• Clinical Research •

Microbial spectrum and risk factors of endogenous endophthalmitis in a tertiary center of Northern China

Lin-Yang Gan[1], Jun-Jie Ye[1], Hui-Ying Zhou[2], Han-Yi Min[1], Lin Zheng[1]

[1]Department of Ophthalmology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences, Beijing 100730, China
[2]Department of Ophthalmology, Beijing Tian Tan Hospital, Capital Medical University, Beijing 100730, China
Correspondence to: Jun-Jie Ye. Department of Ophthalmology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences, Beijing 100730, China. yejunjie_xh@163.com
Received: 2021-11-28 Accepted: 2022-02-25

Abstract

● **AIM:** To study the clinical features, microbial spectrum, associated factors and prognosis of endogenous endophthalmitis (EE) in a group of Chinese patients.

● **METHODS:** The medical records from 32 eyes of 29 patients diagnosed with EE in Peking Union Medical College Hospital from January 2009 to October 2019 were reviewed.

● **RESULTS:** The initial visual acuity (VA) of 30 eyes in this study was worse than 20/400. Twenty-three eyes were diagnosed with fungal endophthalmitis and nine with bacterial endophthalmitis. The most common fungal and bacterial isolates were *Candida* and *Klebsiella pneumoniae,* respectively. Several rare fungi and bacteria species were also isolated from our patients, including *Cryptococcus*, *Paecilomyces*, *Brucella*, and *Bacillus licheniformis*. The leading risk factor for EE was diabetes. The most common extraocular infection locus was genitourinary tract. Vitrectomy was performed on twenty-nine eyes. Eight eyes achieved final VA of 20/400 or better. EE caused by *Candida* had a better prognosis.

● **CONCLUSION:** The visual outcome of EE is based on pathogens and prompt intervention. Early vitrectomy and antimicrobial treatment are beneficial for EE.

● **KEYWORDS:** endogenous; endophthalmitis; risk factors; vitrectomy

DOI:10.18240/ijo.2022.10.17

Citation: Gan LY, Ye JJ, Zhou HY, Min HY, Zheng L. Microbial spectrum and risk factors of endogenous endophthalmitis in a tertiary center of Northern China. *Int J Ophthalmol* 2022;15(10):1676-1682

INTRODUCTION

Endophthalmitis is a serious visual-threatening infectious disease. Most cases are exogenous: secondary to cataract surgery, intravitreal injection, or penetrating injury. Endogenous endophthalmitis (EE) is relatively rare and is caused by the seeding of pathogenic microbe into the eyeball through blood flow. Risk factors include central venous catheterization, total parenteral nutrition, broad-spectrum antibiotics, abdominal surgery, neutropenia, and glucocorticoid therapy and diabetes[1].

Although EE results from transient bacteremia or fungemia, most patients have no obvious systemic symptoms. They would first visit ophthalmic clinic when they experience eye pain and vision loss. Endogenous fungal endophthalmitis, in particular, progresses slowly, and its symptoms may occur a few weeks after fungemia. Therefore, patients without systemic symptoms are often initially misdiagnosed as non-infective uveitis. In addition, hospitalized patients with bacteremia or fungemia may not be able to report their symptoms due to their severe condition, and endophthalmitis may be neglected.

The spectrum of pathogens varies in the literature. Reports from European and American countries[2-4] showed similar proportions of fungi and bacteria, and Gram-positive bacteria are more common compared to Gram-negative ones. However, Gram-negative bacteria are predominant in Asian countries, and the most common pathogens are *Klebsiella pneumoniae*[5-7] and *Pseudomonas aeruginosa*[8]. This is believed to be mainly associated with the high incidence of hepatobiliary diseases in these Asian countries. Etiological diagnosis is related to the choice of antibiotics and antifungal agents in empirical treatments, which is important for the prompt and proper treatment in newly diagnosed patients.

This is a retrospective study conducted in North China. We collected clinical information of patients diagnosed with EE in the Ophthalmology Department of Peking Union Medical College Hospital in the past 10y. Ocular manifestations, risk factors, systemic diseases, treatments, and visual outcomes were reviewed and analyzed.

