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Retrospective study: Investigating the downer cow syndrome

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Abstract

Downer cows, defined as a bright and alert cow recumbent for more than 1 day, are often challenging cases for veterinarians and farmers. This article reviews findings for 37 downer dairy cows that originally become recumbent from hypocalcemia (milk fever) on commercial dairy farms in Australia. Clinical examination determined that they had apparently recovered from primary hypocalcemia, and remained recumbent from secondary damage. There was a wide range of secondary damage, which mainly affected the musculoskeletal system, but other parts of the body were also affected. It was concluded that downer cow syndrome is due to clinically important secondary damage, defined as "secondary damage that can cause recumbency in its own right, or delay or prevent recovery from the primary cause of the recumbency". Clinically important secondary damage in downer cows can present in a wide range of types, and is not necessarily confined to the musculoskeletal system. This study demonstrated a wider range of secondary damage than considered in the literature. When managing recumbent dairy cows, in addition to the primary etiology of the recumbency, veterinarians must consider secondary damage.

Key words: down cow, downer cow syndrome, secondary damage, milk fever

Résumé

Les vaches à terre, définies comme étant des vaches vives et alertes immobilisées au sol pour plus d'une journée, sont souvent des cas problématiques pour les vétérinaires et les producteurs. Cet article examine les observations faites à partir de 37 vaches laitières à terre initialement immobilisées au sol en raison de l'hypocalcémie (fièvre vitulaire) dans des fermes laitières commerciales de l'Australie. L'examen clinique a montré que les vaches s'étaient apparemment rétablis de l'hypocalcémie primaire mais restaient immobilisées au sol suite à des dommages secondaires. Il y avait diverses sources de dommages secondaires qui affectaient surtout le système musculosquelettique bien que d'autres parties du corps étaient aussi touchées. La conclusion était que le syndrome de la vache à terre est causé par des dommages secondaires importants définis comme 'des dommages qui peuvent causés l'immobilisation au sol par eux-mêmes ou qui reporte ou empêche le rétablissement suite à la cause primaire de l'immobilisation au sol'. Les dommages secondaires cliniquement importants chez les vaches à terre se présentent de plusieurs façons et ne sont pas nécessairement limités au système musculosquelettique. Cette étude a mis en évidence une plus grande variété de dommages secondaires que ceux considérés dans la littérature. Dans la régie des vaches laitières à terre, les vétérinaires doivent considérés les dommages secondaires en plus de l'étiologie primaire de l'immobilisation au sol.

Introduction

The term "downer cow syndrome" was coined to explain why some recumbent cows became persistently recumbent. Many veterinarians find downer cows challenging,[6] and over the years it has been an area of confusion, which is reflected by the many different definitions of downer and downer cow syndrome. Downer cows were originally thought to be a complication of hypocalcemia, with 1 definition being a cow that "did not rise within 24 hours of treatment with calcium"[7], and another as recumbency for "more than 24 hours related to the calving period after the animal had received 2 calcium injections and for which there was no obvious reason for being down".[1] However, a Danish study found that only 52% of 43 downer cows studied were associated with hypocalcemia.[8] Weaver[13] defined downer cow syndrome as "a cow down in sternal recumbency for unknown reasons". However, this definition depends on the acuity and thoroughness of the examiner. For the present study, a downer cow is defined as a cow that is in sternal recumbency for more than 24 hours, and is bright and alert.

