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Endogenous Endophthalmitis Due to Streptococcus agalactiae With a Suspected Urinary Source in a Patient Without Systemic Symptoms at Presentation: A Case Report

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Abstract

Group B *Streptococcus* (GBS, *Streptococcus agalactiae*) increasingly causes invasive infection in older adults, and endogenous endophthalmitis due to GBS carries a poor visual prognosis. Urinary tract infection in older adults may present without urinary or systemic symptoms, so the source is difficult to recognize when endophthalmitis is the presenting feature. An 82-year-old man with diabetes mellitus and a bladder tumor under evaluation developed blurred vision and pain in the left eye. Uveitis was initially suspected at a referring hospital; hypopyon appeared the next day, intravitreal vancomycin 1.0 mg and ceftazidime 2.0 mg were given, and he was referred. At presentation, visual acuity was light perception and intraocular pressure was 48 mmHg, with marked corneal opacity, hypopyon, and only mild vitreous opacity on B-scan ultrasonography. He was afebrile without urinary symptoms, yet the white blood cell count was 12,600/μL, C-reactive protein was 7.75 mg/dL, and procalcitonin was 1.05 ng/mL; shivering appeared about 12 hours later. Lens extraction and pars plana vitrectomy were performed the same day. Aqueous humor smear showed phagocytosed chain-forming cocci and culture grew *S. agalactiae*; the undiluted vitreous specimen, taken about 24 hours after the intravitreal injection, was smear- and culture-negative. Midstream urine grew *S. agalactiae* at 10^5 CFU/mL or more with pyuria, and the two isolates had identical susceptibility profiles across 11 agents. Blood cultures were not obtained at presentation. CT showed focal areas of reduced parenchymal enhancement in the right kidney; echocardiography showed no findings supporting infective endocarditis. Systemic therapy totaled 46 days, with two intracameral injections of vancomycin and ceftazidime. An iatrogenic retinal break occurred during the initial surgery, and silicone oil tamponade was required at a second vitrectomy. Two months later, the eye was free of pain, inflammation, and recurrence, but the retina remained detached and visual acuity was no light perception. No evidence of an exogenous source was identified; bacteremia was not documented, and the urinary source remained a presumption. The case shows how narrow the window is in which the route and source of infection can still be established microbiologically.

Categories: Urology, Ophthalmology, Infectious Disease
Keywords: blood culture, endogenous endophthalmitis, group b streptococcus, streptococcus agalactiae, urinary tract infection

Introduction

Endogenous bacterial endophthalmitis is uncommon, but the visual prognosis is poor and ocular symptoms may be the first manifestation of systemic infection. It arises when circulating organisms cross the blood-ocular barrier, usually seeding the richly perfused choroid before infection extends into the vitreous cavity and anterior segment.

Group B *Streptococcus* (GBS) is best known as a neonatal pathogen but increasingly causes invasive disease in older adults: in population-based surveillance of nonpregnant adults in the United States, the incidence rose from 8.1 to 10.9 per 100,000 between 2008 and 2016, and 95% of patients had at least one underlying condition, most often obesity or diabetes mellitus [1]. Endogenous endophthalmitis due to GBS is rare. Yoshida et al. reviewed 53 eyes of 41 patients: 26 (60%) of the 43 eyes with available outcomes reached blindness or death, infective endocarditis was the most frequent source (37%), and urinary tract infection accounted for 15% [2]. A patient with bilateral subretinal abscesses died three days after treatment began [3], so survival as well as vision may be at stake.

Urinary tract infection is among the systemic infections most often accompanying endogenous endophthalmitis, with pneumonia and infective endocarditis [4], yet it may be clinically silent: among 285 patients with bacteremic urinary tract infection (median age: 82 years), 50.5% had no urinary symptoms or signs at the initial contact [5].

We report endophthalmitis in an older man without systemic symptoms at presentation, in whom

Streptococcus agalactiae was isolated from both aqueous humor and urine. We separate three judgments, i.e., identification of the organism, determination of the route, and presumption of the source, and report how far each could be taken. The case illustrates that when endophthalmitis is the presenting feature, the window in which the route and the source can still be established microbiologically is narrow and easily lost.

