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ORIGINAL ARTICLE

Sequential Inflammatory and Pupillary-Block Ocular Hypertension in Streptococcus suis Endogenous Endophthalmitis: An Observational Analytic Case Study

Dewa Ayu Ina Dianata[1], I Gusti Ayu Made Juliari[2*], I Gusti Ayu Ratna Suryaningrum[2], Ni Made Ari Suryathi[2], Anak Agung Ayu Suryapraba Indradewi[3]

[1] Ophthalmology Study Program, Faculty of Medicine, Universitas Udayana, Denpasar, Indonesia
[2] Department of Ophthalmology, Faculty of Medicine, Universitas Udayana/Ngoerah Hospital, Denpasar, Indonesia
[3] Department of Neurology, Faculty of Medicine, Universitas Udayana/Ngoerah Hospital, Denpasar, Indonesia

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**Corresponding author:*
I Gusti Ayu Made Juliari
arie_mata@yahoo.com
ORCID: 0009-0004-1610-360X

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ABSTRACT

Background: Streptococcus suis, a zoonosis acquired from pigs and raw pork, is a rare cause of endogenous endophthalmitis, and no quantitative account of the pressure complications of such an eye has been published.

Objective: To characterize, in a single culture-proven case of Streptococcus suis endogenous endophthalmitis, the two mechanisms and the course of the raised intraocular pressure, and to derive the clinical implications for management.

Methods: a structured analysis of one culture-proven case managed at a tertiary hospital in Bali, Indonesia, over 48 days. Acuity was converted to logMAR on four published scales, all 19 pressure readings were analysed with a repeatability model, and corticosteroid causation was scored with the Naranjo instrument.

Results: a 46-year-old man who had eaten raw pork a week earlier presented with meningitis, reduced hearing and a left eye with light perception, hypopyon and vitritis. Cerebrospinal fluid and vitreous both grew S. suis. Pressure rose in two distinct episodes: an inflammatory one peaking at 29.0 mmHg on day 7, and a pupillary block from posterior synechiae with iris bombé peaking at 30.0 mmHg on day 16, which fell 11.0 mmHg after iridotomy with simultaneous reintroduction of timolol. Corticosteroid causation scored 3, a value largely fixed by the temporal sequence alone. Acuity improved by 0.70 logMAR overall, but the division of that gain across the vitrectomy reversed with the conversion scale: 0.40 before and 0.30 after by clinical convention against 0.10 and 0.60 with measured hand-movement values. The eye remained blind.

Conclusion: raised pressure in such eyes is not one entity, the pupil must be examined before pressure-lowering therapy is escalated, and a logMAR change at this level of vision is uninterpretable unless its scale is stated.

1. Introduction

Endogenous endophthalmitis is an intraocular infection established when micro-organisms reach the eye through the bloodstream rather than through a surgical or traumatic breach of the globe. It accounts for a small minority of all endophthalmitis and carries a worse visual prognosis than the postoperative form, because the diagnosis is frequently delayed and because the patient is by definition systemically unwell[1-3]. A national inpatient analysis of 8,255 admissions in the United States found a bacterial cause in 93.2 per cent[2], and contemporary tertiary series continue to report a poor visual prognosis irrespective of pathogen, final acuity worse than 20/400 in about half of affected eyes, and loss of the globe to evisceration, enucleation or phthisis in a minority[3-6]. In Indonesia, where population-based survey data place the prevalence of blindness at 3.0 per cent in adults aged 50 years and over[7], tertiary ophthalmic and vitreoretinal services are concentrated in a small number of teaching hospitals, so a patient presenting to a district facility with a red, painful eye and a febrile illness may pass through more than one institution before the two problems are recognised as one; that was the pathway this patient took. Prof. Dr. I.G.N.G. Ngoerah General Hospital, the teaching hospital of Udayana University in Denpasar, is the referral endpoint of that pathway for Bali and the eastern Indonesian provinces, and it is one of the few centres in the region where an ophthalmologist, a glaucoma subspecialist, a vitreoretinal surgeon and a neurologist can assess the same patient on the same day.

Streptococcus suis is a Gram-positive, facultatively anaerobic zoonotic pathogen whose natural reservoir is the pig[8,9]. Human infection follows contact with pigs or pork products, or the consumption of raw or undercooked pork, and is reported overwhelmingly from East and Southeast Asia[10,11]. Meningitis is the dominant clinical presentation, and sensorineural hearing loss is its characteristic and often permanent

sequela; septicaemia, endocarditis, arthritis and, rarely, ocular involvement are also described[8,12,13]. The organism carries a capsular polysaccharide that resists phagocytosis and a cholesterol-dependent cytolysin, suilysin, which together permit survival in blood and passage across the blood-brain barrier[14]. The same properties provide a plausible route across the blood-ocular barrier, and it is this shared mechanism, rather than any ocular tropism peculiar to the organism, that makes concurrent meningitis and endophthalmitis biologically coherent[15,16]. For a Balinese population the exposure is not exotic: lawar merah, a ceremonial dish of minced pork, vegetables and fresh pork blood, is prepared and eaten raw, and a large cluster of human *S. suis* infection has been documented on the island in association with such preparations[17].

Ocular involvement by *S. suis* is rare, and the published ophthalmic experience consists of isolated reports rather than series. Endogenous endophthalmitis complicating *S. suis* meningitis has been described in a patient managed in Vietnam[18], and a severe endophthalmitis with bilateral deafness has been reported with genomic characterisation of the causative sequence type[19]. There is no cohort, registry or comparative study of ocular *S. suis* disease, so every statement about its natural history rests on case-level evidence. Beyond that, the ophthalmic literature on this organism is essentially a description of what the eye looks like, and it contains almost no quantitative outcome data. That gap matters because the decisions made in such an eye are quantitative decisions. Whether to give a further intravitreal dose, whether to escalate pressure-lowering therapy, and above all whether and when to perform a vitrectomy, all depend on measuring change rather than on describing appearance[20-22].

