Case report

*Listeria Monocytogenes*: a rare cause of endophthalmitis, a case report

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**A B S T R A C T**

*Listeria monocytogenes* is a known cause of gastroenteritis. Invasive disease can follow bacteremia causing meningocerephalitis, endocarditis and spontaneous miscarriages in immunocompromised patients and pregnant women respectively.

We present the first case in England of endogenous endophthalmitis caused by *L. monocytogenes* following acute gastroenteritis in an immunocompetent host. A 50-year-old South Asian female presented with acute painful unilateral visual loss occurring shortly after an episode of self-limiting gastroenteritis. On examination, the eye was very inflamed with a hypopyon uveitis. A vitreous biopsy confirmed growth of *L. monocytogenes* serotype 1/2a.

Diagnostic delay commonly occurs in endogenous endophthalmitis and exacerbates an already poor visual prognosis. *Listeria* spp. must be considered in ocular inflammation following gastroenteritis. The intraocular inflammation subsided but surgical intervention was required to remove vitreous debris and improve visual acuity.

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**Introduction**

*Listeria monocytogenes* is zoonotic infection caused by a small gram positive bacillus found in ruminants, infection occurs due to consumption of unpasteurized or packaged milk products and meat, contaminated vegetables and exposure to farms. It commonly causes mild febrile gastroenteritis, although can present without gastrointestinal symptoms, and more rarely invasive disease in immunocompromised, elderly or pregnant individuals; including severe meningitis, endocarditis and bone and joint infections [1]. Only a few case reports of *Listeria* endophthalmitis have been published. It is recognised as an infrequent complication of bacteremia or endocarditis with embolic disease [2]. Endogenous sources cause between 2 and 15% of all endophthalmitis, the majority being exogenous secondary to intraocular surgery, intravitreal therapy or penetrating trauma [2]. There are limited case series however *Listeria* spp. has been quoted as causing 4% of endogenous endophthalmitis [2,3]. We present the first case of *Listeria monocytogenes* causing endophthalmitis in England.

**Patient**

A 50-year-old South Asian origin immunocompetent female with no significant past medical history presented with symptoms of profuse diarrhea with abdominal cramps and high fever following consumption of meatballs and smoked salmon in the previous week. During the second day of symptoms she developed acute painful visual loss in her left eye. She had, retro-orbital pain and headache with conjunctival injection and watering. She was seen at an emergency eye clinic and uveitis was diagnosed. She was treated with steroid eye drops for two weeks. Vision initially improved then subsequently deteriorated despite a trial of oral prednisolone 30 mg OD for a further two weeks.

A month after her initial illness there was no improvement in her ocular symptoms. Her vision was counting fingers (CF) in her left eye and normal acuity in her right eye. Her left eye had a severe anterior chamber inflammatory activity, copious flare, keratic precipitates and hypopyon. The posterior fundus was not visible...
due to vitritis and debris, although the retina peripherally was seen to be flat and attached with indirect ophthalmoscopy. A diagnosis of endophthalmitis was made. A vitreous aspiration was performed and she was given an intravitreal injection of vancomycin and foscarnet. She was commenced on valacyclovir 2 g tds, trimethoprim/sulfamethoxazole (SXT) 960 mg bd for 2 weeks and prednisolone 80 mg od. The vitreous fluid had numerous polymorphs on microscopy, gram stain was negative. *L. monocytogenes* was cultured from vitreous humour [1]. Further characterisation at the Gastrointestinal Bacteria Reference Unit, Colindale confirmed *L. monocytogenes* serotype 1/2a, which was fully sensitive to amoxicillin, gentamicin, SXT and vancomycin. She was admitted to hospital for further management. Surveillance blood cultures were negative, blood tests showed CRP <0.3 with normal CBC, LFTs and renal function. No immunodeficiency was identified. Transthoracic ECHO was normal and there was no evidence of any other metastatic infection.

**Treatment**

Treatment was altered based on the microbiological results, oral steroids were stopped immediately and she was commenced on intravenous (IV) amoxicillin 2 g 4 hourly for 6 weeks, and IV gentamicin 80 mg 8 hourly as a synergistic agent for the first 2 weeks followed by 2 weeks of oral SXT. Her eye pain and watering settled as did the intraocular inflammation. Following the intravitreal antimicrobial therapy and 6 weeks of intravenous treatment all the intraocular inflammatory signs decreased. Her visual acuity remained poor due to disorganization of the vitreous following the severe infective/inflammatory episode and a vitrectomy was carried out 5 months after presentation. Following this her vision improved to 6/9, improved but with a residual impaired acuity. The vitreous removed had no bacterial growth on culture.

**Discussion**

Endogenous endophthalmitis in this case was caused by transient bacteremia associated with severe *L. monocytogenes* gastroenteritis. The strain isolated was serotype 1/2a is the most common serotype in England, causing 43% of invasive disease in 2014 [4]. Our patient had a delay in an accurate diagnosis, she was initially diagnosed with uveitis. This is common in patients with endogenous endophthalmitis where a diagnostic delay occurs in 26–29% of patients [5]. This delay in diagnosis compounds an already poor visual outcome due to intraocular damage occurring from the combination of infection and inflammation [5]. Intracocular antimicrobial therapy is required to achieve sufficiently high antimicrobial concentrations in the eye due to the difficulties of adequate penetration of these agents across the blood retinal barrier when given systemically. Our patient exemplifies the difficulties in diagnosis, treatment and the guarded visual outcomes. *L. monocytogenes* is a neurotropic bacterium, first line effective treatment for infections involving central nervous system includes a prolonged course of 6–8 weeks of parental high dose amoxicillin/benzyl penicillin plus synergistic gentamicin [1].

Treatment is usually not indicated for self-limiting gastroenteritis except in pregnancy, immunocompromised patients or those >50 years where 3–5 days of amoxicillin is recommended [1]. SXT has excellent intraocular and intracellular penetration and therefore was added as a follow-on treatment to achieve microbiological clearance. To our knowledge this is the first case of *Listeria monocytogenes* causing endophthalmitis in England.

**Conflict of interests**

No competing interests declared.

**References**