SUBJECTS AND METHODS

Ethical Approval This study was conducted according to the tenets of the Declaration of Helsinki, and approved by the

Ethics Committee of Peking Union Medical College Hospital, the Chinese Academy of Medical Sciences. The data were anonymous and retrospective, the requirement for informed consent was therefore waived by the Ethics Committee of Peking Union Medical College Hospital, the Chinese Academy of Medical Sciences.

This is a retrospective study of EE cases in Peking Union Medical College Hospital from January 2009 to October 2019. EE was defined as: 1) inflammation in one or both eyes; 2) aqueous or vitreous specimens showed positive smear or culture results; 3) no recent history of eye surgery or penetrating trauma (at least one year). Patients with high suspicion of infectious uveitis but negative pathogenic results were excluded. These patients were all admitted to our hospital for surgical treatment, including intravitreal injection, vitrectomy or enucleation/evisceration. The aqueous or vitreous specimens were sent for smear and culture. Those with positive cultures were further tested for susceptibility testing. The decision to perform pars plana vitrectomy (PPV) was based on: 1) initial visual acuity (VA) less than hand motion; 2) clinical worsening or lack of improvement at 24-48h, despite intravitreal antibiotics; 3) B-scan documentation of severe vitreous opacities or membranes; 4) as well as the willingness of patients. PPV with silicone oil tamponade was performed for patients with retinal detachment, retinal tears or proliferative vitreous retinopathy. The main data collected were: demographic characteristics, risk factors, systemic conditions, extraocular infections, pathogenic organisms, treatment, and visual outcome.

RESULTS

Twenty-nine patients (32 eyes) with EE were analyzed in this study. The median follow-up time was 6mo (1-57mo). There were 7 males and 22 females, with an average age of 52 (ranging 25 to 79)y. The median time between the onset of infection and the first visit to our hospital was one month, ranging from 4d to 4mo. Only four patients had a fever before ocular symptoms developed. Four patients received intravitreal anti-infective agents before visiting our hospital, and one patient received PPV. Fifteen patients (51.7%) were misdiagnosed in local hospitals, of which 13 were misdiagnosed as noninfectious uveitis, one as acute retinal necrosis and one as acute optic neuritis respectively. Ten patients had been treated with topical glucocorticoids and eight of them even received systemic steroids. VA at the first visit to our hospital was 20/400 or better in two eyes. Thirty eyes presented with VA lower than 20/400, ranging from 20/600 to no light perception (Table 1). Twelve eyes had mild anterior chamber reaction, and 20 eyes had moderate to severe anterior chamber reaction. Retinal detachment was detected in 10 eyes (patient 2, 3, 6, 9, 10, 16, 18, 24, 26, 29).

Of the 32 eyes (29 patients) with EE, 23 eyes (71.9%, 20 patients) were fungal EE. Most cases (14 eyes) were caused by *Candida albicans*. Two eyes were positive for *Aspergillus* species. *Paecilomyces* species was isolated from two eyes, along with singular case of *Candida magnolia*, *Streptomyces rimosus*, and *Cryptococcus neoformans*. Two eyes showed positive smear with hyphae and spores. Bacterial isolates were only found in nine eyes (28.1%, 9 patients), of which six were Gram-negative and three were Gram-positive. The most common bacterial isolates were *Klebsiella pneumoniae* (3 eyes; Tables 1 and 2).

In this study, most samples were obtained from PPV. Only eight patients underwent vitreous/aqueous tap. Two out of four aqueous tap samples and five out of six vitreous tap samples had positive cultures.

Potential systemic risk factors and extraocular infection loci were found in 69% of our patients. The leading risk factor was diabetes mellitus, followed by systemic glucocorticoid therapy and recent invasive surgical procedures. Ten patients (34.5%) had extraocular infection loci, including genitourinary tracts, lung, and brain. Nearly half the cases of EE caused by *Candida* were associated with genitourinary infection.

Twenty-nine eyes (26 patients) received PPV, 16 eyes were filled with silicone oil. Twenty-four eyes (22 patients) were treated with intravitreal injection of therapeutic agents, as shown in Table 3. Eight eyes (7 patients) were initially treated with intravitreal injection but 7 of them underwent secondary treatment with PPV or evisceration (patient 17) eventually. Four eyes were finally eviscerated (patient 17, 21, 23, 29).

