Available online at www.sciencedirect.com

ScienceDirect

Journal of Current Ophthalmology 28 (2016) 152–154

http://www.journals.elsevier.com/journal-of-current-ophthalmology

Case report

Endophthalmitis caused by *Acinetobacter* spp. as the presenting manifestation of diabetes mellitus

Mohammad Mehdi Parvaresh, Amir Abbas Mehrpouya, Rahimeh Ganji Anari, Mohammadreza Aghamirsalim, Kaveh Abri Aghdam*, Khalil Ghasemi Falavarjani

Eye Research Center, Rassoul Akram Hospital, Iran University of Medical Sciences, Tehran, Iran

Received 15 February 2016; revised 16 May 2016; accepted 16 May 2016
Available online 11 June 2016

Abstract

Purpose: We describe a patient with endogenous endophthalmitis caused by *Acinetobacter* spp. as the first clinical presentation of diabetes mellitus.
Method: A 48-year-old otherwise healthy woman was referred with signs and symptoms of acute endophthalmitis in the left eye. Systemic work-up, vitreous tap, and intravitreal antibiotic injection were performed followed by pars plana vitrectomy.
Results: The laboratory tests confirmed the diagnosis of diabetes mellitus. Vitreous culture was positive for *Acinetobacter* spp., and the organism was sensitive to colistin. One month after surgery, vision was no light perception, and the eye was phthisical.
Conclusion: Diagnostic work-up should be performed even in otherwise healthy patients with endogenous endophthalmitis.

Keywords: Endophthalmitis; Diabetes mellitus; *Acinetobacter* species

Introduction

Endophthalmitis is a sight-threatening ocular condition that usually occurs after surgical procedures or trauma. Endogenous endophthalmitis is less common and occurs when organisms enters the eye by crossing the blood–ocular barrier.[1]

Endogenous endophthalmitis is usually associated with systemic risk factors leading to the defects in immune system.[1,2] Diabetes mellitus (DM) is a chronic immune-compromising illness, and several endogenous endophthalmitis cases have been reported in diabetic patients[3–5]; however, endogenous endophthalmitis as the presenting manifestation of DM is extremely rare.[6] Moreover, endogenous endophthalmitis caused by *Acinetobacter* spp. has been rarely reported.[7,8] Here, we report a patient with endogenous endophthalmitis caused by *Acinetobacter* spp. as the first clinical presentation of DM.

Case report

A 48-year-old female patient was referred with pain, redness, and decreased vision in her left eye during the previous four days. Oral prednisone 50 mg/day had been prescribed by the referring ophthalmologist without improvement in clinical symptoms. At presentation, vision was 10/10 in the right eye and hand movements in her left eye. Slit lamp biomicroscopy examination revealed normal anterior segment and mild scattered dot and blot retinal hemorrhages in the right eye. The cornea showed diffuse stromal haze without keratic precipitates. The anterior chamber demonstrated a hypopyon, and fibrin membrane was noted on the

Financial interest: None of the authors has any financial interest in the subject matter of this paper.

* Corresponding author. Eye research Center, Rassoul Akram hospital, Iran University of Medical Sciences, Sattarkhan-Niayesh Street, Tehran, 14455-364, Iran.
E-mail address: kaveh.abri@gmail.com (K. Abri Aghdam).

Peer review under responsibility of the Iranian Society of Ophthalmology.

http://dx.doi.org/10.1016/j.joco.2016.05.005

anterior surface of the crystalline lens in the left eye. Moreover, the fundus in the left eye was invisible. Ultrasound examination revealed dense vitreous opacities with an attached retina. She had no previous history of ocular and systemic diseases, trauma, or chronic medication use. There were no signs of sepsis like fever, chills, or loss of appetite.

She was admitted with impression of endogenous endophthalmitis, and then vitreous tap and intravitreal injection of vancomycin/ceftazidime were performed. Laboratory examination revealed fasting blood sugar: 280, mg/dl, random blood glucose: 500 mg/dl, and hemoglobin A1C: 9.1. Other endocrinologic and rheumatologic evaluations were unremarkable. No specific site of infection was found by the infectious diseases specialist. The results of chest X-ray and computed tomography (CT) scan of the paranasal sinuses were unremarkable. Blood, urine, and stool cultures were negative.

The next day, no improvement occurred in the patient's clinical signs and symptoms. Pars plana vitrectomy without endotamponade was performed. The vitreous cavity was filled with pus. Intravitreal injection of vancomycin/ceftazidime was repeated at the end of vitrectomy.

Vitreous culture was positive for *Acinetobacter* spp., and the organism was resistant to ciprofloxacin, amikacin, gentamicin and ceftazidime, imipenem, and piperacillin-tazobactam and was sensitive only to colistin. The patient received intravenously 1 g ceftazidime two times a day and 500 mg vancomycin three times a day for three days.

At the first postoperative day, the symptoms improved, the visual acuity was light perception, and the anterior chamber was clear; however, severe vitritis was still present. Vitreous reaction improved during the next days. One month later, vision was no light perception, and the eye was phthisical.

