Clinical and epizootiological study of a leptospirosis outbreak due to *Leptospira canicola* in a feedlot

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ABSTRACT

This report describes the epizootiology, clinical presentation, diagnosis and treatment of an outbreak of leptospirosis caused by *Leptospira canicola* in feedlot calves. The infection appeared to be of high morbidity with a cumulative clinical incidence of 15.6 %, cumulative subclinical incidence of 39 % and high mortality (8.3 %). Clinical disease was diagnosed in 4–8-month-old calves, while subclinical infection occurred in 9–12-month-old calves. Subclinical infection was based on serological evidence only. The zoonotic aspects of the infection are emphasised.

Key words: cattle, feedlot, *Leptospira canicola,* outbreak.

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INTRODUCTION

Leptospirosis is a common zoonotic disease that can affect most mammals[9]. The available evidence suggests that leptospirosis has an enormous economic impact on the livestock industry in tropical and subtropical regions[2–6], where optimal environmental conditions occur year round. Disease in cattle caused by *Leptospira canicola* is relatively uncommon although it has been described on occasion[4,7,8,11].

L. canicola may cause severe disease and even death, especially in young calves[6]. Dogs, wild carnivores, swine and hedgehogs may act as reservoirs of *L. canicola* infection[6]. Our report describes the clinical, pathological, serological and epizootiological findings of an outbreak of leptospirosis in a feedlot.

HERD HISTORY

The feedlot is located in the southern part of Israel (western Negev), with 800 calves (690 Israeli-Holstein and 110 cross-breed) ranging in age from 3 days to 12 months, and kept in 15 pens according to age and breed (Fig. 1).

The Israeli-Holstein calves were purchased from different dairy farms at an age of 3–6 days, and the cross-breed calves were purchased from 3 beef herds at an age of 4–6 months. Neither the feedlot calves nor the parent herds had been recently vaccinated against any *Leptospira* serovar.

CASE DESCRIPTION

During May 1994, approximately 18 calves showed sudden signs of lethargy, inappetence, pyrexia (up to 41.5 °C), icteric mucous membranes and haemoglobinuria. Within 3 days, 8 animals died, several of which were submitted for necropsy. Blood samples taken within the first few days of the outbreak did not reveal any evidence of babesiosis or anaplasmosis.

Post mortem findings included pronounced jaundice, enlarged soft and pale liver, grey patches and petechial haemorrhages on the surface of the kidney, haemorrhages in the lymph nodes and on the epicardium, blood-tinged urine, retention of bile and oedema of the gall bladder mucosa. Two cases exhibited an apical purulent pneumonia. Histopathological examination revealed multifocal tubulo-interstitial nephritis, multifocal portal hepatitis and multifocal areas of liver cell necrosis and inflammation of the gall bladder.

Serum samples from 40 clinically affected calves were submitted to the Kimron Veterinary Institute for serological examination. Three weeks later 209 calves were randomly sampled, comprising 15– 70 % of all animals in each pen (Table 1). All were tested by the microscopic agglutination test (MAT)[3] against the 8 *Leptospira* serovars: *L. canicola, L. pomona, L. grippotyphosa, L. icterohaemorrhagiae, L. hardjo, L. ballum, L. sejroe* and *L. swajizak.* The only serovar with serological titres of a magnitude considered compatible with clinical disease was *L. canicola.* Titres of more than 1:200 were considered to be associated with infection while 1:100 was suspect. Serum titres of 1:50 or less to *L. icterohaemorrhagiae* were seen in 8 samples but were considered non-specific. Serum samples from 4 dogs and 6 humans were also tested.

During May–July 1994, morbidity and mortality occurred in 5 pens with Israeli-Holstein calves aged 4–8 months, comprising 385 calves. Clinical signs as described above were seen in 60 calves (15.6 %) and 32 calves died (8.3 %).

In 150 (39 %) calves aged 9–12 months, the infection was clinically inapparent, although the seroprevalence in pen 2 was 100 %, pen 9, 71 % and pen 15, 50 %.

Attempts to isolate leptospires from the blood or urine were only made after the initiation of antibiotic treatment, owing to the early introduction of parenteral oxytetracycline and oral chlortetracycline. Initially 7 calves exhibiting pyrexia (>40.5 °C), jaundice or haemoglobinuria were bled by aseptic venipuncture and 1 mℓ of blood was inoculated into Kortov's medium with 5 % rabbit serum or EMJH medium with 0.2 % agar[10]. All media incorporated 100 µg/mℓ 5-fluoruracil (Sigma, Israel) and neomycin (Vitamed, Benyamina, Israel). Several months later, 20 calves that had survived the outbreak were slaughtered and 0.5 mℓ of macerated kidney tissue was sampled and inoculated into the same media. All of the attempts failed to isolate the bacteria.

