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CASE REPORT

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# Endogenous endophthalmitis due to *Serratia marcescens* secondary to late-onset empyema Post-Cardiac surgery in an End-Stage renal disease patient on peritoneal dialysis

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## Abstract

Endogenous bacterial endophthalmitis results from bacterial seeding of the eye during bacteremia. A diagnosis of endogenous bacterial endophthalmitis requires clinical findings such as vitritis or hypopyon along with positive blood cultures. *Serratia marcescens* is the second most common pathogen causing hospital-acquired ocular infections. This report describes a case of endogenous bacterial endophthalmitis caused *by S. marcescens* in an older adult with end-stage renal disease (ESRD) on peritoneal dialysis, who had late-onset pleural empyema secondary to coronary artery bypass grafting (CABG). A 61-year-old gentleman presented with a two-day history of cloudy vision, black floaters, pain, swelling, and gradual vision loss in his right eye. There was no history of trauma, ocular surgeries, or previous similar episodes. He had myocardial infarction treated with CABG 3 months back. Examination showed a 3 mm hypopyon in the anterior chamber. He had classic signs of endophthalmitis with positive blood cultures for *S. marcescens.* He was treated with high-dose intravenous meropenem and intravitreal ceftazidime without vitrectomy. Endophthalmitis progressed to complete vision loss in his right eye, requiring evisceration. Endophthalmitis caused by *S. marcescens* is rare, but long-term outcomes can be severe, causing complete vision loss in about 60% of the patients. It is usually hospital-acquired, and the source can be late-onset empyema several months after cardiac surgery, in an immunocompromised patient. Systemic antibiotics should be supplemented with intravitreal agents with or without pars plana vitrectomy.

**KEYWORDS**

carbapenems, endogenous endophthalmitis, gram-negative, intra-ocular, *Serratia*

## 1 | INTRODUCTION

*Serratia* species are gram-negative bacilli of the Enterobacteriaceae group, although they are not a common component of healthy human fecal flora.[1] The genus *Serratia* consists of at least 20 species, of which *S. marcescens* is the main human pathogen. Automated bacterial identification systems and matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) will reliably identify *S. marcescens*. They are usually lactose non-fermenters.[1] Hospital-onset infections due to *Serratia* species are mostly associated with outbreaks linked to environment or medical exposure. The prevalence of *Serratia* as a primary pathogen in bacteremia and pneumonia are 3.4% and 5.3% in Europe and 1.6% and 3% in North America.[2,3] *Serratia* was found to cause 7% of the urinary tract infections in a study from Japan.[4] Though the presence of an invasive device increases the risk of Serratia infections, it is not a predominant pathogen causing device-related infections.[5] *S. marcescens* is known to cause wound infections, urinary tract infections,[4] respiratory tract infections,[6] bacteremia, endocarditis,[7] central nervous system, and ocular infections. *S. marcescens* is the second most common pathogen *after Pseudomonas aeruginosa* causing ocular infections.[8] It usually causes conjunctivitis, keratoconjunctivitis, corneal ulcers, and keratitis. Endophthalmitis caused by *S. marcescens* is rare, but long-term outcomes can be severe, causing complete vision loss in about 60% of the patients.[9] Most cases of *S. marcescens* ocular infections are nosocomial and occur in neonates and children, post-traumatic ocular infections, and in contact lens wearers.[1,10] Endogenous bacterial endophthalmitis results from bacterial seeding of the eye during bacteremia. Less than 0.1% of the cases of bacteremia in the United States are complicated by endophthalmitis.[11] Methicillin-resistant *Staphylococcus aureus* (MRSA) is the major cause of endogenous bacterial endophthalmitis, and endocarditis is the commonest source in the US.[12,13] However, in Asian countries, *Klebsiella pneumonia* endophthalmitis associated with liver abscess accounts for 60% of the cases.[14] A diagnosis of endogenous bacterial endophthalmitis requires clinical findings consistent with endophthalmitis, such as vitritis or hypopyon, along with positive blood cultures.[13–15] Positive cultures from vitreous or aqueous fluids in patients presenting with endophthalmitis is also diagnostic. Recent eye trauma or surgery should be ruled out.[13]