Case Presentation

Day zero is the day of presentation to our department. An 82-year-old man had diabetes mellitus (HbA1c: 6.9%), hypertension, bilateral cataracts, urolithiasis, and benign prostatic hyperplasia. A bladder tumor was under evaluation and transurethral resection was scheduled; no urinary tract instrumentation had been performed. Four days before presentation, he noticed blurred vision and pain in the left eye. The referring ophthalmologist suspected uveitis two days before presentation and started topical steroid and levofloxacin. The day before presentation, hypopyon appeared; intravitreal vancomycin 1.0 mg and ceftazidime 2.0 mg were given with fortified topical vancomycin and ceftazidime, and he was referred the next day.

Findings at presentation

Best-corrected visual acuity was 20/20 in the right eye and light perception in the left; intraocular pressure was 16 and 48 mmHg. The right eye was normal (Figure *1A*). No corneal or scleral wound, ulcer, or infiltrate was present; the Seidel test was negative, and CT showed no intraocular foreign body; there was no history of intraocular surgery and no reported ocular trauma. The left eye showed ciliary injection, corneal edema, cataract, and hypopyon (Figure *1B*), with marked corneal opacity precluding any fundus view; B-scan ultrasonography showed only mild vitreous opacity, without retinal detachment or choroidal thickening (Figure *2*).

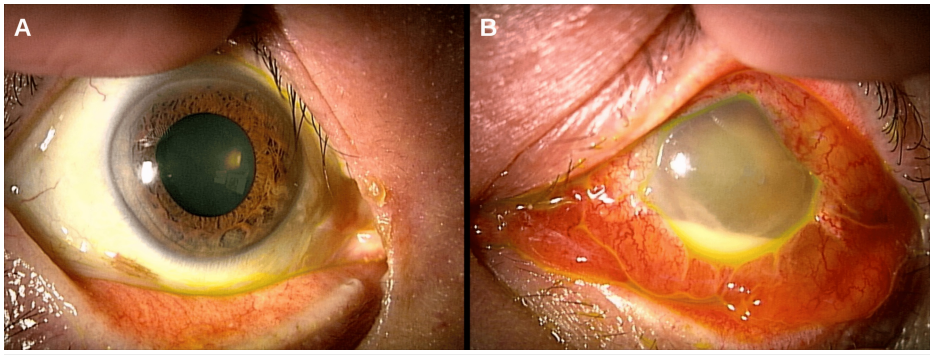


FIGURE 1: Anterior segment photographs at presentation.

(A) Right eye. Cataract is present, without inflammatory findings. (B) Left eye. Marked ciliary injection, corneal opacity, and hypopyon are seen.

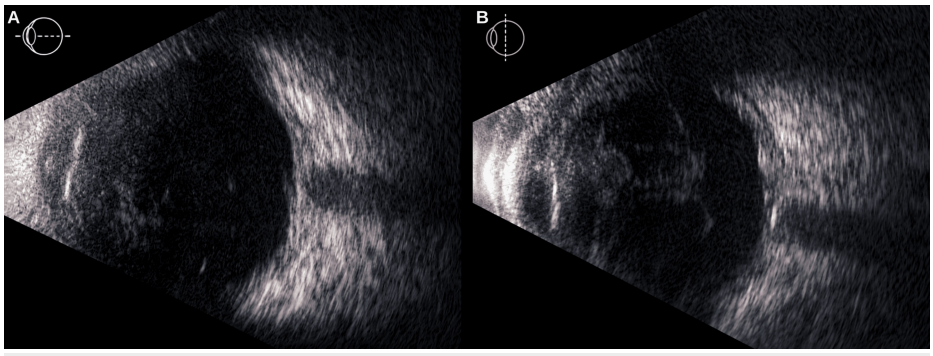


FIGURE 2: B-scan ultrasonography of the left eye at presentation.

(A) Transverse scan. (B) Longitudinal scan. Only mild vitreous opacity is seen, without retinal detachment or choroidal thickening.

He had no systemic symptoms, was afebrile, and had no back pain, dysuria, or costovertebral angle tenderness. Laboratory studies nevertheless showed a systemic inflammatory response: white blood cell count was 12,600/μL, C-reactive protein was 7.75 mg/dL, erythrocyte sedimentation rate was 33 mm at 60 minutes, and procalcitonin was 1.05 ng/mL, with negative beta-D-glucan and endotoxin. Shivering appeared

about 12 hours after presentation, at an axillary temperature of 37.0°C.