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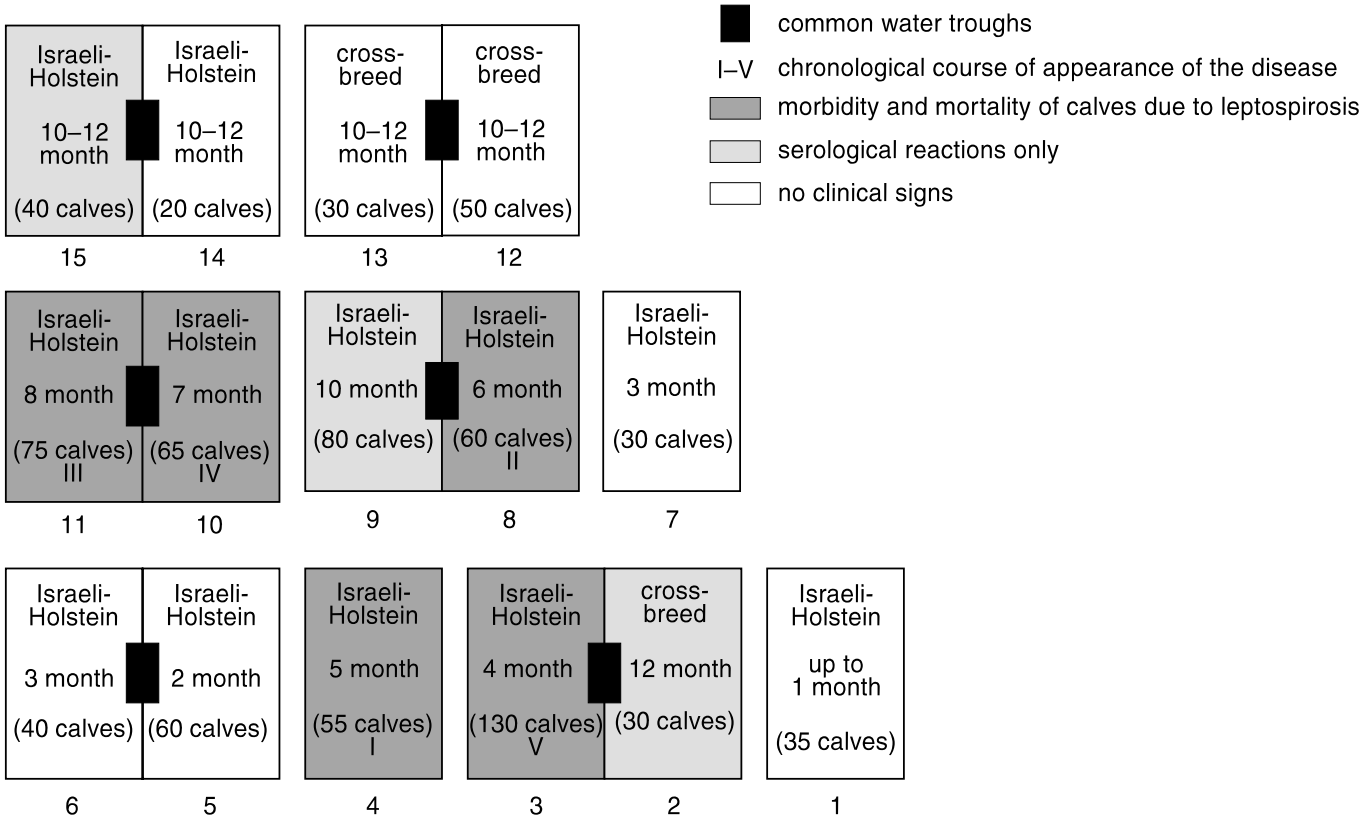

Fig. 1. **Leptospirosis outbreak in a feedlot.**

Dark-field microscopy did not reveal any spirochaete-like bacteria in any of the urine samples tested. Eight of the 20 calves had serological titres varying from 1:100 to 1:400 to *L. canicola.*

Histological preparations of liver tissue from one calf that had died with severe jaundice, haemoglobinuria and a serological titre of 1:200 to *L. canicola* were sent to the University of Guelph, Canada, for immunohistochemistry. A similar preparation was taken to the *Leptospira* OIE reference laboratory in Stormont, Belfast, Northern Ireland, for direct immunofluorescence. Both tests failed to detect the presence of leptospiral antigen.

