predominantly hay and grain-fed yearlings in a pen adjacent to the Chauvin S cows, showed no hair loss. Close visual inspection did not reveal the presence of any lice. Despite this, the cows at Vermilion and Chauvin S were treated with fenthion (Lysoff, Cutter Animal Health, Bayvet Division Chemagro Ltd., Etobicoke, Ontario), and cows at Paynton were treated with ivermectin (Ivomec Pour-On, MSD Agvet, Pointe Claire-Dorval, Quebec).

Many of these cows still lacked hair on 50–70% of their body at the end of calving season; the exposed skin in non-pigmented areas was pink (Figure 1). The epidermis was very smooth and thin with no evidence of bacterial or parasitic infection. Alopecia occurred in many, but not all, of the cows having a CSS calf at Vermilion, Chauvin S, and Paynton. At Chauvin N, where cows ate less straw than the other farms and the prevalence of CSS was lowest, alopecia was not documented in any of the cows. The condition of the cows varied, with cows in poor condition at Chauvin S (Figure 2), moderate condition at Vermilion and Paynton (Figure 1), and excellent condition at Chauvin N.

The owner of the Chauvin S farm reported 25% mortality (20/80) in the pregnant cow herd during the winter. Three cows died in late November and December; the rest died at a rate of one to two cows per week from February until calving. The owner said that these cows were generally in good body condition. They would go off feed, become recumbent, and have difficulty rising. Despite this, when approached they would struggle up and flee. Death occurred two to seven days after the onset of clinical signs.

There was no predilection of CSS for a particular breed of calf; Charolais, Hereford, Shorthorn, and crossbreds were affected equally. There was also no predilection for a particular bull; at two farms, we documented affected calves being sired by three different bulls in three different summer pastures.

Clinical description of affected calves

Congenital posterior paralysis or posterior paresis were common to almost all of the affected calves at all four farms. At Chauvin N, affected calves showed no other clinical signs; the calves were of normal stature, alert, and would look for the dam. These calves were unable to get up on their hindlimbs, and would frequently draw themselves up to rest in a "dog-sitting" position (Figure 3). A few calves appeared normal at birth, but

Figure 3. "Dog-sitting" position commonly seen in young calves affected with congenital spinal stenosis. The hindlimb paralysis can be evident immediately postpartum, or it may take several weeks to develop.

within two weeks to two months developed a progressive posterior ataxia.

Additional congenital signs were evident in calves at the other three farms. Many of these calves had shortened limbs (disproportionate dwarfism) with laxity of all major joints. Some had severe varus deformities of the front limbs. Occasionally there was superior brachygnathia, a domed-shaped cranium, and proportionate dwarfism. Two calves at Paynton had tremors when they attempted to suck.

All of the affected calves showed focal premature closure of axial and appendicular metaphyseal growth plates. Focal closure of vertebral epiphyses resulted in dorsoventral narrowing of the vertebral canal and spinal stenosis; vertebral epiphyseal projections into the vertebral canal resulted in multifocal compression of the spinal cord. Focal areas of premature closure of many long bone epiphyses were accompanied by reduced length of the long bones and malformations of the articular surfaces. Closure or partial closure of cranial base epiphyses in some calves was associated with a domed skull and cerebellar hypoplasia.

Discussion

Our analysis of the three natural experiments documented herein indicates that CSS was associated with feeding moldy cereal straw to pregnant beef cows. An apparent dose-effect relationship strengthens this hypothesis: pregnant cows exposed to the greatest amount of moldy cereal straw during the winter had the highest prevalence of calves with CSS.

The clinical and pathological findings documented in affected calves in the four herds in this study were the same as those seen by Doige *et al* (1) and Orr and McKenzie (2). Clinical signs in the cows were also similar. Rubbing and alopecia were documented to have affected many of the cows in late January in the herd described by Orr and McKenzie (2). In January, four of 36 (11%) cows died in one herd described by Doige *et al* (1), and 90% of the surviving cows in that same herd were losing their hair by February (Townsend H.G.G., 1990, personal communication). Although no mention was made of weather conditions at harvest, cereal straw was the predominant winter feed for pregnant cows in all of these herds (2; Townsend H.G.G., 1990, personal communication).

The most likely cause of the disease is a fungal mycotoxicosis, although the mycotoxin responsible was not isolated. Mycotoxins are secondary metabolites of fungi (molds) which can be toxic to animals (8). A number of potent mycotoxins are known to be produced by *Penicillium* (9) and *Fusarium* (10,11) fungi; species of both of these molds were abundant in straw samples taken from all four farms affected by CSS. Although there are no reports of *Penicillium* or *Fusarium* mycotoxins causing premature focal closure of epiphyses in cattle, a number of these mycotoxins cause developmental skeletal anomalies in other species.