Raised intraocular pressure during active endophthalmitis is a particular difficulty. At least three mechanisms compete. Inflammatory cells, fibrin and proteinaceous debris obstruct aqueous outflow at the trabecular meshwork; corticosteroids given to control the inflammation may themselves raise pressure by remodelling the trabecular extracellular matrix and reducing outflow facility; and a pupil sealed by posterior synechiae through 360 degrees produces iris bombé and a secondary angle closure that no topical drug will relieve[23-27]. These three mechanisms demand three different responses, and distinguishing between them at the bedside in an eye whose cornea is hazy is one of the genuinely hard problems in the management of endophthalmitis. Published case reports rarely attempt the distinction, and when they do they seldom set out the evidence for and against each candidate in a form a reader can check.

A conventional case report would describe this patient's course and stop. What is missing from the literature is not another description but a demonstration that a single case can be interrogated analytically: that its acuities can be placed on a named and defensible conversion scale, that its pressure readings can be given a measurement-error model and an exposure metric, that a drug-causation hypothesis can be scored with a validated instrument rather than asserted, and that the boundary between what one case can and cannot establish can be stated in numbers rather than in hedging[28-30]. This study therefore had four aims: to document, using a structured protocol, the ophthalmic and neurological findings in a culture-proven case of *S. suis* endogenous endophthalmitis managed jointly at a tertiary Indonesian centre; to quantify the visual and intraocular-pressure outcomes against published clinical-significance thresholds and to test whether those conclusions survive a change of conversion scale; to attribute the raised pressure among its competing mechanisms using an explicit evidence table and a validated causality instrument; and to state, quantitatively, the limits of inference available from a single case.

2. Methods

Study design

An observational analytic case study was conducted in a single patient managed at a tertiary referral centre. Clinical observations were extracted from the treating teams' contemporaneous records and analysed within the measurement framework set out below, comprising conversion of categorical low-vision acuity to logMAR, a repeatability model for tonometry, evaluation of outcomes against published clinical-significance thresholds, structured assessment of the competing mechanisms of raised intraocular pressure, and quantification of the inferential limits of a single observation. The unit of analysis was one eye of one patient, followed for 48 days from the onset of systemic symptoms to the final recorded assessment two weeks after vitrectomy. Reporting follows the CARE guideline for case reports[28], whose consensus approach has since been extended by later reporting standards for case-based work[31]. The study corresponds to Level 4 in the Oxford 2011 hierarchy and to level 4.b in the Joanna Briggs Institute hierarchy of evidence for effectiveness[29,32,33].

Setting and case identification

The study was conducted in the Department of Ophthalmology, in collaboration with the Department of Neurology, at Prof. Dr. I.G.N.G. Ngoerah General Hospital, the teaching hospital of Udayana University, Denpasar, Bali, Indonesia. The hospital is the tertiary referral centre for Bali and receives referrals from the provinces of West and East Nusa Tenggara. The patient was the only culture-proven case of ocular *Streptococcus suis* infection managed by the department. No screening of a defined population was undertaken and no denominator is available.

Ethical approval and consent

The study was approved by the Research Ethics Committee of CMHC Research Center, Indonesia

(Approval number 2026/207). Written informed consent for publication of clinical details was obtained from the patient after recovery from the acute illness. The study was conducted in accordance with the Declaration of Helsinki. To limit identifiability, no admission date is reported, the dietary exposure is not linked to a datable occasion, and no photograph of the patient is reproduced; the ocular illustrations are schematic reconstructions drawn to a fixed geometry from the recorded slit-lamp and ultrasonographic descriptions.

Data sources and derived variables

All clinical observations were transcribed from the contemporaneous inpatient and outpatient records and are set out with their analytic annotations in Table 1. The following were added during analysis and do not appear in those records: the ICD-10 codes; the conversion scales, clinical-significance thresholds, repeatability model, causality instrument and evidence hierarchy, all selected after the clinical course had closed; the 10 to 21 mmHg reference band and the laboratory reference ranges, which are conventional adult values; and the doses of intravitreal vancomycin and ceftazidime, the record documenting only the agents administered. Two time scales appear in the source documentation, hospital days in the narrative and days from symptom onset in the treating team's timeline. These were reconciled by anchoring hospital day 1 to day 4 from symptom onset and the final assessment to day 48, both as documented, and the reconciled scale is the one plotted in Figure 1. The two accounts differ on the interval from ocular symptom onset to first ophthalmic assessment, and the full range of 1 to 5 days is reported.

Ophthalmic assessment

The left eye was examined at each documented visit by an ophthalmologist; the right eye is documented once, as normal at presentation. Visual acuity was recorded in the Indonesian convention and converted as described below. Intraocular pressure was measured on 11 occasions, 4 of them in triplicate; the tonometer is not identified in the record, and measured pressure depends on the instrument used and on corneal thickness and biomechanics[34,35]. Anterior-segment findings were documented by slit-lamp biomicroscopy. The posterior segment was assessed by B-scan ultrasonography on days 4 and 20, direct visualisation being precluded until day 48 by corneal oedema, hypopyon and dense vitreous opacity; standardised ultrasonography is the established substitute in this situation and carries prognostic as well as diagnostic information[1,36]. The serial anterior-segment appearance and the two ultrasound examinations are reconstructed in Figure 2. Coverage of the assessment framework is reported in full in Table 2: of its 21 parameters, 6 were obtained, 12 were not obtained and 3 were not indicated. Parameters not obtained included assessment of a relative afferent pupillary defect, gonioscopy, anterior-segment optical coherence tomography or ultrasound biomicroscopy, optical coherence tomography of the peripapillary retinal nerve fibre layer, automated perimetry, serial assessment of light projection, central corneal thickness, and description of the optic disc after it became visible on day 48. No measure of optic-nerve structure or function was therefore available at any point during the illness.