Microbiological and systemic evaluation

Aqueous humor, vitreous, and midstream urine were obtained on day zero, all before the first systemic dose (Table 1). Both the aqueous humor and the urine grew *S. agalactiae*, with pyuria, whereas the vitreous was smear- and culture-negative. The two isolates had identical susceptibility profiles across the 11 agents tested (Table 2) and were not preserved. Blood cultures were not obtained at presentation; three sets taken on day six, after four days of penicillin G, showed no growth.

Specimen	Collection	Dipstick	Smear	Culture
Aqueous humor	Day 0 (initial surgery)	—	Leukocytes 3+, chain-forming cocci 3+, phagocytosis present	*Streptococcus agalactiae*, 2+
Vitreous	Day 0 (initial surgery; undiluted, before infusion; ~24 hours after intravitreal antibiotics)	—	Leukocytes 2+, no organisms seen	No growth after 1 week
Midstream urine	Day 0	Leukocyte esterase 3+, nitrite negative	Chain-forming cocci 2+	*Streptococcus agalactiae*, ≥10^5 CFU/mL
Blood	At presentation	—	—	Not performed
Blood	Day 6 (3 sets, during penicillin G)	—	—	No growth after 1 week

TABLE 1: Microbiological results by specimen.

All specimens on day zero were collected before the first dose of systemic antimicrobial therapy. Intraocular specimens were submitted for urgent Gram staining. “Not performed” indicates that the test was not carried out and is distinct from a negative result. The isolates were not preserved.

Antimicrobial agent	Aqueous humor MIC (µg/mL)	Reported S/R	Urine MIC (µg/mL)	Reported S/R
Penicillin G	≤0.06	S	≤0.06	S
Cefotaxime	≤0.06	S	≤0.06	S
Ceftriaxone	≤0.06	S	≤0.06	S
Imipenem/Cilastatin	≤0.06	S	≤0.06	S
Clarithromycin	≥16	R	≥16	R
Clindamycin	≥16	R	≥16	R
Levofloxacin	≥32	R	≥32	R
Tosufloxacin	≥32	R	≥32	R
Minocycline	≥16	R	≥16	R
Chloramphenicol	2	S	2	S
Trimethoprim-sulfamethoxazole	≤0.5	S	≤0.5	S

TABLE 2: Antimicrobial susceptibility of the aqueous humor and urine isolates.

Both isolates were tested in the same clinical laboratory using the same panel. MIC values and S/R categories are reproduced as reported by the laboratory; the interpretive standard and agent-specific breakpoints could not be verified retrospectively. Vancomycin and ceftazidime were not included in the panel. An identical susceptibility pattern does not indicate strain identity; molecular typing was not performed, and the isolates were not preserved.

MIC: minimum inhibitory concentration; S: susceptible; R: resistant

CT showed a rim-enhancing preseptal fluid collection anterior to the left globe, considered to represent extension of the intraocular inflammation rather than a discrete abscess (Figure *3A*), and extraocularly the known bladder tumor, bladder wall enhancement, benign prostatic hyperplasia, and focal areas of reduced parenchymal enhancement in the right kidney (Figures *3B*-*3D*). Transthoracic and transesophageal echocardiography, on days one and seven, showed no findings suggestive of infective endocarditis.

