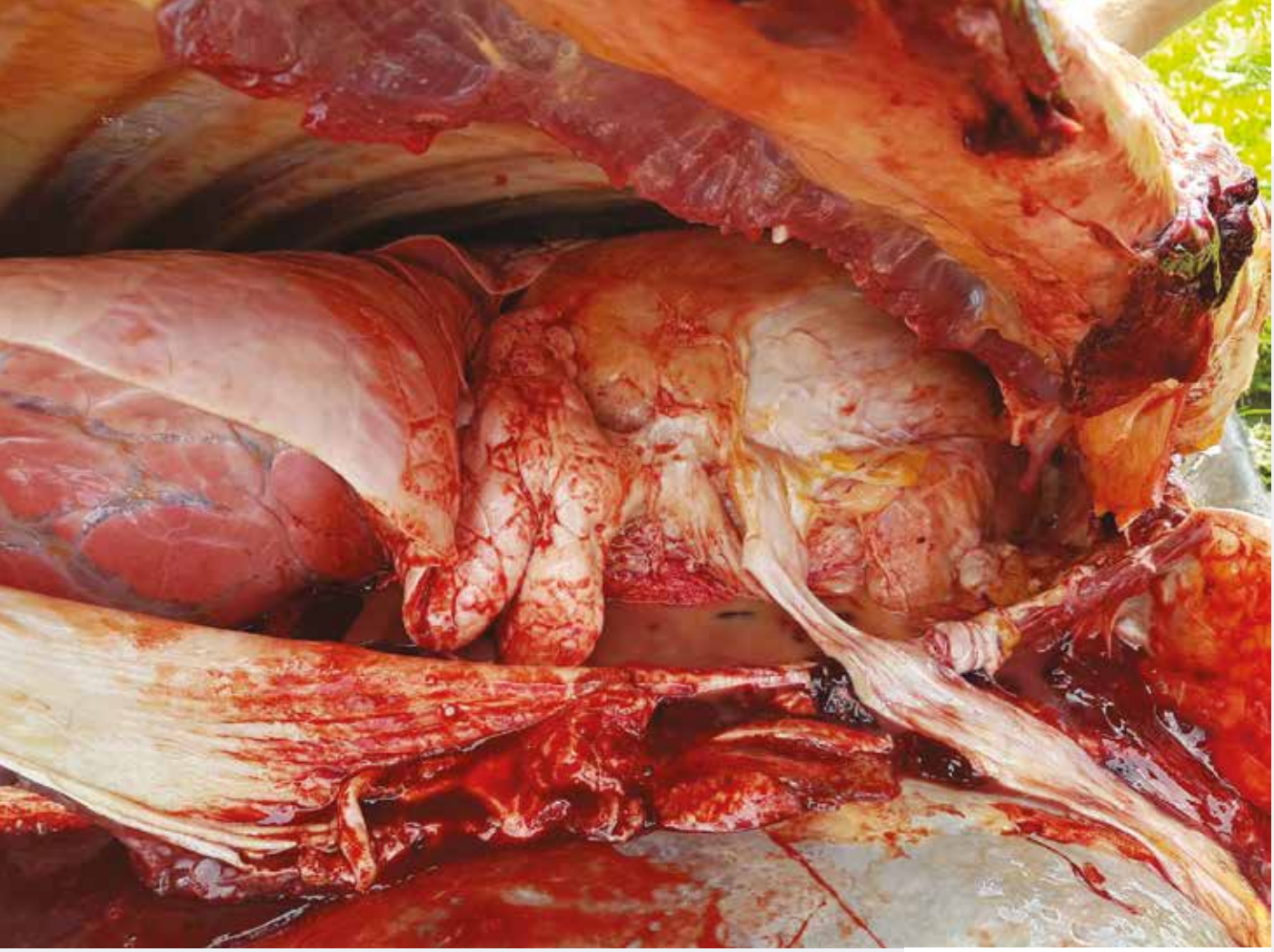


FIGURE 1: On-farm necropsy revealing severe fibrinous pericarditis.

A *hidden* menace

Pathologist **Alice Fraser** of SVS Laboratories, Hamilton, highlights two recent cases of hardware disease in dairy cows arising from feed supplements.

INTRODUCTION

Palm kernel extract (PKE), a by-product from the palm oil industry in Southeast Asia, is widely used as a feed supplement for dairy cows in New Zealand, particularly when pasture availability or forage supplements are scarce. Due to the nature of the initial mechanical extraction process to obtain the oil from the hard palm nut, there is the possibility of contamination of consignments with metal fragments or metal objects from the extraction machinery itself, or from the containers in which PKE is shipped. For this reason, at the feed merchants PKE is passed through large screens fitted with magnets to remove metal contaminants before it is dispatched to dairy farms where it is combined in mixer wagons that are often also fitted with magnets.

However, when PKE demand is high, requiring rapid throughput, the metal decontamination procedures can fail, leaving cows at risk of ingesting metal fragments or objects, leading to hardware disease (or traumatic reticuloperitonitis (TRP)).