Over the years the cause of downer cow syndrome has been poorly understood, and has been associated with many conditions, such as metabolic complex parturient paresis; hypophosphatemia; hypomagnesemia; hypokalemia; hyper- and hypo-adrenocortical activities; cerebral edema;

albuminuria; renal disease; hepatic changes, muscle degeneration; and physical injuries.[8]

Cox et al[5] experimentally induced downer cow syndrome in 8 of 16 healthy cows by maintaining sternal recumbency on the right-hind limb for 6, 9 or 12 hours using halothane anesthesia. The downer cows showed "ischemic necrosis of the caudal thigh muscles, inflammation of the sciatic nerve caudal to the proximal end of the femur", and "evidence of peroneal damage". There was a striking difference between the compressed right-hind limb muscles and the non-compressed left-hind limb muscles, which showed the importance of pressure damage in downer cow pathogenesis.[5] The hamstring group of muscles (biceps femoris, semitendinosus, and semimembranosus) have thick fascial boundaries, which make them particularly prone to damage in a recumbent cow. The compressed muscles swell as lymphatic fluid and venous blood become trapped within the fascial compartments. These hydrostatic forces progressively reduce arterial blood supply causing an ischemic myonecrosis, which in turn leads to further swelling and a destructive cycle ensues.[12] The sciatic nerve is susceptible to compression against the caudal femur just distal to the hip joint in recumbent cattle, which can result in innervation deficits to the hamstring muscle group and the muscles distal to the stifle.[4]

In an earlier study, the hamstring muscle damage was highly variable between the cows and did not affect all of the muscle,[5] whereas the localized damage to the nerve trunks passing through the affected muscles would be expected to have more significant effects as the entire muscle innervated by those nerve trunks would be affected.

Cox[4] considered that pressure damage to the muscles and nerves causes secondary recumbency following primary recumbency for any reason, and in some cases a terminal tertiary stage involving rupture of muscle and ligaments. That study helped de-mystify the downer cow syndrome by clearly showing the role of secondary damage in recumbent cows. This concept was further expanded by Malmo et al,[9] who described downer cow syndrome as "associated with the pathology that develops secondarily to prolonged recumbency". The secondary effects resulting from recumbency they described was local tissue injury from compression of the limbs becoming the common "unifying factor" for all downer cow cases, with musculoskeletal damage while struggling to rise or from crawling forward while unable to rise. They listed hind-limb muscle and nerve damage, radial nerve damage, coxofemoral dislocation, fracture of the femoral neck, and further skeletal injury, such as hemorrhage in or rupture of the adductor or gastrocnemius muscles as likely secondary damage in downer cows.[9]

The causes of persistent recumbency in dairy cows are an interplay between the primary cause and the secondary damage resulting from the recumbency. Some cows will only be affected by the primary cause, some will remain recumbent solely from the secondary damage, and some cows will have both primary and secondary factors.[10] To further investigate secondary damage that could cause downer cow syndrome, several cases of downer cow syndrome under commercial farming conditions in Australia were reviewed. These cases had been recumbent for more than 24 hours, were clinically bright and alert, and had initially become recumbent from hypocalcemia. This cohort of cows was selected because they had apparently clinically recovered from the metabolic cause of recumbency, eliminating the role of the primary cause from the clinical picture. The hypothesis was that there was a wide range of secondary damage that can occur during or following primary recumbency.

Case Selection

Field studies of recumbent dairy cows were conducted in South Gippsland, Victoria, Australia during 3-month periods in the winter seasonal calving months of 2011 and 2012. Cows were included in the study if they were bright, alert and responsive, had been recumbent for at least 1 day, and there was adequate history for the cows to be assessed properly. From this larger study[10] a cohort of cows that had initially become recumbent following the diagnosis and treatment of hypocalcemia by the farmer was formed. The cows were referred to the primary researcher after they had been examined by local veterinarians to exclude primary recumbency from other causes. All of the cows had been administered metabolic solutions (calcium, magnesium, phosphorus, and glucose) intravenously and/or subcutaneously by the farmer, and local veterinarian where appropriate, prior to contacting the primary researcher.