Discussion

Our patient was not aware of her systemic disease, and the DM was detected by the laboratory tests. The patient took oral prednisolone, and this could raise the blood sugar levels; however, the patient's right eye had findings consistent with non-proliferative diabetic retinopathy. Ko et al reported a case of bilateral endogenous endophthalmitis in a 55-year-old man without history of systemic disease that DM was detected via systemic work-up.[6] In addition, urinary infection was found, and blood culture was positive for *Staphylococcus aureus*. Ocular and urinary infections responded to antibiotic therapy; however, *Candida albicans* was isolated from the culture of aqueous humor, and the patient was treated successfully with oral fluconazole.

Acinetobacter spp. are found in the environment, soil, and water and have the ability to colonize almost any surface including the skin, respiratory, and gastrointestinal tracts. There are few reports of post-operative and post-traumatic endophthalmitis caused by *Acinetobacter* spp., and endogenous endophthalmitis caused by *Acinetobacter* spp. has rarely been reported.[7–9] Although diabetes mellitus may be considered a risk factor in our patient, most reported cases in previous articles did not have any history of systemic illnesses.[7,8] Previously reported *Acinetobacter* isolates were from *baumannii* and *calcoaceticus* species; however, considering the rare detection of this infection in our hospital and difficulty in differentiation of the species,[10–12] our laboratory did not report the exact species of the organism.

Acinetobacter spp. infection is a major concern in the field of infectious diseases because of the rapid development of resistance to antimicrobial agents.[10,11] *Acinetobacter* spp. are also intrinsically less susceptible to antibiotics than many virulent bacteriae like *Enterobacteriaceae*.[11] Carbapenems, fluoroquinolones, and doxycycline may have activity against *Acinetobacter* spp. The most important problem in the treatment of *Acinetobacter* infection is acquired multi-drug resistance. The mechanism for acquired resistance includes any or all of the following mechanisms[13]: degradation enzymes against β-lactams, modification enzymes against aminoglycosides, altered binding sites for quinolines, and a variety of efflux mechanisms and changes in outer membrane proteins. Recent clinical isolates of *Acinetobacter* spp. were often found to be multi-resistant to carbapenems, fluoroquinolones, and aminoglycosides. Colistin-resistant isolates are rare, but started to be isolated.[11,12] Our case was resistant to all antibiotics tested except for colistin. Considering adverse consequences of delayed treatment, carbapenems such as Imipenem are often given as a drug of choice for serious *Acinetobacter* spp. infections. However, given the probability that the organism would be most likely resistant to one of the common first line antibiotics, treatment of the infection should be tailored based on antimicrobial susceptibility testing.[12]

In conclusion, diabetes mellitus may rarely be discovered following endogenous endophthalmitis. Even in otherwise healthy patients with endogenous endophthalmitis, a complete diagnostic work-up should be performed.

References

1. Arevalo JF, Jap A, Chee S, Zeballos DG. Endogenous endophthalmitis in the developing world. *Int Ophthalmol Clin*. 2010;50:173–187.
2. Okada AA, Johnson RP, Liles WC, D'Amico DJ, Baker AS. Endogenous bacterial endophthalmitis: report of a ten-year retrospective study. *Ophthalmology*. 1994;101:832–838.
3. Lee SY, Chee SP. Group B Streptococcus endogenous endophthalmitis: case reports and review of the literature. *Ophthalmology*. 2002;109:1879–1886.
4. Lee JY, Kim KH. Endogenous endophthalmitis complicated by pyogenic liver abscess: a review of 17 years' experience at a single center. *Digestion*. 2014;90:116–121.
5. Sheu SJ, Kung YH, Wu TT, Chang FP, Horng YH. Risk factors for endogenous endophthalmitis secondary to klebsiella pneumoniae liver abscess: 20-year experience in Southern Taiwan. *Retina*. 2011;31:2026–2031.
6. Ko Y, Liu CJ, Chung YM, Tsai WC. Bilateral endogenous Candida endophthalmitis as the presenting manifestation of diabetes mellitus. *Eye*. 2001;15:238–240.
7. Chen KJ, Hou CH, Sun MH, Lai CC, Sun CC, Hsiao CH. Endophthalmitis caused by *Acinetobacter baumannii*: report of two cases. *J Clin Microbiol*. 2008;46:1148–1150.
8. Gopal L, Ramaswamy AA, Madhavan HN, et al. Endophthalmitis caused by *Acinetobacter calcoaceicus*. A profile. *Indian J Ophthalmol*. 2003;51:335–340.

9. Falavarjani KG, Neoozadeh S, Modarres M, et al. Isolates and antibiotic resistance of culture-proven endophthalmitis cases presented to a referral center in Tehran. *Middle East Afr J Ophthalmol*. 2012;19:361−363.
10. Doughari HJ, Ndakidemi PA, Human IS, Benade S. The ecology, biology, and pathogenesis of Acenitobacter spp.: an overview. *Microbes Environ*. 2011;26:101−112.
11. Lee K, Yong D, Jeong SH, Chong Y. Multidrug-resistant *Acinetobacter* spp.: increasingly problematic nosocomial pathogens. *Yonsei Med J*. 2011;52:879−891.
12. Howard A, O'Donoghue M, Feeney A, Sleator RD. *Acinetobacter baumannii*: an emerging opportunistic pathogen. *Virulence*. 2012;3:243−250.
13. Tenover FC. Mechanisms of antimicrobial resistance in bacteria. *Am J Infect Control*. 2006;34:S3−S10. discussion S64−73.