

Dark hypopyon in *Streptococcus bovis* endogenous endophthalmitis: clinicopathologic correlations

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Abstract

Purpose The aim of this report is to present a previously unreported causative organism associated with brown-pigmented hypopyon in a patient with endophthalmitis.

Methods This is a retrospective case report which includes clinicopathologic correlations.

Results Vitreous cultures demonstrated *Streptococcus bovis* infection resulting in a brown-pigmented hypopyon, with uveal pigment found intra- and extracellularly on pathologic examination of the pupillary membrane.

Conclusions *S. bovis* endophthalmitis may be a cause of dark hypopyon, especially in patients with a history of liver disease, and, when identified, warrants colonoscopy and cardiac workup.

Keywords *Streptococcus bovis* · Brown/dark hypopyon · Endophthalmitis · Colon cancer · Endocarditis

Introduction

The presence of brown pigment within a hypopyon is a useful diagnostic clue and has been reported in the setting

of necrotic intraocular melanoma as well as *Listeria monocytogenes* and *Serratia marcescens* endogenous endophthalmitis [1, 2].

We report a patient that presented with brown hypopyon in the setting of *Streptococcus bovis* endogenous endophthalmitis, thereby expanding the differential diagnosis for this clinical clue. This report also discusses the systemic associations, especially colon cancer and endocarditis [3].

Case report

A 47-year-old Asian man with a history of cirrhosis and alcoholism was referred with a red, painful left eye of 3-days duration. On examination, visual acuity was 20/70 in the right eye and light perception in the left eye. Pressures were 15 and 20 in the right and left eyes, respectively. Examination of the left eye demonstrated a corneal ring infiltrate, brown hypopyon (Fig. 1a), and a dark pupillary membrane. B-scan ultrasonography demonstrated dense vitreous opacities, and a presumptive diagnosis of endogenous endophthalmitis was made. The patient underwent vitreous tap and intravitreal injection of vancomycin 1 mg/0.1 mL, ceftazidime 2.25 mg/0.1 mL, and voriconazole 100 mcg/0.1 mL.

Blood cultures were drawn and the patient was started on intravenous penicillin 3 million units/4 h and cefotaxime 1 gm/8 h for probable bacteremia. Abdominal ultrasound and CT showed evidence of cirrhosis, and transthoracic echocardiography was normal. Vitreous and blood cultures revealed *S. bovis*. The patient underwent colonoscopy, which ruled out colon cancer.

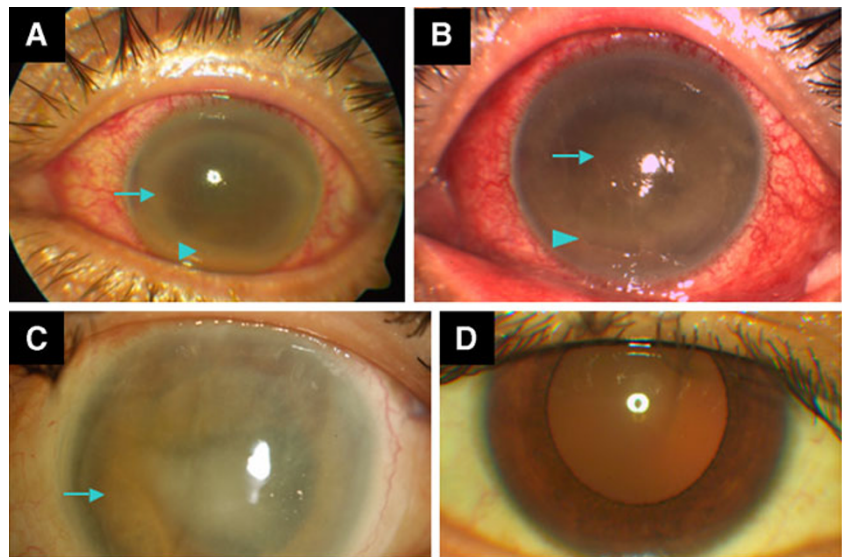
The left eye did not improve (Fig. 1b); on day 5 he underwent a second tap and injection with vancomycin, ceftazidime, and dexamethasone. Vitreous cultures