This issue was previously raised in 2013, when hardware disease cases arose in dairy cows on a Wairarapa farm, leading to measures being put into place by MPI to reduce contamination. Despite these precautionary measures, cases of hardware disease were diagnosed in dairy cows in the Waikato region in spring 2017. The notable aspects included

PHOTOGRAPHY: SUPPLIED

several cows on a property being affected over time, non-specific presenting signs and a protracted course of disease.

The following case reports are written to increase awareness of the potential issues associated with feeding PKE.

CASE 1

The first case occurred in a dairy herd of 400 cows, of which 200 had been purchased in the previous six months due to a recent change to autumn calving. The cows' diet was pasture supplemented with maize and PKE.

Clinical details: Seven unexplained deaths of mature dairy cows occurred over a period of six months, all from the mob of cows purchased. The cows went off their milk, slowly lost body condition and had slightly pale mucous membranes. All cases were refractory to symptomatic therapy and had to be euthanased. Blood samples from an affected five-year-old cow were submitted to the laboratory.

LABORATORY FINDINGS:

Serum biochemistry of the cow showed moderately high globulin of 67g/L (reference range 31-54g/L), consistent with inflammation. Albumin was moderately decreased at 18g/L (reference range 25-40g/L), which was considered to be partly due to inflammation (negative acute phase protein), but at this level of decrease there were likely to be additional losses such as via the gastrointestinal tract. She also had increased liver enzymes, with GLDH minimally increased up to 47U/L (reference range 8-41U/L) and GGT moderately increased up to 106U/L (reference range 1-36U/L), indicating minimal hepatocellular damage and moderate biliary epithelial damage such that spring eczema, liver fluke damage, hepatic abscesses and gastrointestinal lesions were potential differentials. Serology for *Fasciola hepatica* and faecal fluke egg examination were both negative.

NECROPSY FINDINGS:

The cow deteriorated and was euthanased. An on-farm necropsy revealed a severe, chronic pericarditis and pleural-diaphragmatic adhesions consistent with hardware disease. There were also multiple liver abscesses.

WHEN PKE DEMAND IS HIGH, REQUIRING RAPID THROUGHPUT, THE METAL DECONTAMINATION PROCEDURES CAN FAIL, LEAVING COWS AT RISK OF INGESTING METAL FRAGMENTS OR OBJECTS.

Since several cows had been affected and no other potential sources of metal were known, PKE supplies were investigated and a significant amount of metal contamination was found, including metal shards and metal objects (eg razors and spanners). With the benefit of a definitive diagnosis, when a further cow started showing similar signs, she was administered a magnet and made a successful recovery.

CASE 2

Over a period of approximately two months, six mature dairy cows out of a mob of 200 showed non-specific illness, including drops in milk production, ill-thrift and slightly pale mucous membranes. The cows were fed a similar diet to that in case 1. Blood samples from two of the affected cows were submitted for analysis.

LABORATORY FINDINGS:

A *Theileria* panel, including haematocrit, haemoglobin and smear exam on the EDTA blood sample of one cow, showed a mild, non-regenerative anaemia (HCT 0.24L/L, reference range 0.24-0.40L/L, haemoglobin 77g/L, reference range 85-130g/L) with no evidence of Theileria organisms. Biochemistry results revealed a slightly low creatinine (45umol/L, reference range 55-130g/L) due to the loss of body condition (BC). There was a slightly low chloride (81mmol/l, reference range 96-104mmol/l), and the corrected chloride, with sodium at 140mmol/l (reference range 132-152mmol/l), was 82.16mmol/l, indicating that this was effectively a chloride loss only, likely due to pooling of abomasal secretions, or abomasal obstruction (physical or functional). Bicarbonate was significantly increased (41.9mmol/l, reference range 26-34mmol/l), indicating a metabolic alkalosis and again suggestive of abomasal pooling of HCl. High globulins (60g/l, reference range 31-54g/l) indicated inflammation. Overall, the results pointed towards abomasal dysfunction with a likely abomasal-associated inflammatory nidus, such that hardware disease topped the list of differentials.

For the second cow there were fewer biochemistry changes to indicate abomasal dysfunction. There was evidence of severe loss of BC, with a creatinine of 25umol/l, no evidence of electrolyte loss or metabolic alkalosis, but a high globulin level of 61g/l and a slightly low albumin of 23g/l (both consistent with inflammation, with albumin reduction due to hepatic switch to globulin production – albumin being a negative acute phase protein). The results from this cow gave a less specific diagnosis, but the indication of a chronic-active inflammatory disease, combined with the clinical history and the diagnostic findings from the first cow, suggested a similar diagnosis.