Among these with positive fungal isolates, 13 patients received systemic antifungal therapy with fluconazole (11 intravenous and two oral administration). Two patients received intravenous itraconazole (patient 12 and 24, *Aspergillus* species infection). While amphotericin B was administered intravenously in one patient due to allergy to fluconazole (patient 20). Four patients did not receive systemic antifungal therapy. Eight of nine patients with bacterial EE were treated with systemic antibiotics, four with ceftazidime, two with vancomycin, one with imipenem, and one with levofloxacin respectively.

After surgery and antibiotic treatment, inflammation was controlled in 28 eyes and final VA outcomes were available (range: 20/40 to no light perception). The final VA improved in 15 eyes (53.6%) after treatment, and eight eyes (28.6%) achieved a final VA of 20/400 or better (Table 3). Of the eyes with bacterial EE, 4 (44.4%) of 9 were eviscerated, and only one eye had a final VA better than 20/400. However, endophthalmitis caused by fungi, especially *Candida* species, had a trend toward better visual outcomes. The final VA of seven eyes (46.7%) with *Candidal* EE achieved final VAs of 20/400 or better (A typical case was shown in Figure 1).

Table 1 Clinical feature of 29 endogenous endophthalmitis cases

No.	Age/sex	Eye	VA	Risk factors	Extraocular infections	Pathogen
1	36/F	OD	HM		Genitourinary infection	*Candida albicans*
		OS	LP			*Candida albicans*
2	35/F	OD	HM		Genitourinary infection	*Candida albicans*
3	27/F	OS	HM	Surgical abortion		*Candida albicans*
4	63/F	OS	HM	Steroids therapy		Hyphae and spores
5	49/F	OD	HM		Genitourinary infection	*Candida albicans*
6	59/F	OS	LP	Steroids therapy		Spores
7	54/F	OS	LP			*Paecilomyces lilacinus*
8	68/M	OD	HM			*Streptomyces rimosus*
9	55/M	OS	HM	Intestinal surgery		*Candida albicans*
10	37/F	OS	HM		Genitourinary infection	*Candida magnolia*
11	44/F	OS	HM			*Candida albicans*
12	47/F	OD	HM			*Aspergillus*
13	72/F	OD	HM	Diabetes	Brain abscess	*Candida albicans*
		OS	CF			*Candida albicans*
14	47/F	OS	LP		Pneumonia	*Candida albicans*
15	66/F	OD	HM			*G-Bacillus*
16	62/F	OD	20/600			*Candida albicans*
		OS	CF			*Candida albicans*
17	47/M	OD	LP			*Klebsiella pneumoniae*
18	64/M	OD	LP	Diabetes	Genitourinary infection	*Candida albicans*
19	47/F	OD	HM			*G+Coccus*
20	42/F	OS	CF			*Cryptococcus neoformans*
21	66/F	OS	NLP		Bronchiectasis	*Pseudomonas aeruginosa*
22	69/F	OD	LP			*Klebsiella pneumoniae*
23	61/F	OD	NLP	Diabetes	Pneumonia	*Klebsiella pneumoniae*
24	38/M	OD	LP	Steroids therapy		*Aspergillus fumigatus*
25	25/F	OD	20/133			*Brucella*
26	25/F	OD	CF	Surgical abortion		*Bacillus licheniformis*
27	63/F	0S	20/200		Genitourinary infection	*Candida albicans*
28	60/M	OS	LP	Diabetes		*Paecilomyces bainer*
29	79/M	OS	LP	MDS, leukopenia		*Streptococcus salivarius*

VA: Visual acuity; OS: Oculus sinister; OD: Oculus dexter; CF: Counting finger; HM: Hand motion; LP: Light perception; NLP: No light perception; MDS: Myelodysplastic syndrome.