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Fig. 1 a–d External photographs. **a** Left eye, at presentation, dark corneal ring infiltrate (arrow), and brown hypopyon (arrowhead). **b** Four days later, progression of dark hypopyon (blue arrowhead) and development of a brown pupillary membrane (arrow). **c** Left eye, postoperative corneal edema and significant iris heterochromia (arrow). **d** Right eye, showing the patient's original iris color, obtained at a similar time point as **c**



remained positive for *S. bovis* (vancomycin sensitive), therefore, the patient underwent pars plana vitrectomy, lensectomy, pupillary membranectomy, and intravitreal vancomycin on day 9. The vitreous specimen was positive for *S. bovis*, while the pupillary membrane showed extensive uveal pigment, both extracellularly and within necrotic leukocytes (Fig. 2).

On day 19, the patient underwent repeat vitreous cultures and intravitreal injection of vancomycin and dexamethasone. Cultures were negative, however the final visual acuity was light perception and the eye developed significant iris heterochromia (Fig. 1c and d).

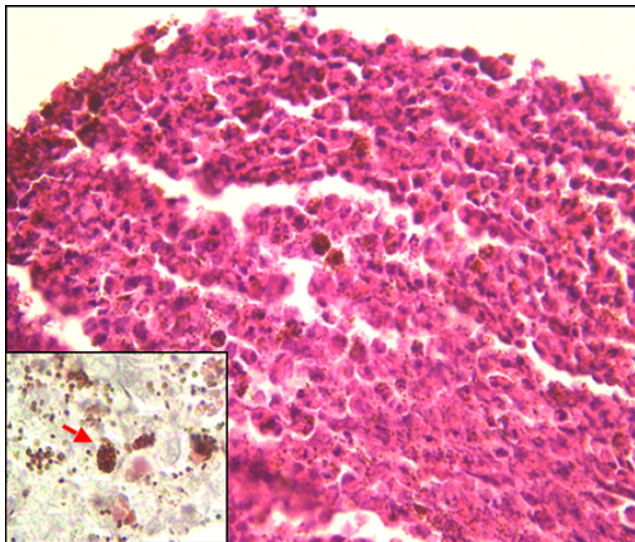


Fig. 2 Histopathological aspect of pupillary membrane, displaying necrotic leukocytes admixed with a large amount of uveal pigment, both extra- and intracellularly (hematoxylin-eosin, original magnification $\times 400$). Gram stain was negative for bacteria (inset, original magnification $\times 1,000$). Note intracellular pigment granules (inset, arrow)

Discussion

This report expands the causes of dark hypopyon to include *S. bovis*, a Gram (+) cocci occasionally isolated from the human digestive tract and known to cause endocarditis, and more rarely, urinary infections and neonatal septicemia and meningitis. Although rarely associated with ocular infections, *S. bovis* has been reported in cases of endogenous endophthalmitis, although the authors did not comment on the color of the hypopyon [4].

An important association between *S. bovis* and colon cancer has been demonstrated, although the exact mechanism remains elusive [2]. A change in colonic flora secondary to hepatic dysfunction was hypothesized to explain *S. bovis* translocating from the intestinal mucosa into the blood stream [5]. Our patient had longstanding liver dysfunction, which likely contributed to his bacteremia; he did not, however, develop colon cancer during 4 years of follow up.

Pink hypopyon has been associated with both *S. marcescens* due to necrosis and bleeding and *Klebsiella pneumoniae* due to red pigment prodigiosin [6, 7]. *L. monocytogenes* endophthalmitis presents with brown hypopyon, profound vision loss, and no identifiable extraocular focus of infection [2]. In contrast to our patient, *Listeria* is associated with significant glaucoma, which, similar to the dark hypopyon, is ascribed to pigment dispersion. While our patient had normal IOP, pigment dispersion (Fig. 2) and eventual iris heterochromia (Fig. 1d) suggest a similar mechanism.

Conclusion

In summary, *S. bovis* endophthalmitis may present with brown hypopyon, and should be suspected in patients with

a history of liver disease. Once *S. bovis* is cultured, colonoscopy and cardiac workup should be performed.

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