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Case Report

Asymptomatic Endogenous Endophthalmitis in a Preterm Neonate with Burkholderia cepacia Sepsis: A Rare Incidental Detection

Article History:

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Abstract:

Background: Endogenous endophthalmitis is a rare but vision-threatening intraocular infection caused by hematogenous spread of pathogens. Diagnosis in neonates is often delayed because external ocular signs may be subtle or completely absent. Burkholderia cepacia, a multidrug-resistant non-fermenting Gram-negative bacillus, is an uncommon cause of ocular infection in any age group and is exceedingly rare in neonates. **Case Presentation**: A 35-week preterm male neonate with respiratory distress and low birth weight developed late-onset sepsis, with blood culture growing Burkholderia cepacia. During routine retinopathy of prematurity (ROP) screening, the right eye showed hazy vitreous media despite absence of conjunctival congestion, chemosis, or hypopyon. B-scan ultrasonography revealed lateral choroidal detachment, consistent with endogenous endophthalmitis. TORCH screening was negative. Persistent ocular involvement despite systemic antibiotics led to vitreous tap with intravitreal vancomycin and ceftazidime; vitreous cultures remained sterile. Limited response prompted administration of intravenous corticosteroids, but vitreous opacity and detachment persisted, and pars plana vitrectomy was advised. The case highlights a rare presentation of asymptomatic endogenous endophthalmitis secondary to Burkholderia cepacia sepsis in a preterm neonate. The absence of ocular symptoms underscores the crucial role of routine ophthalmic screening in septic premature infants. Early detection, targeted therapy, and timely surgical consideration are essential to prevent irreversible visual morbidity.

Keywords: Endogenous endophthalmitis, Burkholderia cepacia, Neonate, Prematurity, Sepsis, Vitritis, Intravitreal antibiotics

INTRODUCTION

Endogenous endophthalmitis is a severe, vision-threatening intraocular infection that results from hematogenous dissemination of pathogens from a distant systemic source into the eye [1]. Unlike exogenous endophthalmitis, which occurs following trauma, ocular surgery, or direct inoculation, the endogenous form is characterized by its subtle onset and diagnostic complexity, particularly in neonates. The neonatal period is marked by immunological immaturity, heightened vulnerability to systemic infections, and frequent need for invasive interventions factors that collectively increase susceptibility to bloodstream-mediated ocular infections [2]. Although endogenous endophthalmitis accounts for approximately 2–15% of all endophthalmitis cases, its occurrence in neonates remains exceptionally rare, often limited to isolated case reports and small series [3].

In neonates, the presentation is frequently nonspecific or completely asymptomatic, contributing to delayed diagnosis and poorer visual outcomes [4]. Prematurity, low birth weight, prolonged neonatal intensive care unit (NICU) stay, sepsis, indwelling catheters, and repeated blood transfusions are recognized risk factors that compromise the integrity of the blood–ocular barriers [5]. These barriers are already underdeveloped in preterm infants, making them particularly susceptible to microbial invasion during episodes of bacteraemia or fungemia [4–6]. Previous studies, such as those by Basu et al. and Murugan et al., have noted that neonatal endogenous endophthalmitis is commonly associated with severe systemic infections, and despite prompt antimicrobial therapy, visual prognosis is often guarded [4,6].

While a broad range of pathogens can cause endogenous endophthalmitis, the microbial spectrum differs geographically [7]. Gram-positive cocci dominate Western literature, whereas Gram-negative bacilli including Pseudomonas, Klebsiella, and Escherichia coli are more frequently reported in Asian cohorts and are associated with more fulminant disease courses [8]. Fungal pathogens, especially Candida species, remain important in neonates with prolonged antibiotic exposure or central line dependence. However, infections due to non-fermenting Gram-negative organisms, particularly Burkholderia cepacia, are exceedingly uncommon [9].

