Histopilus somnus in cattle

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INTRODUCTION

Histophilosis is a bacterial disease of cattle found worldwide. It presents with clinical syndromes that include pneumonia, meningoencephalitis, myocarditis and arthritis. Collectively these presentations are referred to as the *Histophilus somni* disease complex (1). Cases can present showing a single or combination of these syndromes. Since first reported in North America in 1956 the incidence of the various syndromes has changed in North America (2). There have been no Australian reports of a similar change in the incidence of presenting syndromes.

Within Australia, *Histophilus somni* (formerly *Haemophilus somnus*) is commonly viewed as a bovine respiratory disease pathogen, most commonly in feedlots but also post-transport in grazing enterprises.

Prior to 2009, the experience of one of the authors was that histophilosis was rarely diagnosed and only presented as pneumonia post-transport.

Since 2009, District Veterinarians across the North West LHPA, a state livestock health administration district in northern NSW, have diagnosed histophilosis more commonly, with non-pneumonic presentations and in circumstances unassociated with transport.

CASE STUDIES

Case 1 – August 2009

Histophilosis killed seven weaner cattle in a mob of 170 head on a commercial beef operation near Warialda. Another 4 head became ill but recovered with treatment with oxytetracycline antibiotics. The affected animals were home-bred and had not been subject to any of the normal stressors associated with bovine respiratory disease.

The mixed sex weaners in the mob were all home-bred except for 14 heifers introduced from Dorrigo 4 weeks earlier. All deaths and subsequent cases were in the home-bred portion of the mob. The mob had been grazing a fodder oats crop for some months. The oats at the time of the deaths was beginning to dry off.

The initial 6 deaths occurred over an 8 day period. The initial District Veterinarian investigation found the carcasses near and in fence lines in a pattern which suggested that they may have been affected by blindness prior to death.

Necropsy of a steer that had died overnight revealed a moderately autolysed carcass with a generalised septicaemic appearance and slightly swollen liver. Lung changes were not obvious.

Examination of smears for anthrax were negative, as was lead testing on the kidneys. Histology found “marked multifocal to coalescing, suppurative meningoencephalitis with intralesional coccobacilli and vasculitis consistent with bacterial infection, most likely *Histophilus somni***.”
Prior to receiving the above histology result, another heifer was found withdrawn from the mob and seeking shade. Clinical signs included dyspnoea and frothing at the mouth.

This heifer died within a few days and was investigated. On necropsy lung changes were not marked, with froth in the airways and congestion on the dependant side. The liver was moderately swollen and the carcass had a septicaemic appearance. The brain appeared normal.

*H. somni* was isolated from the lung and brain. Histology of the brain revealed a “severe multifocal to coalescing, suppurative and fibrinous meningoencephalitis”.

Case 2 – June 2010

Two hundred steers were weaned on a property at Rowena, transported to Narrabri and treated with a HGP implant and Cydectin Pour On® (moxidectin) on arrival. The steers were kept in yards for two weeks after arrival and fed lucerne hay. They were then released onto oats and had been grazing the crop for four weeks. All were thriving except for one steer noticed to be sick for at least seven days prior to examination.

From a distance the steer appeared depressed, but when approached it became alert and aggressive. The animal was dyspnoeic with the head extended, shallow respiration and frothing from the mouth. It was emaciated and exhibited marked brisket oedema (Image 1).

**Image 1:** Emaciated, dyspnoeic and aggressive steer showing marked oedema of the brisket (Case Study 2).

A tentative diagnosis of traumatic reticulopericarditis (TRP) was made. The steer was euthanased and a necropsy conducted. Lesions suggestive of TRP were absent. The liver and kidneys were enlarged. There were extensive fibrinous lesions and abscesses throughout the thorax. The lungs were consolidated and showed extensive abscessation. The pericardial sac was approximately the size of a soccer
ball, had a thickened wall and contained more than 1L of purulent fluid (Image 2). The heart was shrunken, pale and covered by a 1cm thick fibrinopurulent crust (Image 3). The brain tissue had been obliterated by the gunshot to the head.

**Image 2:** An incision through the pericardial sac, showing the heart bathed in purulent fluid (Case Study 2).

**Image 3:** The pale shrunken heart, encased in a fibropurulent crust (Case Study 2).

Laboratory examination found that the steer had “severe, chronic, suppurative and constrictive pericarditis as well as severe, chronic, suppurative pleuritis and pulmonary abscessation”. The liver pathology was indicative of right sided heart failure. A profuse growth of *H. somni* was isolated from lung samples. It is unknown if the steer had meningoencephalitis as the brain tissue was unsuitable for
histology, however the aggressive behaviour was suggestive of this type of inflammation. The joints were not examined for evidence of arthritis.

