• Letter to the Editor •

Endophthalmitis caused by *Streptococcus dysgalactiae* subsp. *dysgalactiae* after cataract surgery: an unusual case report

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Dear Editor,

I am writing this letter to present an unusual case of *Streptococcus dysgalactiae* subsp. *dysgalactiae* endophthalmitis.

Streptococcus dysgalactiae is a Gram-positive bacterium within the *Streptococcus* genus, and also classified as a β-hemolytic streptococcus. It primarily includes two subspecies: *Streptococcus dysgalactiae* subsp. *dysgalactiae* and *Streptococcus dysgalactiae* subsp. *equisimilis*. *Streptococcus dysgalactiae* is carried by approximately 30% of healthy individuals[1], characterized as an opportunistic pathogen that can cause disease when host immunity is compromised. In humans, it typically causes skin and soft tissue infections, while invasive infections can lead to suppurative arthritis, pleuritis, meningitis, endocarditis and septicemia[2-3]. Severe or even life-threatening infections most commonly occur in elderly patients or those with serious underlying diseases[4]. *Streptococcus dysgalactiae* subsp. *dysgalactiae* typically infects animals, but has rare cases for human infections[5]. Ocular infections in humans are even rarer. A thorough review of the available literature has not reported any previous instances of endophthalmitis caused specifically by *Streptococcus dysgalactiae* subsp. *dysgalactiae*.

Here we report a clinical picture and treatment outcomes of endophthalmitis caused by *Streptococcus dysgalactiae* subsp. *dysgalactiae* in a patient who received combined systemic and local anti-infective therapy. The infection was ultimately brought under control, but the visual prognosis is extremely poor due to retinal damage.

Ethical Approval As this manuscript is a single-case report describing routine clinical management, it does not constitute biomedical research involving human subjects as defined by our institutional review board. Therefore, formal ethical approval was exempted.

Case Presentation An 81-year-old male presented with decreased vision, redness and pain in his right eye for 10h. He had undergone uneventful phacoemulsification with intraocular lens (IOL) implantation in the right eye at a local hospital 2d previously. At the very beginning of symptom onset, he was admitted to the local hospital, where the intraocular pressure (IOP) was 50 mm Hg. A slit lamp examination showed a severe anterior chamber reaction with revealed exudate, and B-scan examination revealed no significant abnormality noted. Therefore, the patient was referred to our hospital for further treatment. The patient had hypertension for 20y, coronary heart disease for 20y with a history of pacemaker implantation and underwent cataract surgery in the left eye 7y ago. He denies any other history of systemic disease. A comprehensive ophthalmic examination revealed uncorrected visual acuity (UCVA) as hand motion (HM), IOP as 48 mm Hg (1 mm Hg= 0.133 kPa), mixed conjunctival hyperemia with significant chemosis, diffuse corneal haze with circumferential gray-white infiltrates at the corneal limbus (Figure 1A, arrow). Cells 3+, flare 3+, and significant fibrinous exudate was observed in the anterior chamber at the pupillary area. With localized posterior synechiae, the IOL is faintly visible but appears well-positioned. The fundus was not visible. He underwent urgent subconjunctival injection of dexamethasone 5 mg. Topical eye drop therapy consisted of: levofloxacin every hour,

