Jejunal haemorrhage syndrome

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looks at this recent disease that has mortality rates of between 85 per cent and 100 per cent and reports on work to understand its causes and raise awareness

THE first account of jejunal haemorrhage syndrome (JHS) was published by Ruggles et al in 1992 and it has been reported in both beef and dairy cows in the United States, Europe and the Middle East.

Since JHS received a veterinary investigation disease analysis (VIDA) code in May 2011, disease surveillance centres (DSCs) have recorded JHS cases diagnosed on postmortem examination. The VIDA gross pathological diagnostic criteria states "segmental jejunal haemorrhage with mucosal necrosis and blood clot in the rumen".

Referring to VIDA data there were three cases in 2012 and nine cases in 2013; six of which occurred in Scotland.

An increase in the number of diagnoses of JHS prompted SAC Consulting Veterinary Services to further investigate the aetiology and predisposing factors related to this condition, which are not fully understood. A clinical summary from the literature is included to raise awareness of this condition that is becoming increasingly important to the UK dairy industry.

Although cases have been reported in beef cattle, JHS is predominantly a disease of adult dairy cows.

Retrospective studies in the literature report a higher incidence of disease in early lactation and older cows (Elhanafy et al, 2013). Berghaus et al (2005) carried out a study to identify specific risk factors for JHS. Findings included larger herds are more likely to have cases of JHS and that JHS is less likely in herds where grazing is part of the cows' diet. The study concluded consumption of high-energy diets appears to be a significant risk factor.

Clinically, cows present acutely with a combination of the following clinical signs:

- lethargy and depression;
- acute milk drop;
- distended abdomen;
- ruminal hypomotility;
- bruxism;
- reduced faecal output;
- tachycardia;
- hypothermia;
- anorexia; and
- dehydration (Dennison et al, 2002; Abutarbush et al, 2005).

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Typically cows are apyrexic. Rectal examination can be useful – although palpation of the portion of jejunum containing the blood clot is unlikely, distended loops of small intestine proximal to the blood clot may be palpable. Melaena is not a consistent finding, although scant dry faeces in the rectum is common (Abutarbush et al, 2005; Dennison et al, 2002).

The clinical signs are suggestive of an intestinal obstruction. More specifically, other differential diagnoses include:

- right-sided dilation/torsion abomasum;
- caecal dilatation/torsion;
- torsion of the root of the mesentery; or
- intussusception.

There is a high mortality rate of between 85 per cent and 100 per cent and often cases are found dead (Abutarbush et al, 2005).

Biochemical and haematological parameters are non-specific, but can provide additional information to the clinical picture.

Neutrophilia and leukocytosis are common haematological findings (Dennison et al, 2002; Abutarbush et al, 2005). Hyperglycaemia is consistently reported in cases of JHS and is thought to be attributed to a stress response to disease (Dennison et al, 2002). Acid base balance is suggestive of gastrointestinal stasis; hypochloraemia, hypokalaemia metabolic alkalosis with a compensatory respiratory acidosis (Abutarbush et al, 2004; Dennison et al, 2002).

Transrectal and transabdominal ultrasonography can be a useful adjunct to clinical examination, haematology and biochemical analysis. However, a definitive diagnosis of JHS can only be made in 20 per cent to 25 per cent of cases as often the pathology is outside the depth of penetration accessible by ultrasonography (Braun et al, 2010).

Dilated loops of intestine are often present in the ventral aspect of the right paralumbar fossa. Braun et al (2010) report distension of the small intestine ranging from 4.3cm to 12cm and absence of motility in the small intestine in 98.4 per cent of cases. Identification of homogeneous echogenic material consistent with an intraluminal blood clot was possible in only 19 per cent of cases. Dennison et al (2002) report similar findings with an identifiable blood clot in only four out of 12 cases examined by ultrasonography.

Treatment

Medical treatment has been largely unsuccessful. Treatments have included intravenous fluid therapy with either trimethoprim and sulfadoxine combination or oxytetracycline; in a study of 11 cases treated medically in this way one case died and 10 were euthanised (Abutarbush et al, 2005).

Dennison et al (2002) reported seven out of eight cows dying despite medical treatment – treatments included a combination of flunixin meglumine, intravenous fluids with electrolytes, calcium salts IV, procaine penicillin, ceftiofur, *Clostridium perfringens* type C and D antitoxin, metoclopramide, three per cent lidocaine, dexamethasone, transfaunation, hypertonic saline, butorphanol, morphine, magnesium sulphate, neutral buffered 10 per cent formalin.

Surgical intervention is reported to be marginally more successful than medical treatment alone.

Dennison et al (2002) performed surgery in 13 cases – four were euthanised intraoperatively due to the extent of the lesions. In two cases the clot was massaged without enterotomy, enterotomy was performed and the blood clot removed in five cases and enterectomy and anastomosis was performed in two cases. Out of the nine remaining cases only four survived.