Table 2 Risk factors and extraocular infections distribution according to microbial spectrum

Pathogen	Patients (*n*)	Extraocular infections			Risk factors			
		Genitourinary tract	Lung	Brain	Diabetes	Steroids	Leukopenia	Surgery
Fungus								
Candida albicans	11	5	1	1	2			2
Aspergillus	2					1		
Paecilomyces	2				1			
Candida magnolia	1	1						
Streptomyces rimosus	1							
Cryptococcus neoformans	1							
Bacteria								
Klebsiella pneumoniae	3		1		1			
Streptococcus salivarius	1						1	
Brucella	1							
Pseudomonas aeruginosa	1		1					
Bacillus licheniformis	1							1
Culture negative								
Hyphae and spores	2					2		
Gram-positive coccus	1							
Gram-negative bacillus	1							
Total	29	6	3	1	4	3	1	3

Table 3 Treatments and visual outcomes

No.	Eye	VA	Pathogen	Systemic treatment	Intravitreous agents (number of injections)	Vitrectomy	Visual outcome
1	OD	HM	*Candida albicans*	Fluconazole	Amphotericin B (3)	Yes	CF
	OS	LP	*Candida albicans*	Fluconazole	Amphotericin B (2)	Yes	HM
2	OD	HM	*Candida albicans*	Fluconazole	Amphotericin B (1)	Yes	20/2000
3	OS	HM	*Candida albicans*	Fluconazole	Amphotericin B (1)	Yes	NLP
4	OS	HM	Hyphae and spores	No	Amphotericin B (3)	Yes	HM
5	OD	HM	*Candida albicans*	Fluconazole	Amphotericin B (3)	Yes	20/500
6	OS	LP	Spore	Fluconazole	Amphotericin B (1)	Yes	CF
7	OS	LP	*Paecilomyces lilacinus*	No	Amphotericin B (2)	Yes	LP
8	OD	HM	*Streptomyces rimosus*	Fluconazole	Amphotericin B (2)	Yes	20/133
9	OS	HM	*Candida albicans*	Fluconazole	Amphotericin B (1)	Yes	CF
10	OS	HM	*Candida magnolia*	No	Amphotericin B (4)	Ye	HM
11	OS	HM	*Candida albicans*	Fluconazole	Amphotericin B (5)	No	LP
12	OD	HM	*Aspergillus*	Itraconazole	No	Yes	HM
13	OD	HM	*Candida albicans*	Fluconazole	No	Yes	20/60
	OS	CF	*Candida albicans*	Fluconazole	Amphotericin B (1)	Yes	20/40
14	OS	LP	*Candida albicans*	Fluconazole	Amphotericin B (2)	Yes	LP
15	OD	HM	*G-Bacillus*	Ceftazidime	No	Yes	HM
16	OD	20/600	*Candida albicans*	Fluconazole	Amphotericin B (2)	Yes	20/133
	OS	CF	*Candida albicans*	Fluconazole	Amphotericin B (1)	Yes	20/80
17	OD	LP	*Klebsiella pneumoniae*	Imipenem	No	No	Eviscerated
18	OD	LP	*Candida albicans*	Fluconazole	Amphotericin B (1)	Yes	LP
19	OD	HM	*G+Coccus*	Levofloxacin	Vancomycin (1)	Yes	HM
20	OS	CF	*Cryptococcus neoformans*	Amphotericin B	Amphotericin B (1)	Yes	LP
21	OS	NLP	*Pseudomonas aeruginosa*	Ceftazidime	Ceftazidime (1)	Yes	Eviscerated
22	OD	LP	*Klebsiella pneumoniae*	No	Ceftazidime (4)	Yes	LP
23	OD	NLP	*Klebsiella pneumoniae*	Ceftazidime	No	No	Eviscerated
24	OD	LP	*Aspergillus fumigatus*	Itraconazole	Amphotericin B (1)	Yes	NLP
25	OD	20/133	*Brucella*	Ceftazidime	Ceftazidime (1)	Yes	20/100
26	OD	CF	*Bacillus licheniformis*	Vancomycin	Vancomycin (1)	Yes	20/1000
27	OS	20/200	*Candida albicans*	Fluconazole	No	Yes	20/133
28	OS	LP	*Paecilomyces bainer*	No	No	Yes	20/400
29	OS	LP	*Streptococcus salivarius*	Vancomycin	No	Yes	Eviscerated

VA: Visual acuity; OS: Oculus sinister; OD: Oculus dexter; CF: Counting finger; HM: Hand motion; LP: Light perception; NLP: No light perception.