Burkholderia cepacia complex (Bcc) comprises a group of inherently multidrug-resistant, aerobic, non-fermenting bacilli known for their association with nosocomial outbreaks, contaminated hospital solutions, and severe infections in immunocompromised patients [10,11]. Their ability to survive in antiseptic environments, form biofilms, and resist multiple antibiotic classes makes them challenging pathogens in the NICU setting. Although B. cepacia bacteremia in neonates is well documented, ocular involvement is extremely rare, with only isolated adult case reports describing keratitis or endogenous endophthalmitis [12]. The organism's natural resistance mechanisms contribute to delayed therapeutic response and often result in poor structural and visual outcomes [13,14].

The present case is notable for several reasons: the infant was preterm, systemically infected, and yet exhibited no external ocular inflammatory signs. The diagnosis of endogenous endophthalmitis was made incidentally during routine retinopathy of prematurity (ROP) screening underscoring the vital role of scheduled ophthalmic evaluations in preterm neonates, especially those with sepsis. Choroidal detachment and vitreous haze identified on B-scan ultrasonography provided crucial early diagnostic clues despite sterile vitreous cultures, a frequent occurrence in neonatal endogenous infections.

Given the rarity of B. cepacia–associated endogenous endophthalmitis in neonates, each documented case adds valuable insight into early recognition, microbiological patterns, management challenges, and expected outcomes. The case reports the importance of integrating ophthalmic screening into the routine evaluation of septic neonates to enable timely intervention and optimize visual prognosis.

CASE PRESENTATION

Patient Information

A male preterm neonate was delivered at 35 weeks of gestation to a primigravida mother via normal vaginal delivery. He weighed 1.6 kg at birth and required immediate admission to a peripheral Neonatal Intensive Care Unit (NICU) for respiratory distress and low birth weight. In the early neonatal period, he developed thrombocytopenia requiring platelet and plasma transfusions. At 20 days of life, he was referred to KLE Hospital due to persistent oxygen dependency and for detailed cardiac evaluation.

Clinical Findings

On arrival, the infant required high-flow nasal cannula (HFNC) respiratory support for seven days, followed by oxygen support via hood. Systemic examination revealed features suggestive of sepsis, and empirical antibiotics were initiated. Echocardiography demonstrated significant mid-muscular and perimembranous ventricular septal

defects (VSD) associated with severe pulmonary hypertension, prompting initiation of decongestive therapy.

Blood culture obtained on day 20 grew Burkholderia cepacia, confirming late-onset sepsis. Antibiotic therapy was appropriately modified according to the organism's sensitivity profile. By day 27 of life, the neonate showed systemic stabilization with improved feeding and stable vital signs.

Ophthalmic Evaluation

Routine screening for retinopathy of prematurity (ROP) was performed during ongoing NICU care. Examination of the right eye revealed hazy vitreous media without external inflammatory signs such as conjunctival congestion, chemosis, corneal clouding, or hypopyon. The left eye was completely normal on evaluation. These atypical and subtle findings raised suspicion for posterior segment pathology.

A B-scan ultrasonography of the right eye revealed lateral choroidal detachment, a finding highly suggestive of endogenous endophthalmitis. To exclude congenital infectious etiologies, TORCH serology was performed and returned negative.

Diagnostic Assessment

Despite targeted systemic therapy for B. cepacia sepsis, ocular findings persisted. A vitreous tap was conducted to obtain intraocular samples, followed by intravitreal administration of vancomycin and ceftazidime. The vitreous culture demonstrated no microbial growth, which is not uncommon in neonatal endogenous endophthalmitis owing to low organism load or prior systemic antibiotics.

Repeat echocardiography showed continued VSD-related hemodynamic burden, requiring escalation of decongestive management. Ocular imaging remained essential in disease monitoring, with persistent vitreous haze and choroidal detachment documented on follow-up B-scan.

