



SHORT PAPER

Oculoglandular Syndrome Caused by *Yersinia pseudotuberculosis* in a Dairy Goat

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Summary

An outbreak of ocular disease in a herd of housed dairy goats was characterized by severe granulomatous conjunctivitis and ipsilateral parotid and submandibular lymphadenopathy. In one case submitted for post-mortem examination, *Yersinia pseudotuberculosis* was isolated from both the conjunctiva and submandibular lymph node. Histopathological examination identified severe chronic active suppurative and lymphoplasmacytic conjunctivitis and suppurative and necrotizing lymphadenitis associated with bacteria of yersinial morphology. Similar pathological changes occur in Parinaud's oculoglandular syndrome in man due to infection with *Y. pseudotuberculosis*.

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Yersiniosis is a significant cause of disease in goats and an important zoonotic infection (Buddle *et al.*, 1988; Smith and Sherman, 1994a). In a New Zealand study, Buddle *et al.* (1988) found that 6.5% of 324 goats subject to necropsy examination over a 15-month period died as a result of yersiniosis. Both *Yersinia pseudotuberculosis* and *Yersinia enterocolitica* are responsible for a variety of clinicopathological presentations in goats including enterocolitis, mesenteric lymphadenitis and, less commonly, septicaemia, with occasional reports of placentitis leading to abortion and mastitis (Cappuci *et al.*, 1978; Jones *et al.*, 1982; Slee and Button, 1990; Seimiya *et al.*, 2005; Brown *et al.*, 2007). With regard to the enteric forms of disease, infection leads to pathology ranging from a fulminant fibrinous or fibrinohaemorrhagic enterocolitis to a chronic erosive/ulcerative and suppurative enterocolitis. Characteristic lesions within the alimentary tract, associated mesenteric lymph nodes and, occasionally, viscera involve microabscess formation with gram-negative bacterial colonies within the intestinal mucosa and other organs (Brown *et al.*, 2007).

Asymptomatic intestinal carriage of the organisms is common, with seasonal variation and excretion and a high incidence of isolation occurring in the colder months of the year (Lanada *et al.*, 2005). This parallels the epidemiology of the disease, in which cold weather is one of the stress factors, together with poor feeding, endoparasitism, transportation and parturition (Slee and Button, 1990; Smith and Sherman, 1994a; Buddle *et al.*, 1998).

Oculoglandular syndrome is a well recognised disease of man, first described by Parinaud (1889). Chin and Hyndiuk (1985) defined oculoglandular syndrome, based on Parinaud's description, as a unilateral granulomatous conjunctivitis associated with ipsilateral preauricular and/or submandibular lymphadenopathies (these occasionally progressing to suppuration). They listed the causes of oculoglandular syndrome – the most frequent of which include *Bartonella henselae*, *Francisella tularensis* and *Sporothrix schenckii*. Occasional causes include infection with mycobacteria, *Treponema pallidum* or *Coccidioides immitis*. Both *Y. pseudotuberculosis* and *Y. enterocolitica* have been isolated in such cases (Bayer and von Herrenschwand, 1919; Kayser *et al.*, 1967; Ahvonen and Dickhoff, 1974; Crichton, 1978; Chin and Nobel, 1977). In 1983, McSporran reported an acute

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ulcerative conjunctivitis with swelling of the ipsilateral parotid lymph node in 3–5-month-old goat kids. *Y. pseudotuberculosis* was recovered both from conjunctival swabs and parotid lymph node aspirates. This is, to the best of the author's knowledge, the only reported case of oculoglandular syndrome in goats, but the pathology of the condition has not been described. We report a recent outbreak of ocular disease in a large goat herd with cases presenting as oculoglandular syndrome. In one case submitted for post-mortem examination, *Y. pseudotuberculosis* was isolated.

An outbreak of ocular disease in a large commercial dairy goat herd of 1200 head was first noted in February 2008. Although no accurate records were kept, incidence was initially high, but by the end of April/early May only occasional cases were seen. The total number of animals affected over this period was approximately 50–60, and these were all lactating nanny goats. Coincident with the onset of ocular disease, an outbreak of enteric yersiniosis due to *Y. pseudotuberculosis*, seen in all lactating groups, was diagnosed following post-mortem examination and bacterial culture of two affected goats. This was thought to be related to changes in diet introduced at that time. The owner was unable to provide accurate morbidity/mortality data for the enteric disease.