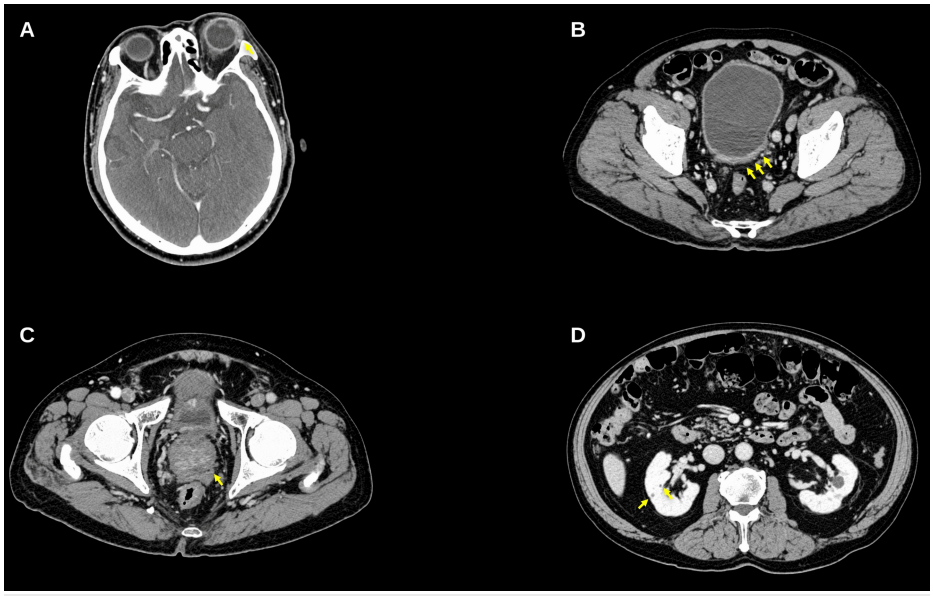


FIGURE 3: Contrast-enhanced whole-body computed tomography at presentation.

(A) Orbital level. A rim-enhancing preseptal fluid collection is seen anterior to the left globe (yellow arrow), considered to represent extension of the intraocular inflammation. No intraocular foreign body is present. (B) Enhancement of the bladder wall (yellow arrows). (C) Benign prostatic hyperplasia (yellow arrow). (D) Focal area of reduced parenchymal enhancement in the kidney (yellow arrows).

Management and outcome

Lens extraction and pars plana vitrectomy were performed on day zero with irrigating solution containing vancomycin 20 µg/mL and ceftazidime 40 µg/mL, so no intravitreal injection was given at the end of surgery. The lens was removed with its capsule, and no intraocular lens was implanted. Retinal hemorrhages were present circumferentially. An iatrogenic retinal break occurred inferiorly, with retinal detachment. Corneal opacity prevented adequate visualization of the inferior periphery, and the break margins could not be identified with sufficient confidence for accurate retinopexy, so treatment was deferred rather than attempted under inadequate visualization; no alternative retinopexy such as transconjunctival cryotherapy, and no adjunct such as perfluorocarbon liquid, was used. Subretinal fluid was drained and fluid-air exchange performed.

Because intraocular inflammation improved insufficiently, vancomycin 1.0 mg and ceftazidime 2.0 mg were injected separately into the anterior chamber on days three and seven. The intracameral route was chosen because the lens, its capsule, and the vitreous had all been removed, so the anterior chamber and vitreous cavity formed a single continuous space; a limbal paracentesis reached that space without a further pars plana entry and kept the needle tip under direct view in an eye whose fundus could not be seen. No further intravitreal or subconjunctival injection was given. Systemic therapy (Table *3*) totaled 46 days, ending with oral amoxicillin through day 45, following the standard four- to six-week course used for GBS bacteremia.

Day	Route	Agent and dose
−2 (referring hospital)	Topical	Steroid; levofloxacin 1.5%, four times daily, for one day
−1 (referring hospital)	Intravitreal injection	Vancomycin 1.0 mg (0.1 mL), ceftazidime 2.0 mg (0.1 mL)
−1 (referring hospital)	Topical	Vancomycin and ceftazidime six times daily for one day; atropine
0	Added to irrigating solution	Vancomycin 10 mg/500 mL BSS (20 µg/mL), ceftazidime 20 mg/500 mL BSS (40 µg/mL)
0	Systemic (intravenous)	Ceftriaxone 2 g, given after all specimens had been collected
1–2	Systemic (intravenous)	Ampicillin/sulbactam 3 g every 6 hours
2–24	Systemic (intravenous)	Penicillin G 24 million units/day (4 million units every 4 hours); changed on the day susceptibility results became available
3	Intracameral injection	Vancomycin 1.0 mg (10 mg/mL, 0.1 mL), ceftazidime 2.0 mg (20 mg/mL, 0.1 mL); injected separately
7	Intracameral injection	Same as above
8	Added to irrigating solution	Same as initial surgery
25–45	Systemic (oral)	Amoxicillin 1,500 mg/day (500 mg every 8 hours)

TABLE 3: Treatment timeline.