Neurological assessment

Neurological evaluation was performed by the attending neurologist, who established the diagnosis of bacterial meningitis, performed lumbar puncture, directed systemic antimicrobial and corticosteroid therapy and assessed hearing clinically[15,16]. Cerebrospinal fluid was examined by the Pandy and Nonne reactions, Gram stain and culture, and the findings are tabulated with their reference ranges in Table 3. Opening pressure, cell count, protein and glucose were not recorded, and no inference regarding intracranial pressure is drawn; each of those parameters carries independent diagnostic weight in adult bacterial meningitis[37,38]. Neuroimaging of the brain and orbit was not performed. Pure-tone audiometry was not performed, and the hearing deficit is therefore described rather than quantified[39,40]. Doses, routes and taper schedules of systemic ceftriaxone and dexamethasone are not recorded.

Conversion of visual acuity to logMAR

Acuity remained below the level at which a letter chart can be used throughout the illness, and four conversions were applied; all four are tabulated against each other in Table 4. The clinical-convention scale assigns counting fingers 2.00, hand movement 2.30 and light perception 2.70 logMAR, and is used throughout unless otherwise stated[41]. The psychophysically measured scale substitutes the values obtained with the Freiburg Visual Acuity Test, 1.98 for counting fingers and 2.28 for hand movement, and retains 2.70 for light perception, which that method cannot measure[42]; it differs from the clinical-convention scale by a constant 0.02 logMAR on the two categories it redefines and is therefore not independent of it. The measured Berkeley scale substitutes the medians obtained with the Berkeley Rudimentary Vision Test in 38 low-vision patients, 2.00 for counting fingers and 2.60 for hand movement[43]; it agrees with the clinical convention at counting fingers and places hand movement 0.30 logMAR worse, and is carried as a full sensitivity analysis because that single difference falls between the two intervals this study compares. The fourth conversion treats the recorded metric fraction as a Snellen fraction, giving 1.78 logMAR for 1/60 and 2.48 for 1/300. In the Indonesian convention 1/60 denotes counting fingers at one metre and 1/300 denotes hand movement, a category rather than a fraction, so this conversion is valid for the former and invalid for the latter. Presenting acuity was light

perception with poor projection, which is functionally worse than light perception with accurate projection and which no published scale distinguishes; 2.70 logMAR is therefore an upper bound on the function of this eye at presentation and every derived improvement a lower bound.

Analysis of the intraocular-pressure record

All 19 recorded readings, grouped into 11 measurement sessions, were analysed; they are plotted individually in Figure 3A. Four of those sessions comprised three readings taken in immediate succession. The within-session standard deviation s_w was estimated from those sessions by equation (1), in which s_i^2 is the variance of session i and k the number of triplicate sessions, giving 0.913 mmHg on 8 degrees of freedom (95% CI 0.617 to 1.749), the interval obtained from the chi-square distribution of the pooled variance[44]. The repeatability coefficient for the difference between two single readings follows from equation (2) and is 2.53 mmHg (95% CI 1.71 to 4.84); the corresponding limit for a single reading compared with the mean of three, which is the comparison this study actually makes, follows from equation (3) and is 2.07 mmHg. Both describe short-term reading variability and contain no between-day, between-observer or diurnal component; time of day was not recorded for any reading, and instrument-to-instrument agreement is itself of this order[45,46]. Exposure to raised pressure was expressed by equation (4) as the area between the session-mean curve P(t) and the 21 mmHg reference limit over the interval in which the curve exceeded it, a cumulative-exposure formulation of the kind used to relate pressure to structural and functional progression[47,48]. Two sessions were recorded on the same day; these were resolved by their mean, by the higher value and by the lower value, and intervals between measurements were interpolated linearly, by last observation carried forward and by next observation carried backward. The range across the nine resulting combinations is reported. No burden statistic was computed for the postoperative hypotony reading, no pressure having been recorded during the 13 days that followed it.

$$s_w = \sqrt{[\ (1/k)\ \Sigma\ s_i^2\ ]} \qquad (1)$$

$$RC = 2.77 \times s_w \qquad (2)$$

$$RC_{mean} = 1.96 \times s_w \times \sqrt{(1 + 1/3)} \qquad (3)$$

$$AUC_{21} = \int [\ P(t) - 21\ ]^{+}\ dt \qquad (4)$$

Clinical-significance thresholds and causality assessment

Change in acuity was evaluated against 0.10 logMAR as the smallest conventionally detectable difference and 0.30 logMAR as a clinically important change; the minimum detectable change for letter-chart acuity has been estimated directly and is of this order[49]. Both thresholds were derived in eyes tested on letter charts; no threshold has been validated for categorical low-vision measurement, and on the clinical-convention scale adjacent low-vision categories are separated by exactly 0.30 logMAR. Intraocular pressure was evaluated against a reference band of 10 to 21 mmHg and a hypotony threshold of 6 mmHg[24,50]. The contribution of corticosteroid to the raised pressure was assessed with the Naranjo adverse-drug-reaction probability scale, a ten-item instrument scored from -4 to 13 in which 1 to 4 indicates a possible, 5 to 8 a probable and 9 or more a definite reaction[51]; its reproducibility between raters and its agreement with other causality instruments are moderate, which is the reason the complete item-level scoring is reported here rather than the total alone[52,53]. It was scored separately for each hypertensive episode, each item was scored explicitly, and the full scoring with its justification appears in Figure 4B. The level of evidence supporting each intervention was assigned using the Oxford 2011 Levels of Evidence[32]. GRADE certainty was not assessed.