## 2 | CASE REPORT

A 61-year-old South Asian gentleman presented to our emergency department with a two-day history of cloudy vision and black floaters, followed by pain, swelling, and gradual vision loss in his right eye. There was no history of trauma, ocular surgeries, or previous similar episodes. A review of other systems was unremarkable. He was known to have hypertension and type 2 diabetes mellitus complicated by diabetic retinopathy and nephropathy that progressed to end-stage renal disease (ESRD) on continuous ambulatory peritoneal dialysis (CAPD) four times a day. His medications included basal-bolus insulin for blood sugar control, amlodipine, metoprolol for blood pressure control, and atorvastatin for dyslipidemia. He was known to have atrial fibrillation on warfarin, aspirin, and a recent myocardial infarction treated with coronary artery bypass graft surgery (CABG) 3 months back. The left internal mammary artery (LIMA) was used for grafting. Examination of his right eye showed normal intraocular pressure, impaired visual acuity (only perception of light), ptosis, redness, conjunctival injection, and a 3 mm hypopyon in the anterior chamber (Figure 1). The fundus of the right eye could not be viewed. His left eye was normal except for features of proliferative diabetic retinopathy on the fundus. B-scan ocular ultrasonography showed increased choroidal thickness and vitreous membranes with a small traction area on the retina of the right eye. Examination of the thorax showed stony dullness and reduced tactile vocal fremitus of the right lung base with decreased air entry in the right lower zone. The abdomen was soft, and there were no clinical signs of peritonitis. Blood tests on admission were significant for mild leukocytosis (12.7×10^3/μL), thrombocytopenia (110×10^3/μL), microcytic, hypochromic anemia (hemoglobin - 7.4 gm/dL), elevated C- reactive protein (149.7 mg/L) and procalcitonin (1.6 ng/mL). Fourth-generation

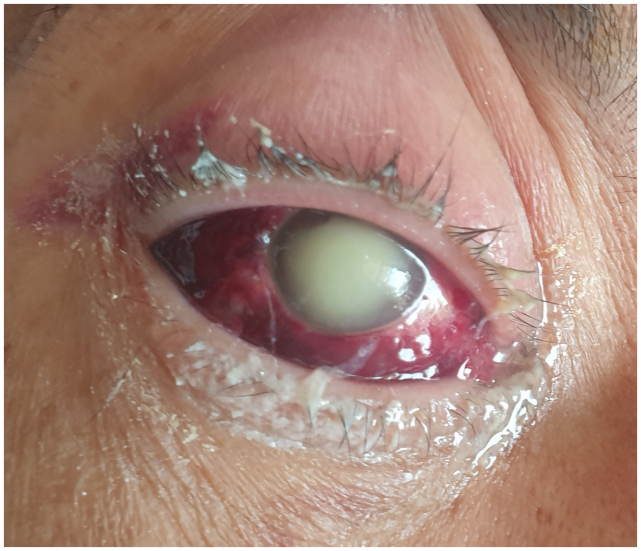

**FIGURE 1** Photograph of right eye showing redness, discharge, and hypopyon.

antigen–antibody test for HIV, treponema pallidum antibody, and rapid plasma reagent (RPR) tests for syphilis were negative. Blood culture collected on the day of admission grew extended-spectrum beta-lactamase (ESBL) producing *Serratia marcescens* in aerobic and anaerobic bottles. As per Clinical and Laboratory Standards Institute (CLSI) break-point values, the organism was sensitive to carbapenems, aminoglycosides, and trimethoprim-sulfamethoxazole. The complete sensitivity panel was available by Day 4. Evaluation of peritoneal fluid done on day 2 showed only 80 white cells and no growth on the culture. Peritoneal dialysis-related peritonitis was hence ruled out. A diagnosis of endogenous endophthalmitis of the right eye was made on day 3, based on ophthalmologic examination findings and the presence of positive blood culture. On the same day, he was started empirically on a double dose of intravenous meropenem (1 g instead of 500 mg daily, adjusted for renal function) to ensure ocular penetration. He was also given intravitreal injection of ceftazidime 2 mg and vancomycin 1 mg twice over 48 h (day 3 and day 5) and maintained on hourly ceftazidime eye drops. The ophthalmologists could not obtain a vitreous sample for culture or PCR, as the patient's general condition was precarious and not fit for emergency pars-plana vitrectomy. Contrast-enhanced computed tomography (CECT) of his thorax and abdomen showed bilateral pleural effusion with enhancement on the right side suggestive of empyema (Figure 2). Ultrasound of the right pleura showed right-sided echogenic pleural effusion with septations and atelectasis in keeping with empyema (Figure 3). A focused trans-thoracic echocardiogram ruled out the possibility of any valve vegetations. Intravenous high-dose meropenem was continued. Repeated blood cultures were negative on day 6. Ultrasound-guided aspiration of the pleural fluid on the right side was done on day 7; however, the biochemical and microbiological evaluation of the sample did not fit the criteria for empyema, probably due to the use of high dose IV meropenem for the preceding week. Pleural fluid glucose was 5.8 mmol/L (104.4 mg/dL), pH 7.3, LDH 107 U/L, and protein 32 g/L. At the same time, total serum protein was 59 g/L, and serum LDH was 167 U/L. Two points in the Light's Criteria were met (pleural fluid protein/serum protein >0.5; pleural fluid LDH/serum LDH >0.6), and it was highly suggestive of exudative pleural effusion. However, there were no features of empyema biochemically or microbiologically, as the pleural fluid glucose was more than 60 mg/dL, pH more than 7.2, LDH not more than three times the upper limit of normal and gram stain, and culture were negative. The diagnosis of empyema was based on loculations in the pleural cavity (Figures 2 and 3) and pus coming out during aspiration. Microbiological and biochemical parameters were negative for empyema, possibly because the patient was on high-dose meropenem for 1 week before the pleural aspiration was done. Surgical thoracoscopy or pleural decortication was not done as the patient was hemodynamically unstable. Despite aggressive efforts to treat the patient, the condition of his right