Table 4 Antimicrobial susceptibility testing

Pathogen	*n*	Result available	Susceptible agents
Candida albicans	11	7	Fluconazole, voriconazole
Aspergillus	2	1	Itraconazole
Candida magnolia	1	1	Fluconazole, voriconazole, itraconazole
Cryptococcus neoformans	1	1	Amphotericin B, fluconazole, voriconazole, itraconazole
Klebsiella pneumoniae	3	3	Ceftazidime
Pseudomonas aeruginosa	1	1	Ceftazidime
Bacillus licheniformis	1	1	Vancomycin
Total	20	15	

Antimicrobial susceptibility testing results were obtained in 17 eyes (15 patients, Table 4). Nine isolates of *Candida albicans* from seven patients and one isolate of *Candida magnolia* were susceptible to fluconazole. Two *Aspergillus* isolates were susceptible to itraconazole. Identifiable Gram-negative and Gram-positive bacteria isolates in this study were susceptible to ceftazidime and vancomycin respectively.

DISCUSSION

EE is relatively rare, accounting for only 5%-15% of all endophthalmitis[1]. However, with the use of broad-spectrum antibiotics and immunosuppressants, the incidence of EE has gradually increased[9]. Other risk factors for EE include diabetes, intravenous medication, and malignant tumors[2,9-10]. EE can also be caused by primary infectious diseases including

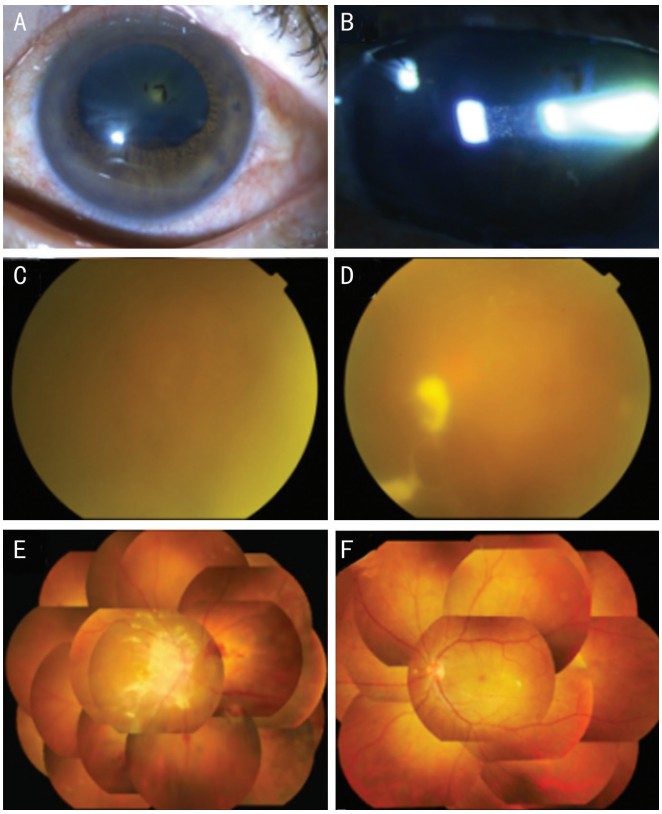

Figure 1 A 72-year-old female (patient 13), suffered from eye pain, reduced visual acuity (VA) and conjunctival injection for one month Slit lamp examination showed anterior synechiae (A) and anterior chamber reaction (B). Fundus photographs were obtained before (C, D) and after vitrectomy (E, F). *Candida albicans* was isolated from vitreous specimen. She received systemic fluconazole for more than 4mo and her VA was OD 20/60 OS 20/400 at final visit.

liver abscess, pneumonia, infective endocarditis, and urinary infection[5]. Sixty-two percent of patients (18/29) in this study had the aforementioned risk factors or extraocular infection loci. The most common risk factors are diabetes and glucocorticoid therapy. For fungal EE genitourinary infection was the leading cause, while the most common primary infection locus of bacterial EE was pulmonary infections.

Most patients with EE showed no systemic symptoms before ocular onset. Only four patients (13.8%) reported fever in our study. If transient bacteremia and fungemia spread to the eye without infecting other organs, patients might be in a good general condition when ocular symptoms occur. And the diagnosis of EE would be delayed. For patients with confirmed EE, it is necessary to screen for potential infectious diseases, *e.g.*, liver and lung for bacterial causes, and urogenital system for fungi. Moreover, magnetic resonance imaging (MRI) can be applied to exclude intracranial infection even the patient presented without neural manifestation.