Peek at al (2009) carried out a retrospective study into surgical treatment of JHS, where treatments were allocated depending on the clinical presentation of the case – 18 out of 31 (58 per cent) survived until initial discharge.

Standing right flank laparotomy and manual massage of the blood

⇨ page 16

⇨ continued from page 14

clot was performed in cases where the intestine was not compromised. This procedure was associated with significant short-term survival compared to enterectomy and anastomosis or enterotomy and removal of the blood clot. However, this may be due to the intestine not being compromised when surgery was performed rather than the surgical procedure itself.

Causal factors

C perfringens type A has been suggested as a causal agent for JHS and has been isolated from many clinical cases of JHS in the literature (Abutarbush et al, 2005; Schlegel et al, 2012; Dennison et al, 2002). Overgrowth of *C perfringens* in the small intestine can occur when there is overflow of rapidly fermentable carbohydrates from the abomasum (Ewoldt and Anderson, 2005).

Overflow is associated with the risk factor for subacute ruminal acidosis (SARA); insufficient effective fibre inclusion in the ration; ration sorting and feeding excessive amounts of soluble carbohydrate. As a result, some authors consider subacute ruminal acidosis as a predisposing factor for JHS (Tajik et al, 2010; Godden, 2003).

This hypothesis may link the identified risk factor associated with high energy density diets to *C perfringens* type A. However, *C perfringens* type A is a common isolate from the gut of cattle without any clinical signs and proliferates quickly after death. Therefore, isolating *C perfringens* from intestinal contents postmortem is of dubious significance (Songer, 1996).

The involvement of *C perfringens* in JHS was questioned when inoculation of the abomasum and small intestine with *C perfringens* type A failed to reproduce the clinical syndrome (Ewoldt and Anderson, 2005). *C perfringens* involvement in the pathogenesis of JHS has yet to be established; however, many authors consider vaccinating with multivalent clostridial vaccines as a preventive measure (Peek et al, 2005; Elhanafy, 2013).

Aspergillus fumigatus DNA has been detected in the gastrointestinal tract of cases of JHS and was not detected in cows with alternative gastrointestinal disease (Socket, 2004). *Aspergillus fumigatus* has the potential to produce genotoxic and cytotoxic mycotoxins such as gliotoxin. Gliotoxin has antibacterial, apoptotic and immunosuppressive effects (Morgavi et al, 2004). Uncertainty remains about whether *Aspergillus fumigatus* is the primary pathogen or whether toxins produced by the fungus result in immunosuppression allowing for a secondary pathogenic process (Elhanafy et al, 2013).

As the specific cause of JHS is as yet unknown, general preventive measures are targeted at maintaining rumen health, minimising SARA and providing good quality feed (Peek et al, 2005).

SAC Consulting Veterinary Services has carried out a retrospective study into 12 cases of JHS submitted to DSCs in Scotland since 2011. No seasonal predisposition was associated with when the cases occurred and no association was noted between age and stage of lactation.

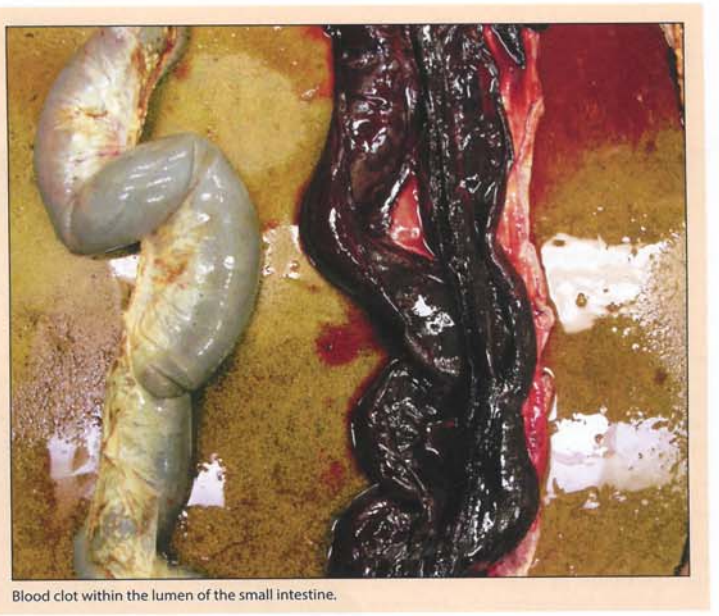
Although the study did not provide sufficient information on the risk factors associated with JHS there were some interesting findings. Histopathological examination of the mesentery in two cases provided additional insight into the pathogenesis of the disease. There was acute, localised, mixed inflammation response and associated vasculitis and perivascular haemorrhage.