Case 3 – July 2010

Five hundred fully clostridial vaccinated, Angus weaner steers and heifers (including home-bred and traded stock) were released onto a 160 hectare oats paddock in early May 2010 near Walgett (Image 4). The first death occurred 3 days after release into the paddock. Four further deaths occurred within 3 weeks. The owner did not notice any illness within the mob. An anthrax exclusion was conducted on a decomposing carcass at the end of May 2010.

Approximately one month later, the death of a weaner and illness of another heifer were investigated. The weaners were part of the mob as above, but were grazing a fresh crop of wheat. The weaner had died a few days earlier. The sick heifer was had a fever and showed respiratory signs. She was euthanased by gunshot and a necropsy conducted. Apart from an enlarged (20%) liver, the abdomen appeared normal. The lungs appeared normal. There was a spectacular pericarditis with pericardial adhesions (Image 5).

Image 4: Weaners (and camels) grazing oats (Case Study 3).
Histology of tissues was not undertaken, but culture of the pericardial fluid resulted in a sparse, pure growth of *H. somni*.

Case 4 – July 2010

Approximately one hundred yearling Angus heifers and steers were placed in an opportunity feedlot near Narrabri in early June 2010. The heifers were all home-bred and a few of the steers were traded.

Two heifers died suddenly about 3 weeks after being placed on feed. A necropsy was conducted on one of the heifers. The rumen contained only grain, despite the ration including both hay and grain. Most organs showed signs of advanced autolysis. Histology was conducted on the brain and the lesions were most consistent with listeriosis, although they were also consistent with histophilosis.

A third heifer was still alive. She had a fever, pale mucous membranes, increased respiratory noise and ataxia. Testing revealed that she was persistently infected with pestivirus. Testing for Chlamydia (Sporadic Bovine Encephalomyelitis) was negative.

Four further heifers became ill the following day. The first was found in lateral recumbency with flaccid paralysis. She did not have a fever. She was euthanased, but no abnormalities were noted at necropsy. Histology of the brain revealed meningoencephalitis consistent with histophilosis. Histology of other organs was normal. Culture of the brain tissue was unrewarding.

The three sick heifers were also examined (Image 6). One had a fever and all showed varying degrees of ataxia/paresis which was more pronounced in the hindlimbs. None showed signs of blindness or altered mentation.
Blood samples from the three heifers were negative for lead. All were treated with oxytetracycline antibiotics. Paired serology samples from the three heifers excluded chlamydia. None of the heifers were persistently infected with pestivirus.

DISCUSSION

Radostits *et al* (1) discuss diseases due to *H. somni* both in the section on Bovine Respiratory Disease and also separately as the cause of the *Histophilus somni* disease complex. In the second syndrome Australia is not included in the list of countries where it has been reported.

A search of peer reviewed literature failed to find any reports of *H. somni* disease complex in Australia. Communication from pathologists at the NSW state laboratory at Menangle indicates that cases do occur. In contrast, *H. somni* as one of the pathogens of Bovine Respiratory Disease in Australia is reported (3) (4).

Within the previous Narrabri RLPB district of the North West LHPA during the 17 years prior to 2009, histophilosis had been only diagnosed twice, both times as bovine respiratory disease associated with transportation stress. In the same period pasteurellosis was diagnosed 26 times, again almost entirely post-trucking. Potential errors in these records include the possibility that a small number of pericarditis cases that were diagnosed as traumatic reticulo-pericarditis may have been due to *H. somni* septicaemia. Likewise, a small number of individual cattle showing nervous signs and responsive to oxytetracyline, but negative for chlamydia serology, may have been due to infection with *H. somni*.

Orr (2), through the detailed analysis of histophilosis submissions to a North American University diagnostic laboratory, showed an increase over time in the incidence of pneumonia and myocarditis and the decrease in cases of encephalitis. Orr suggests that this change may reflect differences in the route of infection, with
inhalation more likely to result in thoracic lesions and haematogenous spread, possibly from a wound, more likely to result in CNS involvement.

If the cases documented in this paper reflect a change in the clinical presentation and risk factors for histophilosis in the North West LHPA, it is worth noting that in 2009 much of the LHPA area was emerging from a decade-long dry period.

REFERENCES