Fungal EE usually has an insidious onset. Symptoms often do not occur until choroid inflammation spreads to the vitreous cavity. Typical vitreous opacities can be significant. EE is easily misdiagnosed as non-infectious uveitis because there is no clear history of ocular trauma. In our study, the diagnoses of 16 patients (55.2%) were incorrect at their initial visit to local hospitals. The rate of misdiagnosis was similar to the literature[4]. Fourteen eyes were misdiagnosed as non-infectious uveitis, and eight patients were treated with systemic glucocorticoids and even immunosuppressants such as cyclophosphamide and cyclosporine. In addition to delaying the correct anti-infective treatment, immunosuppressive treatment could significantly increase the risk of deterioration of the disease.

Bacterial EE often has an acute onset. A Meta-analysis[10] showed that the most common clinical manifestations were decreased vision (90%), followed by eye pain (50%), hypopyon (35%), and vitreous inflammation (33%). Gram-negative bacteria are the main pathogens of bacterial EE, especially in Asian countries. Studies from South Korea[5-6], Malaysia[7], India[8] revealed the most common bacteria were *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*, which were mainly associated with a high incidence of hepatobiliary disease in these countries.

The rate of positive culture in our study was 86.2%. The most common fungal isolate was *Candida* (12/20). Bacterial EE was mainly caused by Gram-negative bacteria (6/9), especially *Klebsiella pneumoniae*. *Aspergillus* is also a common pathogen in fundal EE[11], usually seen in immunosuppressed patients[1]. Two cases of *Aspergillus* were isolated in our study. Patient 24 had been taking glucocorticoid for a long time due to kidney disease, and the other one (patient 12) reported no risk factors. Due to the aggressiveness of *Aspergillus*, these patients usually had poor prognoses. Both of our two patients ended without light perception eventually.

Some rare fungi and bacteria isolates were also collected from our patients, including *Cryptococcus*, *Paecilomyces*, *Brucella*, and *Bacillus licheniformis*. Most of them were case reports in the literature. In addition to AIDS, endogenous cryptococcal endophthalmitis is more common in immunosuppressed patients[11-12]. But our patient was HIV-negative and had no history of immunosuppressants use. The exact cause of the disease remained unclear. Although the patient underwent systemic and intravitreal amphotericin B therapy, the visual outcome was still very poor. EE caused by *Paecilomyces* was reported to have a relatively good prognosis after treatment with amphotericin B and itraconazole[11]. However, our two patients refused systemic antifungal drugs concerning the potential side effects. Unfortunately, they only attained the final VA of light perception.

Brucellosis is a common endemic disease in pastoral areas. But our patient denied any visits to these areas. It was likely that infection was caused by the intake of dairy products or

meat[13-14]. Because of its epidemic areas were limited, the diagnosis of *Brucella*-induced EE beyond the infected areas was challenging. This patient had a good clinical outcome after vitrectomy and systemic antibiotic treatment, and the final VA eventually recovered to 20/100. *Bacillus licheniformis* was very rarely reported in endophthalmitis, case reports were limited to exogenous ones secondary to penetrating eye trauma or cataract surgery[15-16]. To the best of our knowledge, this patient was the first case of EE caused by *Bacillus licheniformis*. The patient developed a fever after a surgical abortion. Although the body temperature returned to normal after systemic antibiotic treatment, ocular symptoms quickly occurred. The patient had a slight improvement in VA, from counting finger (CF) to 20/1000.