From analysis of both these cases it is conceivable the pathophysiology of JHS starts in the mesentery. Interestingly, *C perfringens* was only isolated on two occasions from the intestinal contents of the 12 cases and clostridial enterotoxin testing was carried out by ELISA in nine out of 12 cases – no alpha, beta or epsilon toxins were identified. This finding contradicts the hypothesis that *C perfringens* type A is involved in the pathogenesis of JHS.

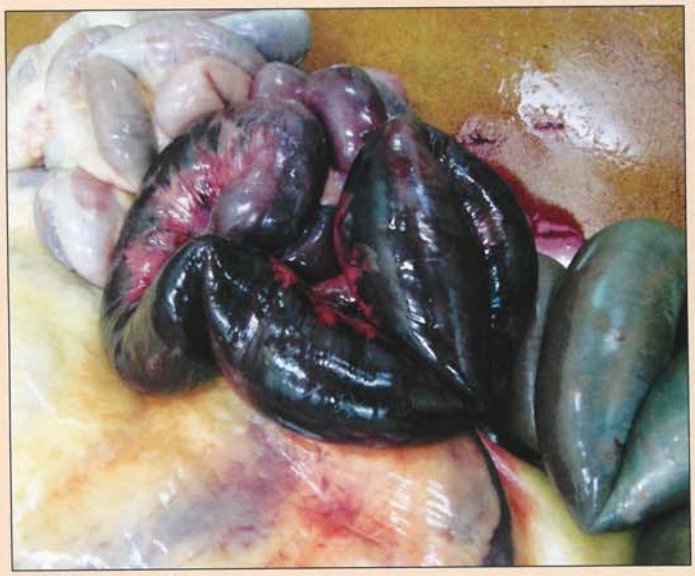
Furthermore, *Lichtheimia corymbifera* and *Rhizopus arrhizus* were isolated from two separate cases. Both are mucoraceous fungi found in decaying plant material. Gastrointestinal mucormycosis is reported in humans and presents as gastrointestinal haemorrhage with ulceration and necrosis (Kahn, 1963). This is a conceivable aetiology for JHS and requires investigation in future cases.

SAC Consulting Veterinary Services is carrying out further research into JHS to try to identify a causal factor and establish preventive measures to reduce the incidence of this acute condition, which is of increasing economic importance to the dairy industry.

We would be interested to hear from any farmers or veterinarians who have any experience of this condition.



Blood clot within the lumen of the small intestine.



Section of affected jejunum.

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graduated from the University of Edinburgh Royal (Dick) School of Veterinary Studies in 2007. Having worked in predominantly farm animal practice for five years, Lee-Anne joined SAC Consulting Veterinary Services in 2013 as a veterinary investigation officer. Since then Lee-Anne has been carrying out research into JHS to further understand its predisposing factors, aetiology and pathogenesis. Lee-Anne has now returned to farm animal practice in Northumberland.

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RVC epilepsy research highlights drug response variables

RESEARCH from the RVC has highlighted factors influencing why some dogs do not respond to antiepilepsy treatments.

The study by the college's canine epilepsy clinic sought to find out why some dogs respond to treatment and become seizure-free, while others continue to have seizures.

It analysed patient data from six years of medical history taken from the epilepsy clinic at the RVC's small animal referral hospital.

At the point of follow up, only 14 per cent of dogs studied were in seizure-free remission.

The results showed seizure density, rather than the number of seizures, was a more accurate sign of achieving remission in canine epilepsy.

Similar results have been found in human epilepsy. Continuing research into the drug treatments of the condition in dogs could improve understanding of the disorder in humans.

In human medicine, epilepsy patients are usually treated with antiepileptic drugs (AEDs) immediately after the onset of the condition.

This study found time to treatment after diagnosis, or the number of seizures experienced before treatment, did not affect the likelihood of achieving remission. The dog's gender was also found to be an important risk factor, with male animals less likely to go into remission than female dogs receiving AED treatments.

Other studies into canine epilepsy have focused on specific breed. Due to the data gathered from the RVC hospital, this research was able to look at how epilepsy affects a wider section of dog types. It found border collies and German shepherd dogs were at a significantly higher risk of not responding to AEDs than other breeds.

Clinical director of the hospital and specialist in neurology and neurosurgery Holger Volk said canine epilepsy was complex and could be very distressing for the dog and its owner

"Drug treatments can be successful in reducing seizures, but it is important to note consistent remission is difficult to attain," Prof Volk added

Co-author of the study Rowena Packer said in its worst form canine epilepsy could be life-threatening to dogs. "But it is a dog's long term quality of life that is most affected," D Packer said. "It can also take a toll on the owner who have to manage this unpredictable, uncontrollable condition."

The research paper will be published in academic journal *PloS One*.