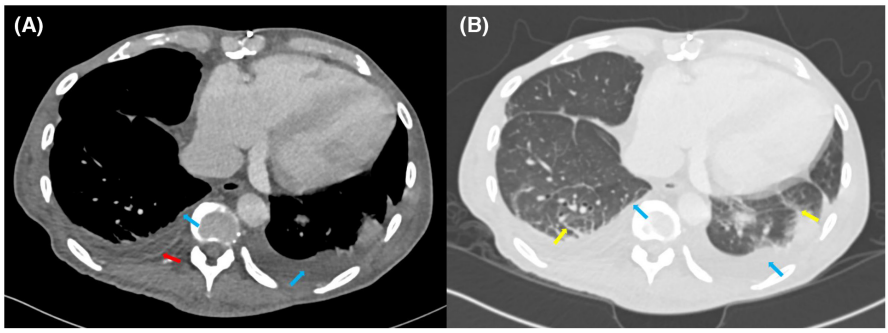


**FIGURE 2** CT thorax with IV contrast (A) mediastinal and (B) lung windows, showing bilateral pleural effusion (Blue arrows) and right-sided pleural enhancement suggestive of empyema (red arrow). Bibasilar atelectatic changes were also noted (yellow arrows).

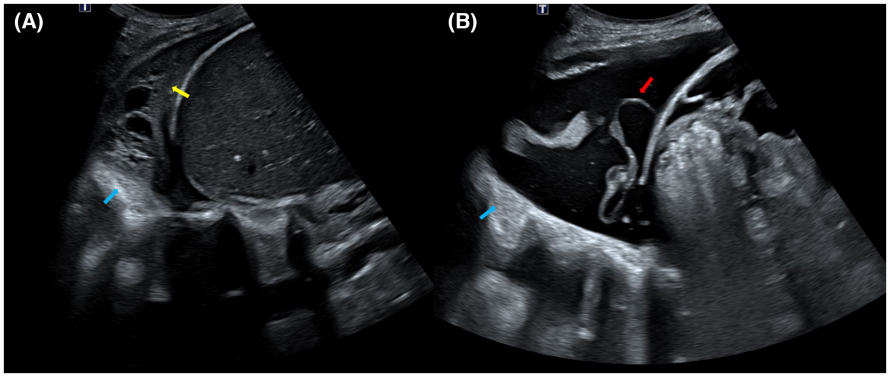


**FIGURE 3** Ultrasound right (A) and left (B) thorax and pleural cavity showing right-sided echogenic pleural effusion with septations (yellow arrow) suggestive of empyema, left sided pleural effusion with septations (red arrow) and bilateral atelectasis (blue arrows).

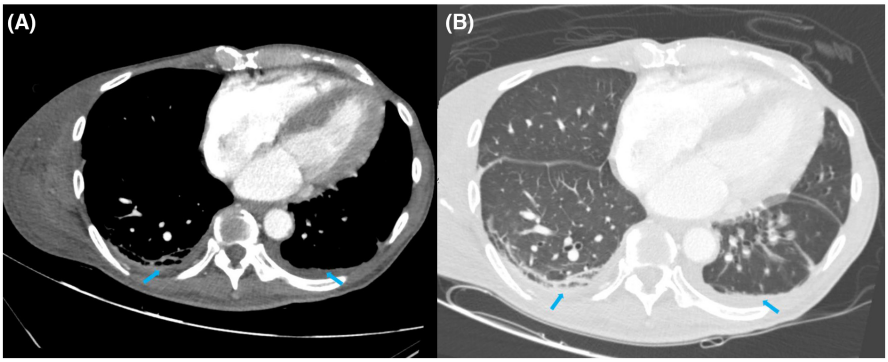


**FIGURE 4** CT thorax with IV contrast (A) mediastinal and (B) lung windows demonstrating significant regression of bilateral pleural effusion (blue arrows). There is complete resolution of the previously seen right-sided empyema.