Tibial dyschondroplasia in turkeys is caused by the production of the metabolite fusarochromanone by *Fusarium equiseti* in grain (12). There is widening of affected growth plates due to overgrowth of chondrocytes (13). Ochratoxin A (OA) fed to young broiler chicks and turkey poults causes generalized skeletal osteopenia and interferes with the formation of endochondral and intramembranous bone (14). In postimplantation rat embryos, OA can cause stunted growth and limb bud development, and decreased mandibular and maxillary size (15); however, OA also causes a number of other concurrent defects in rats (15) and affects development of the central nervous system in a variety of species (16), indicating that its effects are relatively nonspecific. Citreoviridin produced by *Penicillium citreoviride* retards the general development of fetal skeletons in rats, likely as an indirect result of the maternal toxicity of the mycotoxin (17). The skeletal lesions documented to arise from exposure to fusarochromanone, OA, and citreoviridin are very different from those seen in bovine CSS. Unless one of these mycotoxins has a very different effect on the developing bovine growth plate, none of them are likely to be involved in CSS.

Unfortunately, it is unlikely that the toxic principle responsible for CSS will be determined by screening for the presence of known mycotoxins. *Penicillium, Fusarium*, and possibly *Acremonium* produce many other ("unclassified") fungal metabolites (8), any one of which could be the specific toxin responsible for CSS. It is also possible that the condition is related to the presence of not one but several mycotoxins: Huff *et al* (8) state that, where one mycotoxin is found, more mycotoxins are very likely to be present, and a number of toxins could act together to produce a complex of clinical signs.

Until a specific mycotoxin has been clearly associated with CSS, other potential causes of the condition must be considered. Although we found no evidence of a dietary deficiency or excess, other potential causes of CSS have included hypervitaminosis A (1,2), maternal manganese deficiency, warfarin toxicity, frostbide, and plant enzymes (2). Other toxic factors may also have to be investigated. The recent suggestion that tibial dyschondroplasia might be partially prevented by concurrent dietary copper supplementation (18) introduces the complicating possibility that a combination of different factors could contribute to CSS.

There are some similarities between CSS and another congenital skeletal anomaly syndrome reported in beef calves in Canada known as congenital joint laxity and dwarfism (CJLD). Both diseases result from exposure of the pregnant cow to a particular kind of winter feed; CJLD has been associated with feeding grass or clover silage exclusively, without supplementation of hay or grain, to pregnant cows during the winter (19). Both conditions specifically affect the bovine fetal growth plate, although the histological change seen with CJLD is impeded endochondral ossification (19), not premature growth plate closure. As with CSS, the definitive toxic principle responsible for CJLD has not been established. Toxicosis from fungal toxin production is one hypothesized cause of CJLD, although the possibility of a deficiency has not been ruled out (19). Cow-calf producers feeding pregnant cows over winter in Canada should be made aware of the feed-related epidemiology associated with both conditions.

It is crucial that the toxic principle in CSS be identified. Cereal straw forms the predominant part of the winter diet for pregnant beef cows on many prairie farms, and many of the straw bales are exposed to rain prior to stacking. The amount of rain and fungal growth required to reliably produce CSS is unknown. We need to determine the presence and nature of the toxic factor causing CSS, the threshold beyond which the toxin becomes a dangerous problem, and a system for testing straw bales that have been harvested in "less than ideal" conditions. Potential methods of detoxifying contaminated straw bales, beginning with the encouraging results found with other mycotoxins (20–22), should also be investigated.

For now, if cereal straw bales have been thoroughly soaked by rain prior to stacking, and there is obvious mold present when the bales are broken open, we urge cow-calf producers to refrain from feeding any of the bales to their pregnant cows. In less obvious cases, where some rain falls on the straw bales at harvest and mold is not too obvious at feeding, the suspicious bales should still be reserved for nonpregnant stock, or diluted to at most 10% with undamaged feeds (23), and fed to a small test group of animals for several months. Feeding the suspicious feed to all pregnant stock and changing feeds if alopecia or pruritus are noticed is definitely not a management option: by the time the pregnant cows show alopecia, (and remember, one of the herds affected by CSS did not show this sign), much of the fetal growth plate closure is likely complete and irreversible.