Statistical analysis

No inferential test was applied to within-case comparisons, a single eye providing no sampling distribution. Correlation between the ophthalmic and neurological measures was not computed, Spearman's rank correlation being undefined at a sample size of one. Three calculations were performed to quantify the limits of the design: the exact Clopper-Pearson interval for one favourable observation out of one; the sample size at which a two-sided exact binomial test of a single proportion against a known value of 0.20 attains 80 per cent power against 0.40, obtained by enumeration and reported as the smallest size from which power remains at or above 0.80, the power of a discrete test not being monotone in the sample size; and the corresponding two-group requirement by normal approximation. The planning parameters for these calculations are illustrative. Computation was performed in Python 3 using exact integer and rational arithmetic where the quantity permitted it; the analysis script, the verification harness and the derived results file are available from the corresponding author.

3. Results

Presentation and exposure history

A 46-year-old man presented to the ophthalmology service on day 4 of his illness with a one-day history of a cloudy appearance of the left eye. This had been preceded by blurred vision, redness, ocular pain and a whitish discharge from the same eye, and by severe headache, dizziness and reduced hearing which had begun on day 1. He had been admitted to another institution with a provisional diagnosis of meningitis and referred onward. He denied any previous ocular or systemic disease; no systematic screen for diabetes, malignancy or immunosuppression is documented, and an immunocompromised state was suspected clinically but never characterised. The single identified epidemiological exposure was the consumption of pork and of raw lawar merah approximately 7 days before

the onset of symptoms; lawar merah is a traditional Balinese dish of minced pork and vegetables prepared with fresh, uncooked pork blood. Ethnicity was not recorded and is not inferred. The full demographic, exposure and diagnostic profile is set out in Table 1, and the whole 48-day course, with the contributing service named at each milestone, is displayed in Figure 1.

Table 1. Case profile, exposure history, presenting features and recorded diagnoses in a patient with *Streptococcus suis* endogenous endophthalmitis.

Domain	Recorded finding	Analytic note
Demographic and contextual data		
Age and sex	46 years, male	Ethnicity was not recorded and is not inferred here
Setting	Department of Ophthalmology with the Department of Neurology, Prof. Dr. I.G.N.G. Ngoerah General Hospital, Udayana University, Denpasar, Bali, Indonesia	Tertiary referral centre for Bali and the eastern Indonesian provinces
Referral pathway	Admitted elsewhere with a provisional diagnosis of meningitis, then referred	The ocular diagnosis was made only after transfer
Past history	The patient denied any previous ocular or systemic disease	Recorded as a denial. No systematic screen for diabetes, malignancy or immunosuppression is documented, and an immunocompromised state was suspected clinically but never characterised
Dietary exposure	Pork and raw lawar merah, a Balinese dish prepared with fresh pork blood, approximately 7 days before symptom onset	The single identified epidemiological risk factor, consistent with the recognised route for this organism
Presenting symptoms		
Systemic	Severe headache, dizziness, reduced hearing	Onset defined as study day 1
Ocular	Blurred vision, redness, ocular pain, whitish discharge and a cloudy appearance of the left eye	Interval from ocular symptom onset to ophthalmic assessment 1 to 5 days: the narrative and the timeline in the source record disagree, and the five-day reading would place ocular onset before the origin of the day scale
Laterality	Left eye; the right eye was recorded as normal at presentation	Unilateral involvement, in contrast with the bilateral disease in the only comparable published case
Diagnoses as recorded by the treating teams		
Primary ocular diagnosis	Endogenous bacterial endophthalmitis of the left eye due to Streptococcus suis (ICD-10 H44.0)	Culture-proven on vitreous aspirate
Secondary ocular diagnosis	Recorded by the treating team as secondary glaucoma associated with intraocular inflammation, then pupillary block with iris bombé (ICD-10 H40.5)	No optic-nerve data of any kind exist for this eye, so the evidence presented supports secondary ocular hypertension rather than glaucoma; the recorded code is reproduced as recorded
Systemic diagnosis	Bacterial meningitis due to Streptococcus suis (ICD-10 G00.2)	Culture-proven on cerebrospinal fluid
Associated diagnosis	Suspected sensorineural hearing loss (ICD-10 H90.5)	Formal audiometry was not documented, so the deficit is described but not quantified
Interdisciplinary team		
Ophthalmology	Attending ophthalmologists with the ophthalmology trainee; glaucoma and vitreoretinal subspecialty input	Four of the five authors
Neurology	Attending neurologist, Department of Neurology	Meningitis diagnosis, lumbar puncture, systemic antimicrobial and corticosteroid strategy

The study day scale places day 1 at the onset of systemic symptoms and day 4 at admission to the tertiary centre.