Day zero is the day of presentation to our department. Intravitreal injection was given once, at the referring hospital on day −1; no further intravitreal injection was given at our institution, and subconjunctival injection was not performed at any point. Because the lens and its capsule were removed and vitrectomy performed at the initial surgery, the anterior chamber and the vitreous cavity formed a single continuous space. Vancomycin and ceftazidime were injected separately to avoid incompatibility. The total duration of systemic antimicrobial therapy was 46 days.

BSS: balanced salt solution

At a second vitrectomy on day eight, fibrin was removed and residual vitreous excised. Photocoagulation of the inferior break was attempted, but bleeding from the inferior iridectomy (Ando iridectomy) could not be controlled, and the procedure was terminated with silicone oil tamponade. Visual acuity was light perception at presentation and no light perception two months later, assessed in a darkened room with the fellow eye occluded; no acuity was recorded between these points. Intraocular pressure was 4 mmHg, with no pain and no anterior segment inflammation, and the retina remained detached under silicone oil.

Discussion

Presumption of the source and its limits

Ocular symptoms were the first manifestation of systemic infection. The combination that should prompt suspicion is unilateral hypopyon with markedly raised intraocular pressure and only mild vitreous opacity in an older adult who has a systemic inflammatory response but no localizing symptoms. Shivering appeared about 12 hours after presentation at 37.0°C, below the threshold for fever; we interpreted this as the emergence of systemic infection not apparent earlier, although timing alone does not establish its cause.

Findings supporting a urinary source were isolation of the same species from urine and aqueous humor, identical susceptibility profiles, definite pyuria, structural urinary tract abnormalities, and focal areas of reduced parenchymal enhancement in the right kidney. The renal finding raises the possibility of parenchymal involvement and is not explained by colonization alone, but it is not specific, as focal reduced enhancement also follows infarction, scarring, and obstruction, and no formal radiological diagnosis of pyelonephritis was made. The bladder wall enhancement cannot be attributed to infection, because a bladder tumor was under evaluation at that site.

None of this establishes the urinary tract as the source. Bacteremia was never documented, and the negative day six cultures followed four days of penicillin G. An identical susceptibility pattern does not establish strain identity, which requires molecular typing, and the isolates were not preserved. Coexisting asymptomatic bacteriuria cannot be excluded, as that entity is defined irrespective of pyuria and turns on the absence of attributable symptoms [6], which this patient lacked, and separating infection from colonization in GBS bacteriuria in nonpregnant adults is difficult [7]. Two interpretations therefore remain: that the urinary tract was the source of hematogenous dissemination, or that asymptomatic bacteriuria was incidental and the source lay elsewhere.

A self-sealing microperforation or a poorly visualized non-metallic foreign body cannot be excluded absolutely, but no exogenous source was identified by conventional criteria, and mild vitreous opacity is not itself grounds for suspecting one, as the distribution of intraocular inflammation does not determine the route.

Interpretation of the culture results

The negative vitreous smear and culture are primarily attributable to the intravitreal antibiotics given the day before referral. Gan et al. found the first vitreous samples culture-positive in 20 of 29 (69%) patients, whereas second samples taken after intravitreal antibiotics were negative in every case [8]; ours was taken about 24 hours after injection. Other explanations do not apply, as the specimen was undiluted and obtained before infusion and before systemic therapy, and both intraocular specimens were equally exposed to topical agents. Regan et al. reported intraocular culture positivity falling to zero once systemic antibiotics had been started [9], and prior intravitreal antibiotics appear to act analogously.

The absence of organisms on the vitreous smear may additionally reflect a lower posterior organism burden. Why the aqueous humor nevertheless remained smear- and culture-positive was not established. At the time of that injection, the eye was still phakic with an intact vitreous, and in the corresponding nonhuman primate model the same doses produced maximum aqueous concentrations of 151.4 µg/mL for vancomycin and 64.6 µg/mL for ceftazidime, with half-lives of 29.4 and 20.4 hours [10]. A gradient and a finite half-life therefore exist, but the anterior chamber vancomycin concentration is high in absolute terms and well above concentrations usually inhibitory for GBS, so lower anterior exposure does not by itself explain the discrepancy. The preceding intravitreal antibiotics are therefore the principal consideration in interpreting the negative vitreous microbiology, with a lower posterior-segment organism burden as a possible additional contributor; because neither intraocular drug concentrations nor organism burden was measured, the relative contribution of the two cannot be determined, and neither mechanism alone provides a complete explanation. Practically, when intraocular antibiotics have already been given elsewhere, the aqueous humor may still yield the organism when the vitreous does not.