The prognosis of endophthalmitis varies according to the species of pathogenic microorganisms[17-18]. The prognosis of bacterial is often poor. A systematic review revealed that nearly half of the patients reached the final VA worse than 20/200, and 24% of the patients eventually underwent enucleation or evisceration[10]. Gram-negative bacteria, such as *Klebsiella pneumoniae*, had particularly worse prognosis[5]. However, the outcomes of fungal EE were relatively better compared to bacterial EE[3,9,19-20]. In our study, the final VA of 7 eyes (46.7%) with *Candidal* EE attained final VAs of 20/400 or better. However, of the eyes with bacterial EE, four (44.4%) of nine were eviscerated, and only one eye had a final VA better than 20/400. All three patients with *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* infection lost light perception at the end. A former study from northern China reported better VA outcomes with similar fungi/bacteria composition. In their study, 47.6% of the patients achieved the final VA better than 20/400[9], which was higher than that of 28.6% in this study. The initial VA of our patients were different compared to the former study. The proportion of patients whose VA better than 20/400 was 6.7% in our study and 30.4% in the other. The initial VA was an important factor for VA outcomes[7,21].

Early vitrectomy, together with systemic and intravitreal anti-infective agents, is the most effective treatment of EE[1,17-18,22]. The treatment course for bacteria EE was three months in our hospital, and for fungal EE was 4-6mo. Behera analyzed 66 patients with fungal endophthalmitis: 31 patients received early vitrectomy, and 35 patients underwent diagnostic vitrectomy, followed by vitrectomy after positive culture. The final visual outcome of early vitrectomy group was better than the diagnostic vitrectomy group[23]. Yoon observed patients with EE caused by *Klebsiella pneumoniae*, despite systemic and intravitreal antibiotic injection, the infection progressed rapidly until they received vitrectomy[24]. However, for streptococcal endophthalmitis, Kurniawan *et al*[25] found that early vitrectomy within 48h did not seem to change visual outcome[25]. Considering most studies got favorable results, and modern vitrectomy was increasingly less invasive, early vitrectomy should be recommended as the first choice of EE.

The retrospective design of this case study was its main limitation. And we only performed intraocular specimens smear and culture in this study, newer technic like Metagenomic next-generation sequencing was not conducted. And EE was rare than exogenous ones, we only collected 32 eyes of 29 patients in this study, we failed to conduct any statistical analysis. Moreover, we only included patients with positive smear or cultures, suspected EE with negative reports was excluded. This may result in an underestimated result.

In conclusion, the early diagnosis of EE is very challenging. Despite anti-infective treatment and vitrectomy, the overall prognosis is still poor. Fungal EE has a better prognosis than bacterial EE. For patients with uncontrolled diabetes and those receiving glucocorticoid therapy, especially those with potential extraocular infection loci, making the diagnosis of non-infectious uveitis needs to be very careful. Early diagnosis and vitrectomy combined with anti-infective treatment are expected to improve the visual prognosis.

ACKNOWLEDGEMENTS

Authors' contributions: Gan LY analysed and interpreted the data and drafted the manuscript. Ye JJ, Zhou HY, Min HY, and Zheng L, assisted with the acquisition of the data. Gan LY and Ye JJ designed the study. Ye JJ revised the final manuscript. All authors read and approved the final manuscript.

Conflicts of Interest: Gan LY, None; **Ye JJ,** None; **Zhou HY,** None; **Min HY,** None; **Zheng L,** None.

REFERENCES

1 Durand ML. Bacterial and fungal endophthalmitis. *Clin Microbiol Rev* 2017;30(3):597-613.

2 Gounder PA, Hille DM, Khoo YJ, Phagura RS, Chen FK. Endogenous endophthalmitis in Western Australia: a sixteen-year retrospective study. *Retina* 2020;40(5):908-918.

3 Connell PP, O'Neill EC, Fabinyi D, *et al*. Endogenous endophthalmitis: 10-year experience at a tertiary referral centre. *Eye(Lond)* 2011;25(1):66-72.

4 Binder MI, Chua J, Kaiser PK, Procop GW, Isada CM. Endogenous endophthalmitis: an 18-year review of culture-positive cases at a tertiary care center. *Medicine* 2003;82(2):97-105.

5 Cho H, Shin YU, Siegel NH, Yu HG, Sobrin L, Patel A, Durand ML, Miller JW, Husain D. Endogenous endophthalmitis in the American and Korean population: an 8-year retrospective study. *Ocular Immunol Inflamm* 2016:1-8.

6 Chung KS, Kim YK, Song YG, *et al*. Clinical review of endogenous endophthalmitis in Korea: a 14-year review of culture positive cases of two large hospitals. *Yonsei Med J* 2011;52(4):630-634.