Therapeutic Interventions

Given the lack of adequate response to systemic and intravitreal antibiotics, a five-day course of intravenous corticosteroids was administered. However, the therapeutic response was minimal, and vitreoretinal pathology showed little improvement. Persistent vitreous opacification and choroidal detachment warranted consideration of surgical intervention. In view of the refractory vitreoretinal pathology, pars plana vitrectomy of the right eye was performed. The procedure was uneventful, and the infant retained vision with no postoperative visual loss.

Follow-up and Outcome

Throughout the clinical course, the left eye remained completely normal with no signs of involvement. The surgery was uneventful, and postoperative recovery was satisfactory. Follow-up examinations showed a clear vitreous cavity, resolution of the choroidal detachment, and a normal-appearing posterior segment. The right eye remained structurally stable with no postoperative complications or vision loss. Figures obtained during evaluation included B-scan ultrasonography demonstrating lateral choroidal detachment (Figure 1) and fundus photography showing a hazy vitreous obscuring the posterior pole (Figure 2).

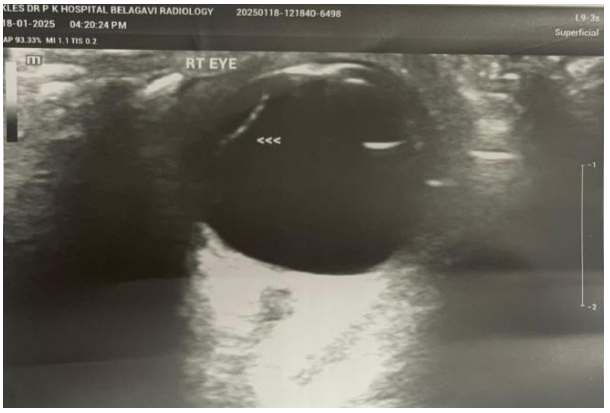

Figure 1. B-scan ultrasonography of the right eye showing lateral choroidal detachment suggestive of endogenous endophthalmitis.

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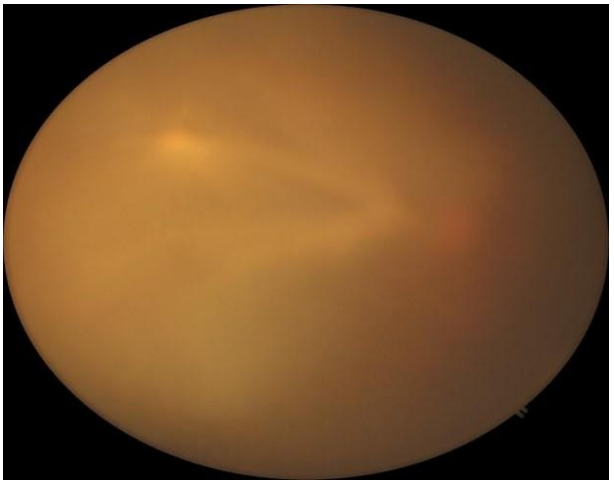

Figure 2. Fundus photograph of the right eye showing hazy vitreous media obscuring visualization of the posterior pole.

DISCUSSION

Endogenous endophthalmitis in neonates is a rare but devastating ocular emergency resulting from

hematogenous spread of pathogens to the posterior segment of the eye [15]. Its diagnosis is often delayed because neonates rarely manifest classical ocular inflammation, making incidental detection such as during routine retinopathy of prematurity (ROP) screening clinically invaluable [11,13]. The present case represents a highly uncommon occurrence of Burkholderia cepacia–associated endogenous endophthalmitis in a preterm neonate, notable for its asymptomatic presentation and refractory response to therapy.