tobramycin-dexamethasone every 2h, and timolol twice daily. Additionally, anterior chamber paracenctesis was performed to lower the IOP. Four hours later, the patient's symptoms and signs worsened. Ophthalmic examination of right eye at this time showed that visual acuity (VA) was light perception (LP) questionable and IOP was 40 mm Hg. Corneal edema and exudation in anterior chamber increased significantly, where an approximately 1 mm hypopyon was observed in the inferior anterior chamber. Moreover, an exudative membrane was visible over the iris and the anterior surface of the IOL. The iris stromal details were blurred. B-scan showed high echoes in the vitreous cavity of the right eye (Figure 1B, arrow). Laboratory results from the blood culture indicated a state of inflammation: neutrophil count 8.43×10^9/L, neutrophil percentage 91.9%, high-sensitivity C-reactive protein 6.88 mg/L. Upon admission, the patient underwent vitreous biopsy and pars plana vitrectomy (PPV) through emergency surgical treatment. Following the removal of hypopyon in the anterior chamber and exudative membrane over the iris and IOL, copious yellow purulent secretion was observed within the capsular bag. The IOL along with the capsular bag complex was subsequently extracted. The vitreous cavity was filled with extensive purulent opacities, with purulent exudative membranes covering the posterior pole retinal surface, involving both the optic disc and the macula. Approximately 1 mL of vitreous fluid was aspirated and sent for bacterial/fungal culture and drug sensitivity test. After removing pus and purulent exudative membranes as possible, approximately 5 mL of silicone oil was injected into the vitreous cavity. Vancomycin (10 mg/mL) 0.05 mL, ceftazidime (20 mg/mL) 0.05 mL, and dexamethasone 250 mg/0.05 mL were intravitreally injected at the end of the surgery.

Postoperatively, the patient was instructed to maintain a prone position with antimicrobial and anti-inflammatory therapy topically and systemically. Topical levofloxacin and tobramycin-dexamethasone eyedrops were prescribed at a frequency of every 2h. To achieve high local drug concentrations, a daily deep fornix injection of ceftazidime (250 mg/mL) 0.5 mL, vancomycin (10 mg/mL) 0.5 mL, and dexamethasone (3 mg) 0.6 mL was scheduled weekly. Systemic treatment included intravenous ceftazidime (1 g twice a day for 5d) and vancomycin (500 mg daily for 3d) to mitigate the risk of systemic spread and control severe orbital infection. After 3d, *Streptococcus dysgalactiae* subsp. *dysgalactiae* was identified by bacterial culture. Drug test indicated sensitivity to a variety of antibiotics, including levofloxacin, vancomycin, and cefotaxime (Table 1), which were susceptible to the received therapeutic regimen. With combined topical and systemic treatment, the inflammation was controlled effectively. Two weeks postoperatively, the patient

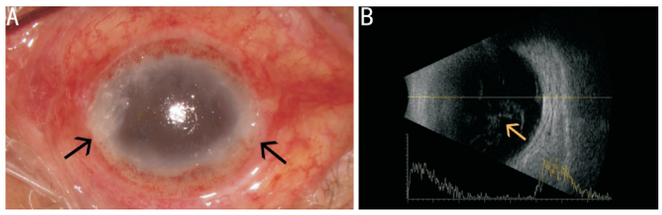

Figure 1 Anterior segment photograph and ocular B-scan at presentation A: Diffuse corneal edema and dense infiltrate in the peripheral cornea (arrow); B: High echoic shadows are visible in the vitreous cavity (arrow).

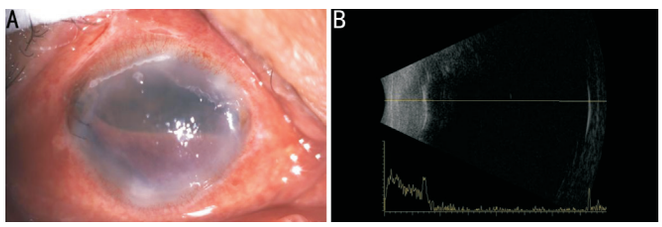

Figure 2 Anterior segment photograph and ocular B-scan on postoperative day 8 A: Corneal edema decreased with clumpy pigmentation in the anterior chamber; B: B-scan showed silicone oil filling in the vitreous cavity.