Each cow's detailed history was collected from the farmers and referring veterinarians to ensure that clinical signs during initial recumbency and response to treatment were consistent with hypocalcemia as the primary cause of the recumbency. Most of the dairy cows included in the study had recently calved, were initially found by the farmer in acute recumbency (sternal or lateral), and were depressed with a cold, dry nose, but without signs of dehydration. Intravenous administration of calcium solution, with or without magnesium, phosphorus and glucose, immediately restored the cow to normal mentation with a moist nose, but the cow failed to rise. Cows were subsequently given further metabolic treatments by the farmer, but were still recumbent the following day. Cows remained bright and alert at this point, and failed to be rise despite more metabolic treatments administered by the farmer. Veterinary assistance was sought, and the local veterinarians examined the cows clinically. Cows initially determined to become recumbent from hypocalcemia and remained recumbent after further treatment with calcium, magnesium, phosphorus, and glucose solutions intravenously were referred to the primary researcher (PJP).

The primary researcher conducted a thorough physical examination on each cow at the first visit to exclude other possible primary causes of recumbency and ongoing signs of clinical hypocalcemia. In the absence of these findings a thor-

ough musculoskeletal clinical examination was conducted. Samples for blood electrolytes, such as calcium, magnesium, and phosphorus, were not collected. The musculoskeletal examination included:

- assessment of the spinal column for fractures;
- assessment of the limbs for damage to joints, tendons, ligaments and muscles;
- nerve function assessment by:
  - flexor-withdrawal reflex, patellar reflex, and muscle tone assessment in the recumbent position;
  - observation of postural responses when the cows tried to stand and/or when they were lifted, using chest straps for cows that failed to bear weight on the forelimbs when lifted with hip clamps; and
  - flexor-withdrawal reflex and muscle tone assessment were repeated in the elevated position;
- blood samples of 26 of the cows were taken to determine serum analysis of creatinine phosphokinase (CK) and aspartate amino transferase (AST) levels after the cow had been down for more than 1 day.

Musculoskeletal abnormalities and physical conditions were diagnosed using standard methods, but the diagnoses of some specific musculoskeletal conditions are listed below:

- sacroiliac damage was diagnosed by an increased laxity of the sacroiliac joint when subjected to motion palpation;
- sciatic nerve dysfunction was indicated by a number of signs, depending on which branches of the nerve were involved, such as increased patellar reflex, decreased sensations of the caudal and/or anterior pastern, proprioceptive defects, and/or a tendency for an anterior/medial displacement of the leg when lifted;
- femoral nerve dysfunction was determined by decreased or absent patellar reflex and a tendency for a caudal displacement of the hind limbs when trying to stand;
- brachial plexus paralysis was determined by flaccid paralysis and negative flexor-withdrawal reflex of the forelimb, which was assessed in both the prone and raised positions;
- radial nerve paralysis was indicated by flaccid lower forelimb function and a proprioceptive deficit when lifted but normal upper-limb function;
- tibial paresis was diagnosed by hyperextension of the stifle, mild over-flexion of the hock, and a slightly flexed fetlock, but with the claw in a normal position when the cow was lifted; and
- compartment syndrome was diagnosed when CK levels were above the time-adjusted threshold levels of 50, 44, or 38 times the upper normal CK level (250 U/L),[a] which represented values of 12,500, 11,000, and 9,500 U/L for cows recumbent for 1, 2, or 3 days, respectively, or when AST levels were ≥ 7.4 times the upper normal reference range (1,110 U/L). Muscle enzyme values above these thresholds indicated a less than 5% chance of recovery.[2]

Exposure was defined as cows that had subnormal body temperature, and were depressed after being exposed to cold weather events. Heart failure was diagnosed clinically by a fast, weak, erratic heartbeat. A clinical judgement on each cow was made as to whether the failure to rise after a milk fever episode was due to hypocalcemia, the secondary complications following the recumbency, or a combination of both.

Clinically significant secondary damage was defined as secondary damage that could cause recumbency in its own right, or delay and prevent recovery from the original cause of the recumbency.