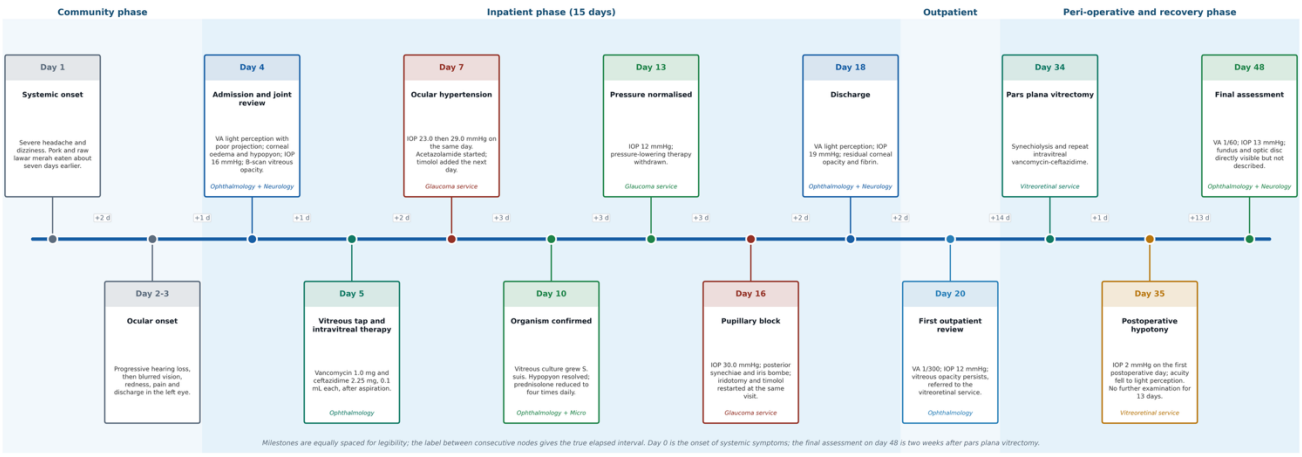

Figure 1. Clinical course and interdisciplinary management of *Streptococcus suis* endogenous endophthalmitis over 48 days. Milestones are equally spaced for legibility and the true interval between consecutive milestones is printed on the axis. Day 1 is the onset of systemic symptoms. The contributing service is named beneath each milestone.

Ophthalmic and neurological findings at presentation

The right eye was recorded as normal at presentation. That single entry is the whole of the record for that eye: no acuity, no pressure and no description of the disc, macula or vessels appears at any visit, and no inference about the optic nerve or about intracranial pressure is drawn from it. In the left eye, visual acuity was light perception with poor projection, which converts to 2.70 logMAR or worse. The eye was markedly congested with a minimal yellowish discharge. The cornea showed haze and oedema and there was a frank hypopyon. The anterior chamber could not be assessed further, and the iris, pupil, lens and posterior segment could not be examined at all, because of the corneal opacity and the intensity of the intraocular inflammation. Intraocular pressure was 16 mmHg. B-scan ultrasonography demonstrated echogenic vitreous opacities of low to moderate reflectivity with moderate mobility, with no retinal detachment demonstrated; ultrasound through dense vitritis does not exclude a shallow or localised detachment, and the two examinations performed are illustrated schematically in Figure 2B. Neurological assessment identified severe headache, dizziness and reduced hearing with tinnitus, and no other deficit was recorded. All of these findings, together with the parameters that were not obtained and the reason in each case, are tabulated by eye in Table 2.

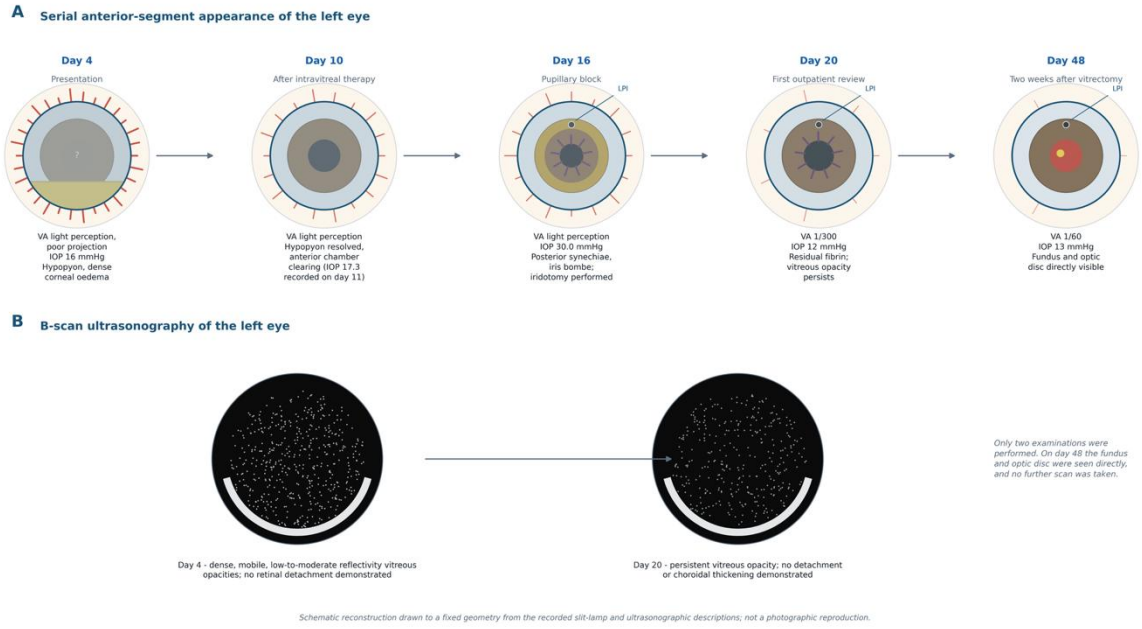

Figure 2. Serial ocular findings in the left eye. (A) Anterior-segment appearance at five time points, from the hypopyon and dense corneal oedema of day 4, through the posterior synechiae and iris bombé of day 16 that produced the second pressure excursion, to the clear media of day 48; the day 4 panel shows the pupil obscured, as it was recorded. (B) The two B-scan examinations that were performed, on days 4 and 20. Both rows are schematic reconstructions drawn to a fixed geometry from the recorded descriptions and are not photographic reproductions.

Table 2. Structured ophthalmic and neurological assessment: findings by eye, and every parameter of the assessment framework that was not obtained, with the reason in each case.