Local and systemic treatment

Intraocular drug concentrations were not measured, and anatomical continuity alone does not establish equivalence of drug distribution, posterior-segment exposure, duration of therapeutic concentration, or therapeutic effect between the intracameral and intravitreal routes. Anterior segment safety was not assessed directly, as specular microscopy was not performed. In vitro, vancomycin reduces the viability of cultured human corneal endothelial cells only above 5.0 mg/mL [11], whereas 1.0 mg distributed through the continuous anterior chamber and vitreous cavity corresponds to about 0.2 mg/mL after mixing; consistent with this, the same doses in vitrectomized aphakic macaque eyes produced maximum aqueous concentrations of 205.6 µg/mL and 260.0 µg/mL without change in corneal endothelial cell density at one month [10]. Those data came from an animal model after intravitreal rather than intracameral injection, the agent tested in vitro alongside vancomycin was cefuroxime rather than ceftazidime, and the transient endothelial concentration after a limbal injection would have been higher and was not measured.

Systemic therapy was continued for 46 days, following the standard four- to six-week course for GBS bacteremia and reflecting that infective endocarditis, the most frequently reported source in GBS endophthalmitis [2], could not be definitively excluded: echocardiography showed no supporting findings, but the transesophageal study followed five days of high-dose penicillin G, and because pretreatment blood cultures were lacking, one major criterion of the modified Duke criteria could not be evaluated. No optimal duration has been established for endogenous endophthalmitis, and a negative blood culture obtained during therapy does not exclude bacteremia.

Outcome and limitations

Infection was controlled, but the retina did not reattach, and visual function was not recovered. Infection-related factors included severe endophthalmitis and an intraocular pressure of 48 mmHg at presentation, together with the circumferential retinal hemorrhage; surgery-related factors were the iatrogenic break and the resulting detachment, which could not be treated at either operation. Hypotony of 4 mmHg and the persistent detachment at the final examination may also have contributed. The detachment was absent at presentation and arose from a single iatrogenic break, so the anatomical outcome is not a consequence of the endophthalmitis itself; no finding demonstrating the mechanism of visual loss was obtained, and the

contributions of these factors cannot be separated. A case has been reported in which the systemic condition improved while the affected eye progressed to phthisis bulbi [12], illustrating that control of infection, anatomical reattachment, and recovery of vision are distinct outcomes.

This single case demonstrates neither treatment efficacy nor causal relationships. Blood cultures were not obtained at presentation, so direct evidence of bacteremia and of the route of dissemination is lacking, and the isolates were not preserved. An iatrogenic retinal break led to retinal detachment, so infection-related and surgery-related contributions to the outcomes cannot be distinguished. Specular microscopy was not performed, so a direct effect of the intracameral injections on the corneal endothelium can neither be demonstrated nor excluded. No visual acuity was recorded between presentation and two months, so the temporal course of visual decline could not be determined.

Conclusions

Aqueous humor culture established endophthalmitis caused by *S. agalactiae*, and systematic assessment at presentation identified no evidence of an exogenous source. A urinary source remains a presumption: isolation of the same species from urine and the identical susceptibility profiles provide grounds for suspicion but establish neither strain identity, nor bacteremia, nor the direction of dissemination. Once endogenous endophthalmitis is suspected, blood cultures should be obtained before antimicrobial therapy is started. A positive result establishes bloodstream infection and supports a hematogenous route, but does not by itself identify the primary focus, which requires concordant isolation from the suspected site and, ideally, demonstration of strain identity. In the present case, this opportunity was missed, and both the route and the source of infection remained unconfirmed by microbiological evidence. Infection was controlled, but neither anatomical reattachment nor recovery of visual function was achieved. In severe GBS endophthalmitis, control of infection and recovery of visual function may follow distinct courses.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ryusuke Hashimoto, Kazusa Saito, Takayuki Baba

Acquisition, analysis, or interpretation of data: Ryusuke Hashimoto, Kazusa Saito, Takayuki Baba

Critical review of the manuscript for important intellectual content: Ryusuke Hashimoto, Kazusa Saito, Takayuki Baba

Drafting of the manuscript: Kazusa Saito

Supervision: Takayuki Baba

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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