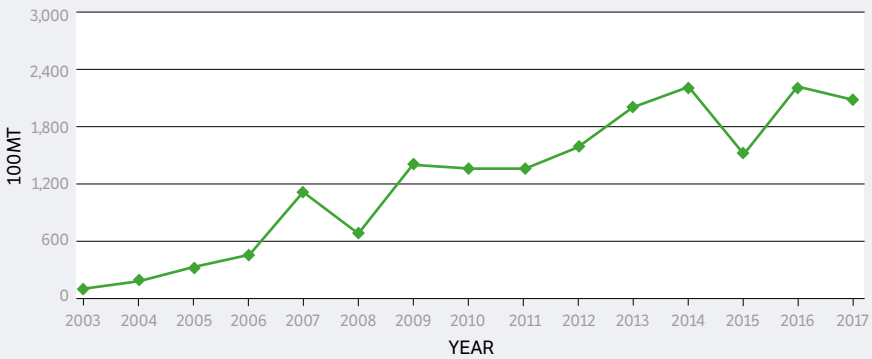
DISCUSSION

Hardware disease in cattle is a relatively common consequence of perforation of the reticulum following ingestion of sharp metallic objects. The four potential outcomes of ingestion of metal objects are (Miesner and Reppert, 2017):

1. Penetration of the reticular wall without entering the peritoneal cavity, causing focal reticulitis and mild clinical disease.

GRAPH 1:

New Zealand palm kernel meal imports by year



2. Perforation of the reticular wall and entrance to the peritoneal cavity, causing acute localised TRP.

3. Perforation of the reticular wall and entrance to the peritoneal/thoracic cavity, resulting in pericarditis, myocarditis, abscessation, vagal indigestion or other secondary disease.

4. Attachment to a prophylactic magnet administered into the reticulum.

The typical presentation of acute cases of hardware disease includes a sudden drop of milk production, anorexia, reluctance to move and signs indicative of cranial abdominal pain, such as abducted elbows and a grunt on the wither test.

The cases reported above, and other similar cases coming through the laboratory, tended to be of a more insidious nature, with vague, chronic clinical signs. This may be due to the small size of metal shards in PKE causing smaller inflammatory lesions with a more protracted disease process.

The increased number of cases was probably due to a huge demand for PKE because of wet winter and low pasture growth. Due to the higher PKE throughput, magnets in the feed/mixer wagons at processing sites may have become clogged with metal fragments and not cleared at regular intervals. In short, the metal decontaminating system had likely become overwhelmed and/or the magnets used to pick up the metal were not completely reliable, and the level of metal contamination in PKE consignments leaving the feed merchants had probably increased. The effect of high levels of small metal fragments was such that multiple cows were affected, and clinical signs were more chronic and insidious than those seen in the usual causes of hardware disease.

This is not a new issue for PKE. Similar cases were reported in the press (Stuff.co.nz) in December 2013, involving a dairy farm in Wairarapa where two cows died of hardware disease. In that case the farmer took the feed company to the Disputes Tribunal for compensation. Following this incident MPI investigators put regulatory checks in place at the level of palm oil producers.

In October 2016 MPI denied the discharge of a PKE consignment from a ship carrying PKE from an unregistered Malaysian facility due to the biosecurity risk.

This would not be a balanced article on PKE if it did not mention the industry from which this supplement is a by-product. The palm oil industry in Indonesia/Malaysia produces millions of tonnes of palm oil per year, extracted from the native forest tree *Elaeis guineensis*, for use in numerous manufactured foods, healthcare and engineering products, etc throughout the developed world. The consumer demand for this oil comes with far-reaching ecological, environmental and cultural costs, the devastating details of which are beyond the scope of this article.

As a result, Landcorp has ceased using PKE on any of its dairy farms. Fonterra has more recently pledged to purchase PKE only from palm oil firms that are not involved in any further deforestation. Fonterra has also requested that dairy farmers limit the amount of PKE used to no more than 3kg/cow/day since it affects the milk composition, giving indications that the cooperative is taking some steps to refocus on milk quality versus milk quantity. For a pasture-based industry, the use of supplements such as PKE has increased considerably in the past 10 years (see Graph 1; IndexMundi).

CONCLUSION

This article outlines cases in which hardware disease affected multiple cows on two Waikato properties as a result of PKE metal contamination, and draws our attention to the potential day-to-day, on-farm issues that can result from PKE use. There is also a bigger picture to consider: the amount of supplements in dairy cow diets imported by the dairy industry, and awareness of the provenance of these supplements. On the one hand PKE is a useful, economical feed supplement for dairy cows; on the other it can affect cow health and milk quality and its use supports a highly destructive palm oil industry. VS

REFERENCES:

Miesner MD, Reppert EJ. Diagnosis and treatment of hardware disease. *Veterinary Clinics: Food Animal Practice* 33(3), 513–523, 2017

Websites:

www.indexmundi.com/agriculture/?country=nz&commodity=palm-kernel-meal&graph=imports

www.msdvetmanual.com/digestive-system/diseases-of-the-ruminant-forestomach/traumatic-reticuloperitonitis

www.stuff.co.nz/business/9525310/Farmer-loses-cows-to-feed-hardware

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