Parameter	Right eye	Left eye	Reference / normal	Interpretation
Findings recorded, from the 6 of the 21 framework parameters that were obtained				
Visual acuity, day 4	Recorded as normal; no acuity value is recorded	Light perception with poor projection (logMAR 2.70 or worse)	0.00 logMAR	ICD-10 category 4 in the affected eye. Poor projection is worse than accurate projection and no scale distinguishes them, so 2.70 is an upper bound on function
Visual acuity, day 20	Not re-recorded	1/300, hand movement equivalent (logMAR 2.30)	0.00 logMAR	An improvement of 0.40 logMAR, all of it before vitrectomy
Visual acuity, day 35	Not re-recorded	Light perception (logMAR 2.70)	0.00 logMAR	A deterioration of 0.40 logMAR on the first postoperative day, which exceeds this study's own threshold for a clinically important change
Visual acuity, day 48	Not re-recorded	1/60, counting fingers at 1 m (logMAR 2.00)	0.00 logMAR	Total change 0.70 logMAR from presentation; ICD-10 category 3, one category better and still blind
Intraocular pressure	Not recorded at any visit	2 to 31 mmHg across 19 readings in 11 sessions	10–21 mmHg	3 sessions above and 1 below the band. The tonometer is not named in the record
Conjunctiva and sclera	Recorded as normal at presentation	Marked congestion with minimal yellowish discharge at presentation, settling to minimal injection by day 48	Quiet	Paralleled the intraocular inflammation
Cornea	Recorded as normal at presentation	Haze and oedema with Descemet folds; residual inferior haze at day 20; near clear at day 48	Clear	The principal obstacle to posterior segment assessment, and an unquantified source of applanation bias
Anterior chamber	Recorded as normal at presentation	Hypopyon at presentation; fibrin; shallow with iris bombé on day 16; residual fibrin at day 20	Deep and quiet	Hypopyon resolved 5 days after intravitreal therapy
Iris and pupil	Recorded as normal at presentation	Not assessable at presentation; posterior synechiae with iris bombé on day 16; laser peripheral iridotomy performed	Round, reactive, no synechiae	The mechanism of the second hypertensive episode
Vitreous and retina (B-scan)	Not indicated	Echogenic opacities of low to moderate reflectivity with moderate mobility on day 4; persistent opacity on day 20; no retinal detachment demonstrated at either examination	Optically empty vitreous, retina attached	Two ultrasound examinations, plus the intraoperative view on day 34 and the fundus view on day 48. Ultrasound through dense vitritis does not exclude a shallow or localised detachment
Fundus, direct view	Recorded only as normal at presentation; the disc was not separately described	Not visible until day 48, when the fundus and optic disc became directly visible	Visible disc and macula	No description of either disc exists in the record, so no statement about the optic nerve or about intracranial pressure rests on this row
Hearing and eighth cranial nerve	-	-	Normal hearing	Reduced hearing with tinnitus, improving with treatment; never quantified
Headache and meningism	-	-	Absent	Severe headache and dizziness at presentation; headache absent by the first outpatient review
Parameters not obtained (12 of the 21) – listed so that the analysis is not				

read as more complete than it is				
Dilated fundus examination	-	Not obtained	-	Not possible until day 48 because of corneal oedema, hypopyon and dense vitreous opacity.
Description of the optic disc once it became visible on day 48	-	Not obtained	-	The record states that the fundus and optic disc were visible but contains no description of colour, margin, cup-disc ratio or pallor. This is the single most consequential omission in the ophthalmic record.
Relative afferent pupillary defect, graded with neutral density filters	-	Not obtained	-	Not performed. It was obtainable: the swinging-flashlight test reads the consensual response of the normal FELLOW pupil and does not require the affected pupil to be visible. This, and not the absence of tomography, is the principal measurement failure of the case.
Gonioscopy	-	Not obtained	-	Not recorded. It is the standard test that separates pupillary block from peripheral anterior synechial closure and confirms that a block has been relieved.
Anterior-segment optical coherence tomography or ultrasound biomicroscopy	-	Not obtained	-	Not recorded. Either images the iris-lens configuration through a hazy cornea when the slit lamp cannot.
Optical coherence tomography of the peripapillary retinal nerve fibre layer	-	Not obtained	-	Not attempted. Media opacity and unsteady fixation precluded it during the acute illness; by day 48 the stated obstacles no longer applied and it was still not attempted.
Automated perimetry (SITA Standard 24–2)	-	Not obtained	-	Not attempted. Fixation and cooperation, rather than acuity as such, are the limiting factors; by day 48 kinetic perimetry with a large target would have been feasible.
Serial assessment of light projection	-	Not obtained	-	Light perception with poor projection is itself a gross field assessment, and it is the only afferent measurement available through opaque media. It was recorded once, on day 4, and never repeated.
Central corneal thickness or pachymetry	-	Not obtained	-	Not recorded, so the direction and size of the applanation bias introduced by corneal oedema cannot be estimated.
Neuroimaging of the brain and orbit	-	Not obtained	-	Not documented in the source record.
Cerebrospinal fluid opening pressure, cell count, protein and glucose	-	Not obtained	-	Not documented. No statement about intracranial pressure can therefore rest on the cerebrospinal fluid.
Formal pure-tone audiometry	-	Not obtained	-	Not documented, although hearing loss is the sequela this organism is known for.

Parameters not indicated (3 of the 21) – a test that was not needed is not a deficiency				
Ishihara colour vision	-	Not indicated	-	Not testable at light-perception acuity.
Hertel exophthalmometry	-	Not indicated	-	No proptosis or orbital signs.
Frisen grading of optic-disc swelling	-	Not indicated	-	The disc of the affected eye was never seen during the acute illness, so no grade is assignable. No papilloedema was demonstrated in either eye.

Visual acuity is converted on the clinical-convention scale defined in the Methods. A dash indicates that the parameter is not eye-specific or was not recorded for that eye. The assessment framework is the authors' own construction and is not a published protocol. The relative afferent pupillary defect is the most consequential of the parameters not obtained: it was obtainable throughout, because the swinging-flashlight test reads the consensual response of the normal fellow pupil.