Table 1 Culture and drug sensitivity results

No.	Antibiotic (route)	MIC (μg/mL)	KB (mm)	Breakpoint	Interpretation (S/I/R)	Group
1	Tetracycline		7	18–23	R	
2	Penicillin G	0.064		0.125–4	S	A
3	Ampicillin		24	13–17	S	A
4	Clindamycin		7	15–19	R	A
5	Erythromycin		7	15–21	R	A
6	Cefotaxime		28	25–28	S	B
7	Ceftriaxone		25	24–27	I	B
8	Cefepime		25	21–24	S	B
9	Vancomycin		21	≥17	S	B
10	Levofloxacin		17	13–17	S	C
11	Linezolid		21	≥21	S	C

Culture results: One bacterial species was isolated and identified: *streptococcus dysgalactiae* subsp. *dysgalactiae*. *Streptococcus dysgalactiae* subsp. *dysgalactiae* growth condition: +++. MIC: Minimum inhibitory concentration; KB: Kirby–Bauer; S: Susceptible; I: Intermediate; R: Resistant.

was asymptomatic for pain and the ophthalmic examination of the right eye showed VA as HM/10 cm and IOP as 8.0 mm Hg, with reduced conjunctival congestion and edema. Corneal infiltrates significantly improved and corneal edema decreased with few folds in Descemet's membrane. The anterior chamber contained hemorrhagic opacities in the lower half of its volume and the fundus could not be visualized clearly. B-scan showed silicone oil filling in the right eye (Figure 2). Inflammation was effectively controlled.

DISCUSSION

With the advancement in surgical techniques and the use of povidone-iodine to disinfect the conjunctival sac

preoperatively, the incidence of endophthalmitis after cataract surgery has significantly decreased[6]. Concurrently, as the widely adoption and improvement of PPV, along with enhanced surgeons' awareness, the majority of patients who suffered post-cataract endophthalmitis are able to preserve useful vision through timely intervention nowadays. However, severe visual impairment or even evisceration still occur intermittently due to high virulence or delayed treatments. Previous retrospective studies have indicated that, with similar rates of virulent pathogens after endophthalmitis, patients presenting early (<5d after cataract surgery) had better visual outcomes compared to those presenting late (≥5d) after intravitreal injection with or without PPV. Specifically, 90% of early-presenting patients attained a VA of ≥20/40, compared to 68% in the late group[7]. This case illustrates a particularly fulminant case of post-cataract endophthalmitis, characterized by an exceptionally rapid onset within 28h and progression to severe intraocular damage within 24h. While the inflammation was controlled successfully with aggressive intervention, irreversible visual impairment resulted. Although postoperative best corrected visual acuity was evaluated, no significant improvement was observed relative to the baseline. As silicone oil was instilled into the vitreous cavity during the surgery, the observed anterior chamber pigmentation was considered to be associated with iris depigmentation. Preoperative corneal edema was severe, and pronounced inflammatory reactions were observed in the anterior chamber postoperatively, precluding clear visualization of the fundus.

In our case, this patient was elderly and had a history of hypertension, coronary heart disease, and pacemaker implantation, all of which are predisposing factor to this bacterium. Currently, endophthalmitis caused by *Streptococcus dysgalactiae* is rarely reported. Park *et al*[8] reported a case of endophthalmitis caused by *Streptococcus dysgalactiae* subsp. *dysgalactiae.* This patient developed ocular pain and sudden vision loss on the second day after cataract surgery and was transferred to a tertiary hospital on the fourth day. Combined treatments including PPV, anterior chamber irrigation, IOL extraction, and intravitreal antibiotic injection were given, followed by systemic and local antibiotic therapy. However, corneal phthisis was noted, indicating a poor prognosis on postoperative day 25. Jiang *et al*[9] reported a case of endophthalmitis caused by *Streptococcus dysgalactiae* subsp. *equisimilis* after cataract surgery, which occurred on the first postoperative day. Immediate intervention included PPV and intravitreal injection of vancomycin (10 mg/mL) 0.1 mL and ceftazidime (20 mg/mL) 0.1 mL were performed, along with intravenous piperacillin-tazobactam 4.5 g every 8h+levofloxacin 0.5 g daily and topical 0.02 mg/mL vancomycin+0.04 mg/mL ceftazidime eye drops every hour. The inflammation was gradually controlled, but the final visual outcome remained poor. Furthermore, Nakata *et al*[10] reported a case of infective endocarditis caused by *Streptococcus dysgalactiae* that initially presented as endogenous endophthalmitis. Given the history of pacemaker implantation, a transthoracic echocardiogram was performed, which revealed no signs of associated infection.