eye worsened, and by day 10 of admission, 90% of the anterior chamber was filled with hypopyon, and there was diffuse corneal edema and subconjunctival hemorrhage. Vision of the right eye could not be saved. Evisceration was done on day 14. He was treated with IV meropenem 1 g daily (high dose for CAPD patients) for 4 weeks. A CT thorax after 2 weeks of treatment showed regression of the pleural effusion (Figure 4). However, the patient's renal and liver functions worsened progressively, and he passed away due to cardiac arrest, multi-organ failure, and dyselectrolytemia on day 32 of hospitalization.

## 3 | DISCUSSION

Endogenous endophthalmitis is an uncommon but potentially devastating intraocular infection in which pathogens reach the eye via the bloodstream.[13] Our patient had classic symptoms of endophthalmitis with positive blood cultures for *Serratia marcescens*, which is sufficient to diagnose endogenous bacterial endophthalmitis. Evaluation of systems revealed no possible source other than the lungs. He had radiologically diagnosed pleural empyema. According to a meta-analysis by Zetting D et al.,[16] CT findings of pleural enhancement, thickening, loculation, fat thickening, and fat stranding had a specificity of more than 90% for a diagnosis of pleural empyema. Many previous studies have shown that pleuro-pulmonary complications can occur after CABG, more commonly when LIMA is used for grafting.[17–20] In a prospective case series of 389 patients evaluated 28 days after cardiac surgery, two-thirds of patients had a pleural effusion after a CABG.[17] In contrast to early pleural effusions, which are generally small, 10 percent of late pleural effusions are large (i.e., an effusion that occupies more than 25 percent of the hemithorax), and 5 percent of them had features of empyema.[17] So, it can be hypothesized that a minor pleural injury during the CABG lead to the seeding of Serratia, which is predominantly a hospital-acquired organism, into the pleural cavity of our patient. This most likely lead to late-onset empyema, bacteremia, and endophthalmitis.

50% of the patients with endogenous bacterial endophthalmitis complain of only eye pain and decreased vision. Fewer than 20 percent have a fever on presentation, and 40 percent have an unremarkable general physical examination.[13,15,21] Among ocular infections, uveitis and keratitis are caused mainly by viruses and parasites. Endophthalmitis is generally attributed to bacterial or fungal seeding of the eye, with vitreous and/or aqueous humor involvement, during bacteremia.[13,22] The etiological agent causing endogenous bacterial endophthalmitis varies with the patient population and geographic location. Streptococci (*S. milleri group, group A, group B, and S. pneumoniae*) cause 30 to 50% of cases in North America and Europe, while gram-negative bacilli account for only 30% of the cases.[21,25] In contrast, most cases in Asian countries are caused by gram-negative bacilli, especially *K. pneumonia*.[14,23] In 75% of the cases, the causative agent was identified from blood, CSF, urine, or intra- ocular samples.[24] *Serratia spp*. is intrinsically resistant to ampicillin, amoxicillin, ampicillin-sulbactam, amoxicillin-clavulanate, narrow-spectrum cephalosporins, nitrofurantoin, tetracycline, macrolides, and colistin.[1] They are usually susceptible to third- and fourth-generation cephalosporins, carbapenems, fluoroquinolones, aminoglycosides, trimethoprim-sulfamethoxazole, piperacillin-tazobactam, and aztreonam.[25] Inducible chromosomal broad-spectrum beta-lactam resistance had been described in literature after exposure to certain antibiotics. Common suggested mechanisms include both AmpC beta-lactamase production and the production of extended-spectrum beta-lactamases (ESBLs) or carbapenemases.[26] It is challenging to treat infections caused by *Serratia* since antibiotic options are limited for multidrug-resistant isolates. Therefore, it is crucial to consider the potential for emergent AmpC, beta-lactamase-mediated resistance to third-generation cephalosporins when selecting initial treatment for infections in sites with impaired drug penetration like endophthalmitis. Previous studies suggested that ceftazidime-avibactam, meropenem-tazobactam, imipenem-relebactam, plazomicin, cefiderocol, and eravacycline are all appropriate treatment options to target