Laboratory and microbiological findings

A single set of blood tests was taken on day 4. The leucocyte count was 22.17 x 10^3/uL, 2.02 times the upper reference limit, and the platelet count was 119 x 10^3/uL, 31 x 10^3/uL below the lower reference limit. Sodium was 134 mmol/L and chloride 98.2 mmol/L. No serial inflammatory markers were taken, so nothing in this study tracks the inflammation over time. Cerebrospinal fluid examination showed strongly positive Pandy and Nonne reactions, and both Gram-positive and Gram-negative organisms were reported on the smear. Culture of the cerebrospinal fluid grew ***Streptococcus suis***; tuberculous and fungal causes had been considered at presentation and were not pursued once the organism was known. The Gram-negative component was not confirmed on culture; conventional culture, paediatric blood-culture bottles and polymerase chain reaction differ substantially in yield in this setting, and only conventional culture was used here[54]. An over-decolourised or contaminated smear is the commonest explanation and a repeat stain would have settled it at negligible cost, but the record contains nothing that would decide between the possibilities, so the discrepancy is reported in Table 3 rather than resolved. Vitreous aspirate taken on day 5 also grew ***Streptococcus suis***, with the result available on day 10. The isolation of the same organism from the cerebrospinal fluid and from the vitreous strongly supports haematogenous ocular seeding; no blood culture was taken, so the bacteraemic phase itself was never demonstrated and the inference is not established. All laboratory, cerebrospinal fluid and ultrasonographic results are given with their reference ranges in Table 3.

Table 3. Laboratory, cerebrospinal fluid, microbiological and ultrasonographic findings with reference ranges and clinical interpretation

Investigation	Result	Reference range	Interpretation
Haematology and biochemistry (day 4, a single set)			
Leucocyte count	22.17 x 103/uL	4.0–11.0 x 103/uL	Raised; 2.02 times the upper reference limit
Platelet count	119 x 103/uL	150–450 x 103/uL	Low by 31 x 103/uL; consistent with systemic bacterial sepsis
Sodium	134 mmol/L	136–145 mmol/L	Low by 2 mmol/L; recognised in bacterial meningitis
Chloride	98.2 mmol/L	98–107 mmol/L	Within the reference range
Cerebrospinal fluid			
Pandy reaction	Strongly positive	Negative	Raised cerebrospinal fluid globulin
Nonne reaction	Strongly positive	Negative	Raised cerebrospinal fluid globulin
Gram stain	Gram-positive and Gram-negative organisms seen	No organisms	The Gram-negative component was not confirmed on culture. An over-decolourised or contaminated smear is the commonest explanation and a repeat stain would have settled

			it; the discrepancy is reported rather than resolved
Culture	Streptococcus suis	Sterile	Established the systemic diagnosis
Opening pressure, cell count, protein, glucose	Not documented	-	Their absence prevents any statement about intracranial pressure
Ocular microbiology			
Vitreous aspirate culture (day 5 sample)	Streptococcus suis	Sterile	Reported on day 10. The same organism in both compartments strongly supports haematogenous ocular seeding; without a blood culture it does not establish it
Ocular ultrasonography (B-scan): two examinations			
Day 4	Echogenic vitreous opacities, low to moderate reflectivity, moderate mobility; no retinal detachment demonstrated	Optically empty vitreous	Supports endophthalmitis. Reported qualitatively only: no standardised reflectivity, no grading of opacity density and no comment on posterior vitreous detachment
Day 20	Persistent vitreous opacity; no detachment or choroidal thickening demonstrated	Optically empty vitreous	Persistence despite intravitreal therapy was the indication for vitrectomy
Investigations that would ordinarily be requested and were not documented			
Blood cultures	Not documented	Sterile	Would have confirmed the bacteraemic phase directly and is the missing link in the seeding argument
Glycated haemoglobin, human immunodeficiency virus serology, immunoglobulins	Not documented	-	An immunocompromised state was suspected but never characterised
Formal pure-tone audiometry	Not documented	-	Hearing loss is the commonest sequela of this organism's meningitis and was left unquantified
Serial inflammatory markers	Not documented	-	One full blood count was taken. Nothing in this study tracks the inflammation serially

Intraocular therapy and the response of the inflammation

Topical levofloxacin, prednisolone acetate, tropicamide and a lubricant were begun on day 4, with intravenous ceftriaxone and dexamethasone directed by the neurology service; the doses, the frequency of the prednisolone acetate before its later taper, and the dexamethasone schedule are not recorded. There was no clinical improvement at 24 hours, and on day 5 vitreous aspiration was performed followed by intravitreal vancomycin and ceftazidime at the standard doses of 1.0 mg and 2.25 mg in 0.1 mL each, and hourly fortified vancomycin and ceftazidime drops. The hypopyon resolved and the anterior chamber cleared by day 10, 5 days after the injection, at which point the iris became visible for the first time and prednisolone acetate was reduced to four times daily. Acuity did not change. The serial anterior-segment appearance across the whole course is shown in Figure 2A.

The first hypertensive episode: an inflammatory mechanism

One day after the intravitreal injection the pressure was 14 mmHg. On day 7 it rose, with serial measurements of 24, 22 and 23 mmHg giving a session mean of 23.0 mmHg, and later the same day 29, 30 and 28 mmHg, a session mean of 29.0 mmHg. Oral acetazolamide 250 mg twice daily and potassium aspartate 600 mg daily were started, and topical timolol was added after glaucoma review the following day. The session mean fell to 17.3 mmHg by day 11, 4 days after the episode began, and to 12 mmHg by day

13, at which point pressure-lowering therapy was withdrawn. Three things acted over that interval – acetazolamide, timolol and the resolution of the inflammation itself – and the record cannot separate them. Throughout this episode the pupil was not visible and no iris configuration could be assessed. The whole pressure record, with every individual reading, the therapy in force at each point and the area above 21 mmHg, is plotted in Figure 3A.