Clinical signs displayed by affected animals throughout the outbreak included sudden onset unilateral mucopurulent ocular discharge, blepharospasm, moderate to marked chemosis and conjunctival hyperaemia, mild to moderate corneal opacity and miosis. As the condition progressed there was moderate swelling of the eyelids and nodular proliferation of the palpebral conjunctiva, with similar nodular lesions at the medial canthus and extending down the face. Continuing corneal opacity and limbal/peripheral corneal neovascularisation was seen in some cases. There was ipsilateral mild to marked parotid and submandibular lymph node enlargement, occasionally with excoriation of the overlying skin and occasional lymph node abscessation. The affected goats remained bright, alert and continued to eat and they were non-pyrexia.

To investigate this problem, a lactating adult goat weighing 36 kg and in fair body condition was submitted for post-mortem examination to the Veterinary Laboratories Agency (VLA) at Sutton Bonington in March 2008. On gross examination there was severe proliferative conjunctivitis of the right eye characterized by nodular/granular thickening of the palpebral conjunctiva. A moderate quantity of dry yellow purulent exudate was adherent to the eyelids and the medial canthus of the right eye. The ipsilateral parotid and submandibular lymph nodes were moderately

enlarged, the former being approximately $3 \times 2 \times 1$ cm and the latter $2.5 \times 2 \times 0.5$ cm. The cut surface of both lymph nodes showed multifocal to coalescing 1–2 mm diameter punctate, and 0.5–1 cm long and radiating, pale cream foci often with a dark slightly depressed centre. The periphery of the lymph nodes was reddened and there was slight thickening of the lymph node capsule. Other findings in the carcase included mild fibrous pleuritis, fibrinous pericarditis and mummified fetal material within the uterus.

Samples of conjunctiva and submandibular lymph node were cultured on 5% sheep blood agar and MacConkey agar at 37°C for 48 h aerobically (in the presence of 5% CO₂ between 24 h and 48 h). A profuse growth of a *Yersinia* spp. was isolated from both sites and was identified as *Y. pseudotuberculosis* using API20E (bioMérieux, Lyon, France) and by demonstration of motility at 22°C but not at 37°C. No *Listeria* spp. were identified from the same tissues using *Listeria*-selective agar and broth (the latter subcultured to *Listeria*-selective agar at 24 h, 48 h and 7 days) incubated at 30°C for 24 h and 48 h. Denaturing gradient gel electrophoresis (DGGE) of 16S ribosomal DNA polymerase chain reaction (PCR) amplicons (Nicholas *et al.*, 2008) failed to detect any *Mycoplasma* spp. in conjunctival tissue.

The right eye, eyelids and parotid and submandibular lymph nodes were fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections (4 µm) were stained with haematoxylin and eosin (HE) and Gram stain. The palpebral conjunctiva showed extensive loss of normal architecture characterized by widespread erosion/ulceration with small islands of moderately hyperplastic epithelium containing small numbers of neutrophils and lymphocytes. The eroded/ulcerated surfaces were covered by a thin layer of viable and degenerate neutrophils, mucus and proteinaceous material. Within the lamina propria there was a marked lymphoplasmacytic, macrophage and neutrophilic infiltrate with moderate angiogenesis/neovascularisation. Multifocally, moderate to large bacterial colonies of gram-negative coccobacilli were present from the ulcerated surface to the deep lamina propria. These were surrounded by large numbers of viable and degenerate neutrophils (Figs. 1 and 2).

Both the parotid and submandibular lymph nodes contained multifocal to coalescing irregularly shaped large areas of moderately sized bacterial colonies (gram-negative coccobacilli) surrounded by large numbers of viable and degenerate neutrophils with associated necrotic cellular debris, and an outermost narrow infiltrate of macrophages. The intervening parenchyma contained large numbers of plasma cells, lymphocytes and fewer macrophages. Marked

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