**TABLE 1** Name, dose, and routes of antibiotics that have managed to achieve intravitreal concentration more than the MICs of wild-type pathogens covered by the specific antibiotic agent.[28]

| **Vancomycin** | **15 mg/kg** | **IV** |
|---|---|---|
| Cefazolin | 50 mg/kg | IV |
| Meropenem | 2 g | IV |
| Rifampicin | 150 mg/300 mg/600 mg | PO |
| Linezolid | 600 mg | PO/IV |
| Daptomycin | 10 mg/kg | IV |
| Moxifloxacin | 400 mg | PO |

multidrug-resistant gram-negative organisms, particularly carbapenem-resistant Enterobacteriaceae.[5–7] Several factors might guide the selection between these agents, including the availability of resistance testing, source of infection, and risk of side effects. Empiric therapy is usually selected based on institutional antibiogram data; carbapenems are generally considered the treatment of choice for most multidrug-resistant isolates.[21,26]

Penetration of systemic medications into the posterior eye segment is limited due to the blood-retinal barrier. For instance, blood-retinal barriers are poorly penetrated by intravenously administered aminoglycosides, and they do not achieve therapeutic intraocular concentrations in the vitreous cavity.[27] Table 1 summarizes the name, dose and routes of antibiotics that have managed to achieve intravitreal concentration more than the MICs of wild-type pathogens covered by the specific antibiotic agent. The table is adapted from a review article by L. Brockhaus et al.[28] Systemic antibiotics alone will not effectively treat endophthalmitis. Optimum treatment consists of intravitreal antibiotics such as ceftazidime or an aminoglycoside with systemic antibiotics that cross the blood-eye barrier.[28] Benefits of early vitrectomy (within 7 days of symptom onset) and systemic and intravitreal antibiotics have been established in exogenous (post-surgical/ post-injection/ post-traumatic) endophthalmitis. Patients with exogenous endophthalmitis may gain increased visual benefit when surgery is performed within 7 days.[29–32] However, there are only limited data about the benefit of vitrectomy in endogenous endophthalmitis. In a study by Negretti et al.,[32] among six patients with endogenous endophthalmitis, three (50%) had an improvement in visual acuity (VA), and two (33.3%) had unchanged VA following vitrectomy. 16.7% of patients had VA better than 20/40, 66.7% of patients had VA worse than 5/200, and 16.7% of patients had loss of eye. At the same time, complications such as retinal detachment (24.2%), macular hole (3%), hypotony (6%), suprachoroidal hemorrhage (3%), and enucleation/ evisceration (6%) were noted post-vitrectomy.[32] In our patient, a panel of expert ophthalmologists decided not to do a vitrectomy after considering the risks and benefits.

## 4 | CONCLUSION

Endogenous endophthalmitis due to *Serratia marcescens* is extremely rare and often associated with poor ocular prognosis. It should be suspected when an immunocompromised patient with a history of recent thoracic or cardiac surgery or hospitalization presents with ocular pain or deterioration of visual acuity. Ours is a rare case where minor pleural trauma during CABG leads to late-onset pleural empyema, followed by bacteremia and endophthalmitis due to an MDR hospital-acquired bacteria. Systemic antibiotics that can achieve therapeutic levels in the eye should be used at the recommended doses and supplemented with intravitreal antimicrobials. Unlike exogenous endophthalmitis, the role of pars-plana vitrectomy is not well established in endogenous endophthalmitis and should be dealt on case-to-case basis. Systemic antibiotics are typically continued for at least 3–4 weeks or as the extraocular infection of the patient dictates.

### AUTHOR CONTRIBUTIONS

**Sreethish Sasi:** Conceptualization; investigation; methodology; writing – original draft; writing – review and editing. **Hazem Faraj:** Writing – original draft; writing – review and editing. **Raja Barazi:** Writing – original draft; writing – review and editing. **Jouhar Kolleri:** Writing – original draft; writing – review and editing. **Chitrambika P:** Methodology; writing – original draft; writing – review and editing. **Muna A. Rahman Al Maslamani:** Writing – original draft; writing – review and editing. **Maisa Ali:** Writing – original draft; writing – review and editing.

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### CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

### DATA AVAILABILITY STATEMENT

Data supporting the conclusions of the study are all available free of cost through open access journals and websites.

### ETHICAL APPROVAL

The manuscript of this case report was approved by Institutional Review Board (IRB), Medical Research Centre (MRC) of Hamad Medical Corporation. Approval Number: MRC-04-22-198.

## CONSENT

A written and informed consent was obtained from the patient for publication of his case information and images.

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