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The final draft of this paper was submitted after the untimely death of Dr. Cecil Doige. Dr. Doige gave momentum to the scientific curiosity behind investigations into outbreaks of neonatal skeletal abnormalities such as the one described herein. Dr. Doige, with his interest in osteopathology, provided the continuity necessary to precisely identify similar lesions in the affected herds over a number of years. We are grateful to have been able to participate with him in the practice of herd medicine. CVJ

References

1. Doige CE, Townsend HGG, Janzen ED, McGowan M. Congenital spinal stenosis in beef calves in western Canada. Vet Pathol 1990; 27: 16–25.
2. Orr JP, McKenzie GC. Unusual skeletal deformities in calves in a Saskatchewan beef herd. Can Vet J 1981; 22: 121–125.
3. Ribble CS, Janzen ED. Investigation of a congenital defect problem in cow-calf herds. Proc Am Assoc Bov Pract 1988; 21: 96–100.
4. Rousseaux CG, Ribble CS. Developmental anomalies in farm animals II. Defining etiology. Can Vet J 1988; 29: 30–40.
5. Schwabe CW, Riemann HP, Franti CE. Epidemiology in Veterinary Practice. Philadelphia: Lea & Febiger, 1977.
6. Lilienfeld AM, Lilienfeld DE. Foundations of Epidemiology. New York: Oxford University Press, 1980.
7. Snow J. On the mode of communication of cholera. In: Snow On Cholera. New York: The Commonwealth Fund, 1936.
8. Huff WE, Kubena LF, Harvey RB, Doerr JA. Mycotoxin interactions in poultry and swine. J Anim Sci 1988; 66: 2351–2355.
9. El Banna AA, Pitt JI, Leistner L. Production of mycotoxins by *Penicillium* species. Syst Appl Microbiol 1987; 10: 42–46.
10. Mirocha CJ. Mycotoxicoses associated with *Fusarium*. In: Moss MO, Smith JE, eds. The Applied Mycology of *Fusarium*. Symposium of the British Mycological Society. Cambridge, UK: Cambridge University Press, 1984: 141–155.
11. Trenholm HL, Prelusky DB, Young JC, Miller JD. A practical guide to the prevention of *Fusarium* mycotoxins in grain and animal feedstuffs. Arch Environ Contam Toxicol 1989; 18: 443–451.
12. Krogh P, Christensen DH, Hald, B, *et al.* Natural occurrence of the mycotoxin fusarochromanone, a metabolite of *Fusarium equiseti*, in cereal feed associated with tibial dyschondroplasia. Appl Environ Microbiol 1989; 55: 3184–3188.
13. Bashey RI, Leach RM, Gay CV, Jimenez SA. Type X collagen in avian tibial dyschondroplasia. Lab Invest 1989; 60: 106–112.
14. Duff SR I, Burns RB, Dwivedi P. Skeletal changes in broiler chicks and turkey poults fed diets containing ochratoxin A. Res Vet Sci 1987; 43: 301–307.
15. Mayura K, Edwards JF, Maull EA, Phillips TD. The effects of ochratoxin A on postimplantation rat embryos in culture. Arch Environ Contam Toxicol 1989; 18: 411–415.
16. Kuiper-Goodman T, Scott PM. Risk assessment of the mycotoxin ochratoxin A. Biomed Environ Sci 1989; 2: 179–248.
17. Morrissey RE, Vesonder RF. Teratogenic potential of the mycotoxin, citreoviridin, in rats. Food Chem Toxicol 1986; 12: 1315–1320.
18. Wu W, Cook ME, Smalley EB. Partial prevention of fusarochromanone-induced dyschondroplasia and immunosuppression by concurrent dietary copper supplementation [abstract]. Poultry Sci 1988; 67 (Suppl 1): 174.
19. Ribble CS, Janzen ED, Proulx JG. Congenital joint laxity and dwarfism: A feed-associated congenital anomaly of beef calves in Canada. Can Vet J 1989; 30: 331–338.
20. Colvin BM, Sangster LT, Haydon KD, Beaver RW, Wilson DM. Effect of high affinity aluminosilicate sorbent on prevention of aflatoxicosis in growing pigs. Vet Hum Toxicol 1989; 31: 46–48.
21. Müller HM. Maβnahmen zur minderung von mykotoxinbildung und -anreicherung in futtermitteln. Dtsch Tierärztl Wochenschur 1989; 96: 363–368.
22. Speer M, Süssmuth R. Bacterial tests as indicators for the detoxification of the mycotoxin penicillic acid by ammonia treatment. Food Chem Toxicol 1987; 25: 31–34.
23. Blood DC, Radostits OM. Veterinary Medicine, 7th ed. London: Baillière Tindall, 1989: 1308.