Clinical Findings

The cohort was composed of 37 dairy cows from 28 commercial dairy herds. The study cows were a subgroup from a larger study which included 218 downer dairy cows due to any primary cause from 96 herds.[10]

Following referral from local veterinarians, 14 of 37 (38%) cows were attended by the primary researcher on the first day of recumbency (day 0), 11 (30%) cows on day 1 (second day), 9 (24%) cows on day 2, and 3 (8%) cows on day 3. No cows were clinically judged to remain recumbent due to hypocalcemia when first attended by the primary researcher as they were all bright and alert; the heartbeat was normal for rate, rhythm, and volume when auscultated with a stethoscope; noses were moist and warm; there were efforts to stand. All 37 cows were deemed recumbent due to clinically important secondary damage to the musculoskeletal system.

Table 1. Types of secondary damage recorded in 37 downer cows.

Type of secondary damage	N	%
Elevated CK*	26	100
Femoral nerve	25	68
Compartment syndrome*,†	9	35
Brachial plexus	5	14
Radial nerve	5	14
Tibial paresis	3	8.1
Exposure	3	8.1
Hip dislocation	2	5.4
Sciatic nerve	2	5.4
Sacroiliac damage	2	5.4
Heart failure	1	2.7
More than 1 type of damage	17	46
Total cows	37	100

*Only 26 cows were blood tested

†Compartment syndrome diagnosed when a cow had a CK level greater than the time-adjusted critical level determined by Clark et al,[2] which predicted <5% chance of recovery.

The occurrence and types of secondary damage recorded by the primary researcher on the initial visit are shown in Table 1, noting that 17 of 37 (46%) cows had more than 1 type of damage concurrently.

Muscle enzymes were analyzed in 26 of the 37 (70%) cows. Creatinine phosphokinase levels ranged from 1,910 to 81,900 U/L (7.6 to 328 times the upper normal limit), 1,811 to 73,860 U/L (7.2 to 295 times the upper normal limit), and 900 to 19,830 U/L (3.6 to 79 times the upper normal limit) for cows recumbent for 1, 2, and 3 days, respectively. Nine of 26 (35%) cows were diagnosed as having compartment syndrome on the basis of CK levels above the time-adjusted threshold that represented less than a 5% chance of recovery.[2] AST levels ranged from 13 to 775 U/L, and no cows had AST levels > 7.4 times the upper normal limit of 150 U/L (1,110 U/L).

Discussion

The cause(s) of recumbency with the downer cow syndrome can be difficult to determine because of the interplay between the primary cause of recumbency and the secondary effects from the recumbency. Some cows will be persistently recumbent solely because of the primary cause, some from a combination of primary cause and secondary complications, and other cows from only the secondary complications. The cohort of downer cows in the current study were selected because the primary cause of recumbency, hypocalcemia, appeared resolved prior to the lead researcher's first examination of the animals. Thus, the clinical presentation was no longer complicated by the primary cause of the recumbency, hypocalcemia.

This study further expands the concept that downer cow syndrome is caused by secondary pressure damage by detailing specific conditions that seemed to be, on the basis of clinical findings, the cause of the persistent recumbency following the initial hypocalcemia. There were 11 different types of secondary damage recorded in the present study, as shown in Table 1, and 17 cows had more than 1 type concurrently.

Hypocalcemia is a condition commonly encountered by dairy farmers who are usually able to recognize the clinical signs and administer calcium with or without phosphorus, magnesium, or glucose. In the past, many farmers normally only administered such solutions subcutaneously and it was common for veterinarians to be called to attend hypocalcemic cows. Presently, most farmers treat their hypocalcemia cases intravenously, which has greatly decreased the number of downer cows attended by veterinarians. When the veterinarian is called, they usually find that the cows are bright and alert and have normal heart function by auscultation. Some cows will regain ambulation after further intravenous calcium solutions are administered, but many remain unchanged. If the cow fails to rise at this point, the veterinarian must decide on the next step. Some veterinarians continue to assume the recumbency is due to metabolic deficiencies and leave the cow to be treated by the farmer with additional calcium solutions, some veterinarians assume that the problem is secondary muscle damage from the recumbency and prescribe anti-inflammatory drugs, and some veterinarians will conduct a thorough muscluoskeletal examination to determine various types of secondary damage that can occur. This study highlights the importance of the latter approach as it documents the wide range of secondary damage that can occur following initial recumbency due to hypocalcemia. The variety and occurrence of secondary damage found in these cases is useful information for clinical veterinarians.