7 Muda R, Vayavari V, Subbiah D, Ishak H, Adnan A, Mohamed SO. Endogenous endophthalmitis: a 9-year retrospective study at a

tertiary referral hospital in Malaysia. *J Ophthalmic Inflamm Infect* 2018;8(1):14.

8 Ratra D, Saurabh K, Das D, Nachiappan K, Nagpal A, Rishi E, Bhende P, Sharma T, Gopal L. Endogenous endophthalmitis: a 10-year retrospective study at a tertiary hospital in south India. *Asia Pac J Ophthalmol* (*Phila*) 2015;4(5):286-292.

9 Zhang H, Liu Z. Endogenous endophthalmitis: a 10-year review of culture-positive cases in Northern China. *Ocular Immunol Inflamm* 2010;18(2):133-138.

10 Jackson TL, Paraskevopoulos T, Georgalas I. Systematic review of 342 cases of endogenous bacterial endophthalmitis. *Surv Ophthalmol* 2014;59(6):627-635.

11 Chakrabarti A, Shivaprakash MR, Singh R, Tarai B, George VK, Fomda BA, Gupta A. Fungal endophthalmitis: fourteen years' experience from a center in India. *Retina* 2008;28(10):1400-1407.

12 Amphornphruet A, Silpa-Archa S, Preble JM, Foster CS. Endogenous cryptococcal endophthalmitis in immunocompetent host: case report and review of multimodal imaging findings and treatment. *Ocular Immunol Inflamm* 2018;26(4):518-522.

13 Oray M, Cebeci Z, Kir N, Turgut Ozturk B, Oksuz L, Tugal-Tutkun I. Endogenous *Brucella* endophthalmitis: a case report. *Saudi J Ophthalmol* 2017;31(2):106-108.

14 Al-Kharashi AS. Endogenous endophthalmitis caused by brucella melitensis. *Retin Cases Brief Rep* 2016;10(2):165-167.

15 Padhi TR, Sharma S, Das S, Das T. Bacillus licheniformis as a cause of delayed-onset recurrent pseudophakic endophthalmitis-a rare case report. *Retin Cases Brief Rep* 2012;6(1):43-45.

16 Maucour MF, Brugniart C, Ducasse A, Brasme L, Bajolet O. Bacillary endophthalmitis. Four case reports. *J Fr Ophtalmol* 1999;22(3):371-376.

17 Shao EH, Yates WB, Ho IV, Chang AA, Simunovic MP. Endophthalmitis: changes in presentation, management and the role of early vitrectomy. *Ophthalmol Ther* 2021;10(4):877-890.

18 Chen KJ, Sun MH, Hou CH, *et al*. Susceptibility of bacterial endophthalmitis isolates to vancomycin, ceftazidime, and amikacin. *Sci Rep* 2021;11:15878.

19 Haseeb AA, Elhusseiny AM, Siddiqui MZ, Ahmad KT, Sallam AB. Fungal endophthalmitis: a comprehensive review. *J Fungi* (*Basel*) 2021;7(11):996.

20 Das T, Agarwal M, Anand AR, *et al*. Fungal endophthalmitis: analysis of 730 consecutive eyes from 7 tertiary eye care centers in India. *Ophthalmol Retina* 2022;6(3):243-251.

21 Lim HW, Shin JW, Cho HY, *et al*. Endogenous endophthalmitis in the Korean population: a six-year retrospective study. *Retina* 2014;34(3):592-602.

22 Morris RE, Kuhn F. Complete and early vitrectomy for endophthalmitis. *Eur J Ophthalmol* 2021;31(6):2794-2795.

23 Behera UC, Budhwani M, Das T, Basu S, Padhi TR, Barik MR, Sharma S. Role of early vitrectomy in the treatment of fungal endophthalmitis. *Retina* 2018;38(7):1385-1392.

24 Yoon YH, Lee S, Sohn JH, Lee SE. Result of early vitrectomy for endogenous klebsiella pneumoniae endophthalmitis. *Retina* 2003;23(3):366-370.

25 Kurniawan ED, Rocke JR, Sandhu SS, Allen PJ. Predictors of visual outcome and the role of early vitrectomy in streptococcal endophthalmitis. *Clin Exp Ophthalmol* 2018;46(4):424-431.