Humeral fractures in dairy heifers

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A case series of humeral fractures in dairy heifers on one spring-calving dairy farm in the Manawatu has been previously reported at the 2008 DCV conference. In that instance, six of 200 crossbred heifer replacements were found to have spontaneous fractures of the humerus within the first couple of months of lactation. Two of these heifers had suspected greenstick fractures and recovered after being dried off but a third had a suspected greenstick fracture, was dried off but fractured the other humerus within a few days and was also euthanased. Copper deficiency was diagnosed on the basis of low copper levels in serum or liver of several of the affected animals. A planned copper injection for this group of animals was missed in early winter (3-5 months previously). Following treatment with copper injection no further cases were reported.

In the past four years, further cases of humeral fractures in dairy heifers from many parts of New Zealand have been reported to veterinarians at Massey University either directly or via veterinary diagnostic laboratories. On one farm, 10 heifers were affected. Some outbreaks have been associated with copper deficiency but this has been ruled out on other farms. This emerging syndrome appears to be occurring predominantly in dairy heifers early in their first lactation but occasionally prior to calving or in cattle in their second lactation. Cases are characterised by acute forelimb lameness with no history of trauma and the heifers are typically in good condition. The heifers usually sustain these fractures in the paddock or on the tracks rather than in the yard or milking shed. Mid-shaft, spiral fractures are seen in all cases that are necropsied and histologically there appears to be a reduction in the porosity of trabecular bone (possibly due to excessive pararathyroid activity) with growth arrest lines in some cases and evidence of resorption cavities in cortical bone. Examination of matched control animals needs to be undertaken to establish the significance of these findings.

We hypothesise that these spontaneous fractures are occurring as a result of skeletal fragility that may be due to a growth check during the rearing period rather than a specific traumatic event in early lactation. The forelimb supports 60% of bodyweight and is therefore more at risk. The pathogenesis may involve a combination of predisposing factors but inadequate bone mass is likely to be the primary problem. Possible predisposing factors include:

- High genetic potential for milk production.
- Previous copper deficiency leading to skeletal fragility.
- Inadequate dietary calcium due to maize silage feeding.
- Pre-calving DCAD modification may exacerbate any osteoporosis.

Copper deficiency has been associated with fractures in a number of species due to copper being required for the cross linkage of collagen (as a component of lysyl oxidase). Copper status at the time of fracture is not necessarily relevant as bone formed during a period of copper deficiency will remain fragile until replaced by normal remodelling.

Information about herds with cases of humeral fractures in heifers were called for via the DCV membership and in December 2011 a survey was sent to these farmers to gather information about the management of that group of heifers as well as specific details about the affected animals. Results from this preliminary survey are not yet available but will be presented at the conference. This survey may be followed by a case-control study if funding

permits but we are also happy to receive samples of fractured and control bones for computed tomography to assess mineral status as well histomorphometry. Biochemical investigation of bone resorption markers in late gestation and early lactation may also be carried out. Samples that should be sent for a thorough investigation include:

1. The fractured humerus – ideally chilled and sent ASAP or sawn sagitally and fixed in formalin or frozen. Collect contralateral humerus – to allow comparison.
2. Serum and EDTA (two of each if possible) and preferably sent after the serum has been spun down, send chilled.
3. Liver biopsy/sample – for copper analysis.
4. 30-40 hairs (approximately the thickness of a pencil) from the tail which will be stored in case DNA analysis for genetic risk factors is undertaken. The bulbous part of the hair root needs to be present and ideally there should be no faecal or moisture contamination. Freeze this sample if not sending immediately.
5. Urine sample – send fresh or with other chilled samples or freeze and send with frozen/formalin fixed samples at your convenience.

At this stage the only recommendations we can suggest to farmers are to ensure that heifers are fed optimally throughout the rearing period when they are establishing bone mass, ensure that copper status remains adequate throughout their life and that DCAD reduction in very late pregnancy may be detrimental to heifers who don't require dietary modification to minimise the risk of hypocalcaemia anyway.

The authors can be contacted to include other affected farms in the survey and it is intended that the results will be distributed to production animal veterinarians via NZVA publications. The authors wish to thank Alan Julian (Gribbles Veterinary Pathology) for the referral of cases and the veterinarians and farmers who have contacted us to date.