Comparative studies consistently highlight the distinct pathogen spectrum in neonatal endogenous endophthalmitis across regions. While fungal organisms, particularly Candida spp., predominate in Western literature, Asian cohorts report higher prevalence of Gram-negative bacilli including Pseudomonas, Klebsiella, and Escherichia coli. Basu et al. documented six neonatal cases of endogenous endophthalmitis secondary to sepsis, most of which involved Gram-negative pathogens and exhibited poor visual outcomes despite timely intravitreal and systemic treatment [6]. Similarly, Murugan et al. reported 11 pediatric cases in South India where delayed presentation and systemic instability contributed to irreversible retinal damage [4].

In comparison with these commonly encountered organisms, Burkholderia cepacia remains exceedingly rare as an ocular pathogen. Most published cases pertain to adults, primarily postoperative keratitis or endophthalmitis following contaminated ophthalmic solutions or surgical instruments. Örnek et al. described B. cepacia keratitis progressing to endophthalmitis after cataract surgery, and Beca et al. reported a small series demonstrating uniformly guarded visual outcomes despite aggressive antimicrobial therapy [12,16]. Kurumkattil et al. documented an adult case of endogenous endophthalmitis due to B. cepacia, emphasizing the organism's low virulence but high capacity for chronic intraocular infection [13]. These comparative data underscore both the rarity and clinical difficulty of managing B. cepacia endophthalmitis [13].

The intrinsic multidrug resistance of B. cepacia complicates management. Its well-known resistance mechanisms include efflux pumps, biofilm formation, and limited permeability of antimicrobial agents. These biological characteristics can result in poor intraocular penetration of systemic antibiotics, thereby necessitating early intravitreal therapy. Standard intravitreal agents vancomycin for Gram-positive organisms and ceftazidime for Gram-negative organisms remain the empirical first line, although B. cepacia sensitivity to ceftazidime is variable. Vitreous cultures may be negative in neonates due to prior systemic therapy or low microbial load, as observed in the case.

The indication for pars plana vitrectomy in neonatal endophthalmitis remains debated. Comparative adult series, including the Early Vitrectomy Study for endogenous endophthalmitis, support early surgical intervention in cases with severe vitritis or poor response to medical therapy [12]. However, neonatal data are limited, with variable outcomes reported. Some authors advocate early vitrectomy to reduce organism burden and clear inflammatory debris, while others recommend conservative therapy due to the fragile ocular structures of preterm infants [15,16]. Persistent vitreous opacification and choroidal detachment in the neonate aligned with published indications favouring vitrectomy, particularly in cases refractory to intravitreal and systemic therapy.

The case further emphasizes the essential role of routine ophthalmologic screening in preterm neonates with systemic infections. The absence of pain, redness, or external inflammation as in the present infant mirrors observations from previous neonatal series where ocular pathology was detected only through structured screening protocols. In a population vulnerable to rapid progression and permanent vision loss, early detection through ROP examinations provides a critical window for diagnosis.

The case contributes to the extremely limited literature on neonatal B. cepacia endogenous endophthalmitis and highlights the need for heightened vigilance, interdisciplinary coordination, and individualized management based on organism-specific behavior and disease severity.

CONCLUSION

Endogenous endophthalmitis in neonates is an uncommon but visually destructive condition that often remains clinically silent until advanced stages. The case highlights an exceptionally rare presentation of asymptomatic unilateral endogenous endophthalmitis caused by Burkholderia cepacia in a preterm neonate with late-onset sepsis. The organism's intrinsic multidrug resistance and limited response to systemic and intravitreal therapy underscore the therapeutic challenges associated with such infections. Comparative reports in the literature consistently demonstrate poor visual prognosis for Gram-negative endogenous endophthalmitis, further emphasizing the importance of early recognition.

Routine ROP screening played a pivotal role in detecting posterior segment pathology in the infant, reaffirming its value beyond retinopathy surveillance. Timely imaging, multidisciplinary evaluation, and consideration of surgical intervention such as pars plana vitrectomy are essential when medical therapy

fails to achieve adequate resolution.

The case reports the need for heightened ophthalmic vigilance in septic preterm infants and adds meaningful insight to the limited literature on B. cepacia ocular infections.

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