Based on the existing reports and present case, we observe that endophthalmitis caused by *Streptococcus dysgalactiae* typically presents early post IOP operatively, characterized by acute onset, rapid progression, and has significant destructive effects on intraocular tissues. Elevation of IOP may be observed in the initial stages. Early manifestations must be differentiated from toxic anterior segment syndrome (TASS), which requiring close monitoring of disease progression for prompt diagnosis and timely treatment. Pathogen identification is paramount throughout the diagnostic and therapeutic process of infectious endophthalmitis. Before pathogen identification, broad-spectrum antibiotic therapy (topical and/or systemic) can be initiated. Once the causative organism is identified, sensitive antimicrobial therapy should be instituted promptly based on drug susceptibility results[11-12]. A review of this case and the literature indicates that *Streptococcus dysgalactiae* is generally susceptible to most antibiotics commonly used for postoperative infectious endophthalmitis[13]. Although susceptibility testing for ceftazidime was not specifically performed in this case, the empirical selection of this agent was strictly guided by Chinese expert consensus on the diagnosis and management of infectious endophthalmitis after ophthalmic surgery (2022)[14]. According to these national guidelines, intravitreal vancomycin combined with ceftazidime remains the first-line therapeutic regimen for acute bacterial endophthalmitis. Importantly, the consensus explicitly advises against the use of other third- or fourth-generation cephalosporins, such as ceftriaxone, cefotaxime, and cefepime for intraocular administration due to their suboptimal pharmacokinetic profiles and poor vitreous penetration. Therefore, the choice to continue ceftazidime therapy was based on standardized protocols designed to ensure broad-spectrum coverage against common endophthalmitis pathogens.

Timely PPV is critical for patients with rapidly progressing disease and severe ocular signs, while it also should be performed decisively in cases where infection remains uncontrolled after intravitreal antibiotic injection or if vision continues to decline rapidly and ocular signs worsen persistently. This intervention directly reduces the intraocular pathogen load and inflammatory mediators, aimed to control infection and preserve vision[14]. Expert consensus indicates that VA of LP at 1 m is one of the indications for PPV in acute bacterial endophthalmitis following ophthalmic surgery[14]. In

our case, the patient's VA in the right eye was HM and the IOP was 48 mm Hg upon admission, with no obvious hypopyon in the anterior chamber. Given the excessively high IOP, the risk of intraoperative complications was deemed unacceptably high. Based on the guidance provided in the consensus, after initiating intensive topical antimicrobial therapy, we first performed anterior chamber paracentesis to reduce IOP, and closely monitored the affected eye at intervals of every 2–4h. Four hours later, the patient's VA progressively declined to LP and posterior segment involvement worsened. Therefore, the patient underwent emergency PPV, IOL extraction, silicone oil injection combined with intravitreal administration of vancomycin, ceftazidime, and dexamethasone within 24h of symptom onset. Postoperative treatment included systemic intravenous administration of ceftazidime and vancomycin, complemented by daily subconjunctival injections of the same antibiotics, ultimately controlling the infection.

In conclusion, endophthalmitis caused by *Streptococcus dysgalactiae* subsp. *dysgalactiae* after cataract surgery is clinically rare. Its early clinical manifestations are atypical yet progress rapidly, typically resulting in a poor visual prognosis. Even with early surgical intervention controlling the infection, recovery of useful functional vision is still challenging. Therefore, special attention is warranted for patients with established universal high-risk factors for endophthalmitis from various pathogens, such as advanced age, comorbid systemic conditions, or a history of previous surgery in clinical practice. Every step of the perioperative process must be rigorously optimized for effective infection prophylaxis, which includes the use of preoperative antibiotic eye drops, intraoperative povidone-iodine, intracameral antibiotics at the end of surgery, and timely postoperative follow-up. Concurrently, maintaining a high index of suspicion to enable early diagnosis and prompt initiation of treatment is paramount.

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Conflicts of Interest: Tian DD, None; **Hao LH,** None; **Wu CR,** None; **Pei C,** None.

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