Cox et al[5] found muscle damage in the caudal thigh to be a common feature of downer cow syndrome. Our study agrees with this finding, as elevated CK enzymes were found in all 26 cows that were tested. The significance of the elevated CK levels in persistently recumbent cows following treatment for hypocalcemia could be debated, as Cox also recorded elevated CK enzyme levels in cows that had recovered and were ambulatory after general anesthesia.[5] Compartment syndrome was deemed to be present in cows in the present study when CK levels were above the time-adjusted level that predicted a less than 5% chance of recovery, as determined by Clark et al.[2] However, it is possible that the initial muscle damage in cows with CK levels lower than these cut-off values may have contributed to the cow's failure to rise following hypocalcemia treatment. If the clinician fails to find clinical abnormalities when examining a down cow to explain the cow's recumbency, then measuring serum CK is justified as muscle damage may be the reason. AST levels can also be used to diagnose compartment syndrome and predict a less than 5% chance of recovery if above a threshold level.[2] There was poor correlation between CK and AST levels in cows in the present study as none of the 9 cows with CK levels above the critical threshold had AST levels above the threshold. This finding is contrary to findings by Clark et al[2] as they reported both CK and AST could be used to predict non-recovery in downer cows.

Secondary femoral nerve damage was found in 68% of the cows in the current study. These cows failed to rise due to lack of extension of the stifle joint, a tendency for the hind limbs to assume a caudal position when the cow tried to stand because of a lack of hip flexion, and decreased or absent patellar reflexes. It is postulated that the hyperextension of the lower back that occurs when a cow crawls and tries to stand could cause overstretching of the fourth and fifth lumbar femoral nerve roots. Many of the cows were observed by the farmers to crawl after becoming recumbent. Femoral nerve damage is usually associated with calves subjected to hip-lock during a difficult birth,[3] although Vermuth et al stated that it "can be caused by pressure or overstretching of the nerve when recumbent cattle attempt to rise".[12] Data from our study suggests that secondary femoral nerve damage is common as a result of recumbency, which is contrary to the literature.

Two cows had clinical symptoms of sciatic nerve damage, which would agree with the findings of Cox et al.[5]

Malmo et al stated that "although uncommon, radial paralysis can occur following prolonged periods in lateral recumbency" and be a cause of downer cow syndrome, but they did not list brachial plexus paralysis as part of the syndrome.[9] These 2 forelimb neuropathies were diagnosed in 10 of 37 (27%) cows in our study, which suggests that forelimb neuropathies could be a more common complication from recumbency than previously reported. Vermunt et al postulated that the brachial plexus could become compressed between the scapula and the ribs during prolonged lateral recumbency, and that the pressure caused by such recumbency is a major cause of radial nerve damage.[12] The farmers reported that all 10 of the cows (100%) afflicted by a forelimb neuropathy had been found in lateral recumbency at some stage prior to the lead researcher's visit. It is important that veterinarians consider forelimb neuropathies when examining recumbent cows as our data suggests that it is more common than documented in the literature.

Any unsteady cow struggling to rise is at risk of ventral hip dislocation; this was diagnosed at the researcher's first visit in 2 of 37 (5.4%) cows. This emphasizes the need for the veterinarian to include hip assessment as part of their clinical examination of the down cow.

Heart failure was a feature of 1 (2.7%) cow, which presumably occurred from calcium being administered intravenously too quickly or excessively. Many farmers do not monitor the cow's heart when administrating intravenous calcium, so this is always a potential risk.