Diagnosis of this leptospiral outbreak caused by *L. canicola* was, therefore, based on serological results supported by clinical signs, pathological findings, and response to treatment.

Serological examination of blood samples from 4 dogs owned by the feedlot workers showed no antibody titre. No clinical signs were observed or serological reactors detected among persons handling the infected cattle.

The animals were treated in 2 stages: 1) animals with clinical symptoms were treated once parenterally with long-acting oxytetracycline (Pfizer, 10 mg/kg) as cover for possible *Anaplasma marginale* infection, followed by dihydrostreptomycin at a dose rate of 20 mg/kg intramuscularly daily for 3 consecutive days; 2) medication of the feed with chlortetracycline (Aureomycin, Auropac®, American Cyanamid Company, Wayne, New Jersey) at a dosage of 50 mg/kg during a period of 12 consecutive days.

Seven days after the initiation of treatment, affected animals showed an apparent recovery. Nevertheless, the general health of the affected calves had deteriorated and there was a decrease in weight gain. These calves reached slaughter weight more than 6 weeks later than the expected average for the feedlot, based on previous seasons and compared with those that had shown no clinical or serological evidence of infection (estimated losses of approximately 70 kg body weight per animal).

Table 1. **Reciprocal of *Leptospira canicola* antibody titre according to feedlot pen.**

	Titre				Number of calves		Clinical disease
Pen	Negative	20–200	200–400	800–6400	sampled	in pen	
1	10	0	0	0	10	35	no
2	12	2	4	3	21	30	no
3	16	0	5	11	32	130	yes
4	12	2	0	7	21	55	yes
5	9	0	0	0	9	60	no
6	9	0	0	0	9	40	no
7	5	0	0	0	5	30	no
8	10	0	3	11	24	60	yes
9	13	1	3	7	24	80	no
10	6	1	2	4	13	65	yes
11	10	1	5	4	20	75	yes
12	6	0	0	0	6	12	no
13	5	0	0	0	5	30	no
14	4	0	0	0	4	20	no
15	3	0	1	2	6	40	no

The animals in the feedlot were followed clinically for 1 year, and no new clinical cases were recorded.

DISCUSSION

In this outbreak, the fact that only young calves were clinically affected while the older calves only reacted serologically, may indicate that the younger calves were more susceptible to infection. As the feeding and crowding conditions were similar in all the pens on this feedlot, it seems that the manifestation of clinical disease due to *L. canicola* might be age related. This is in contrast to the opinion expressed by Van der Hoeden[5]. High antibody titres were noticed in the infected calves during this outbreak (>1:6400), which is consistent with an acute infection. Similar observations with this serovar were made by Van der Hoeden[5].

Van der Hoeden *et al.*[6] claimed that, in the southern district of Israel, jackals are an important source of *L. canicola* infection. Although jackals were seen in the feedlot yards and sheds at night, capture of jackals is not permitted by the nature conservation authority, and it was therefore not possible to examine this species.

The essential epizootiological features for leptospirosis in cattle are environmental conditions that favour the survival of the organism. These include mud, swampy areas and standing water in the paddocks and sheds contaminated with infected urine[1], and moderate temperatures, as experienced during the months of the outbreak.

Although spirochaete-like organisms have been identified by dark-field microscopy, and *L. canicola* was isolated from the urine of serologically positive free-range cattle in northern Israel, there has, up to now, been no evidence to implicate the bacteria with clinical disease in these areas. As several of the calves involved in the present outbreak originated from these herds, they could possibly have served as vectors for *L. canicola* infection.

Human infections were not observed. In general, the pathogenicity of *L. canicola* for man is low[4]. Nevertheless, *L. canicola* may be regarded as a potential public health hazard[5].

Little has been documented on the dynamics of *L. canicola* excretion in cattle urine. According to Van der Hoeden *et al.*[6] the period of excretion is relatively short and only low numbers of *Leptospira* organisms are excreted in the urine of cattle. This is not supported by the present outbreak, where the disease seemed to be disseminated throughout the infected pens within an extremely short period.

The fact that all isolation attempts were unsuccessful, but were attempted after initiation of antibiotic treatment, suggests that specific treatment of all the apparent clinical cases coupled with a massive blanket regime of in-feed treatment of all the animals on the premises might eliminate the carrier state as well as the disease.

This outbreak, in contrast to the subclinical infection we have recently seen in extensively managed herds in northern Israel, seems to indicate a different epizootiological picture for acute *L. canicola* infection in intensively managed feedlot calves. The behaviour of the calves in crowded feedlot conditions, associated with urine licking and urination in water troughs, probably acted as a major contributing factor for the spread of infection amongst the animals within the pens.

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