Exposure was recorded for 3 (8.1%) cows, and in 2 of those cows exposure was considered to be a major contributor to ongoing recumbency. Herds in the study area graze in fields year around. Winter temperatures commonly range from a minimum of 32°F (0°C) degrees to maximum of 50 to 54°F (10 to 12°C), which, when combined with rain and wind-chill factors, can make conditions unpleasantly cold, especially for recumbent cattle. Two of the 3 cows were in wet and muddy conditions when first found by the farmer. They did not rise following the farmer's treatment with calcium, and were left in the paddock for more than 12 hours where they were subjected to cold conditions. Cows with hypocalcemia usually have a subnormal body temperature due to poor tissue perfusion from weak cardiac function, and this would be further compounded by cold conditions. It is probable that hypothermia could have decreased the absorption rate of the subcutaneous calcium, thus delaying recovery from the hypocalcemia, as well as directly affecting limb muscle function through decreased tissue perfusion. Both factors may have contributed to preventing these cows from rising after treatment for hypocalcemia. One of the cows subsequently developed secondary femoral nerve damage from crawling during that first day and the second cow developed brachial plexus paralysis after becoming cast laterally the day after initial hypocalcemia. It is possible that neither of these complications would have occurred if the cows' recovery from the initial hypocalcemia had not been complicated by cold environmental conditions. It is important that the attending veterinarian considers the environment for recumbent cows as part of their management plan to help avoid such complications.[11]

Persistent recumbency seemed to be caused entirely by secondary damage in the 37 downer cows included in this study. There was a wide range of types of secondary damage, including some not affecting the musculoskeletal system. It is proposed that downer cow syndrome be redefined to be "caused by clinically important secondary damage, being secondary damage that can cause recumbency in its own right, or delay or prevent recovery from the primary cause of the recumbency".

When veterinarians are examining down cows it is important to look for secondary damage. The primary cause of the recumbency needs to be determined, but it may not be the reason for the ongoing recumbency. This is particularly relevant for cows that initially become recumbent from hypocalcemia as most farmers have administered adequate amounts of calcium prior to seeking veterinary assistance. Whilst more metabolic treatment may be an appropriate part of the veterinarian's treatment, a broader focus is needed for many of these cases.

Limitations of the Review

The major limitation of this study is that the diagnosis of hypocalcemia as the initial cause of the recumbency was made by the farmers. Blood for mineral assays was not collected prior to the cows first receiving calcium solutions as the cows were from commercial dairy herds, not study herds. Nor were mineral assays performed when the lead researcher attended the cows as that was not included in the protocol for the larger study that this cohort of cows was drawn from. The lead researcher was satisfied that based on clinical history, including response to the farmer-administered calcium solutions, all cows in this cohort did initially become recumbent from hypocalcemia. While the fact that the primary diagnosis of hypocalcemia was only made clinically could be deemed to be a weakness of this study, this is a scenario commonly encountered by veterinarians. Field veterinarians often do not have cow-side access to mineral and electrolyte analysis, and usually rely on clinical examination and observations when determining their diagnosis and immediate treatment protocols. This study highlights the need for veterinarians to consider the full range of possible secondary damage when examining downer cows rather than only focusing on the primary cause of recumbency, or just the more commonly known secondary causes.

Conclusions

Selection of this cohort of down cows allowed the downer cow syndrome to be studied in isolation from the primary cause of the recumbency. The reason for ongoing

recumbency in the 37 cows in this study appear to be from clinically important secondary damage. A wide range of types of secondary damage was diagnosed, some of which was not damage to the musculoskeletal system, and some conditions that are not currently listed in the downer cow syndrome literature. Veterinarians must consider secondary damage when examining any recumbent cow as the management of the secondary complications that can arise may be different to that of the primary cause of the recumbency, and may be the only reason for the persistent recumbency.

Downer cow syndrome was redefined to be "caused by clinically important secondary damage, which is secondary damage that can cause recumbency in its own right, or delay or prevent recovery from the primary cause of the recumbency".

Endnote

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