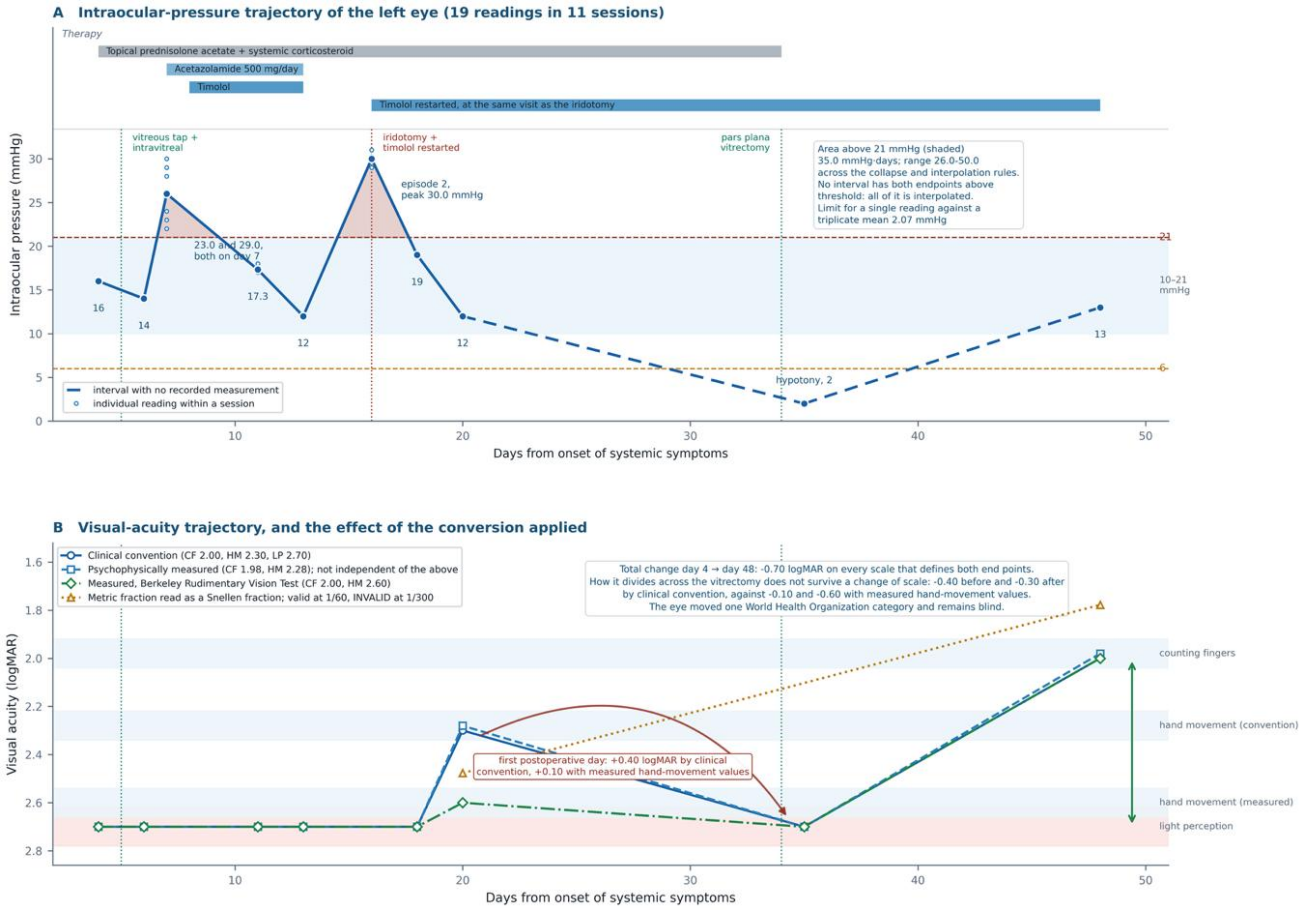

Figure 3. Quantified outcome trajectories. (A) Intraocular pressure, showing all 19 individual readings, the session means, the two hypertensive episodes, the shaded area above 21 mmHg and the single hypotony reading after vitrectomy; segments spanning intervals with no recorded measurement are dashed, and every unit of the shaded area lies on such a segment. (B) Visual acuity, including the clinically important deterioration on the first postoperative day, and the effect of converting the categorical notation 1/300 as though it were a Snellen fraction.

The second hypertensive episode: a mechanically distinct pupillary block

On day 16 the pressure rose again, with serial measurements of 31, 29 and 30 mmHg, a session mean of 30.0 mmHg. This was a rise of 18.0 mmHg from the 12 mmHg recorded three days earlier, which is 8.7 times the limit of 2.07 mmHg appropriate to a comparison between a single reading and a triplicate mean, or 4.6 to 12.9 times that limit under its own confidence interval. It is a real change on any of these figures. The rise also followed the withdrawal of acetazolamide and timolol three days earlier, which is a competing explanation the record cannot exclude. What the record does contain is a direct observation: by day 16 the anterior segment had cleared enough for the iris to be examined, and re-evaluation by the glaucoma division identified posterior synechiae with iris bombé and a pupillary block. Laser peripheral iridotomy was performed **and timolol was restarted at the same visit**. By day 18 the pressure was 19 mmHg, a fall of 11.0 mmHg within 2 days, attributable to the two together and not to the laser alone. No gonioscopy was performed before or after the procedure, so the block was neither formally separated from peripheral anterior synechial closure nor confirmed to have been relieved. The iris configuration is shown in the day 16 panel of Figure 2A, and the evidence for and against each candidate mechanism is set out in Figure 4B.

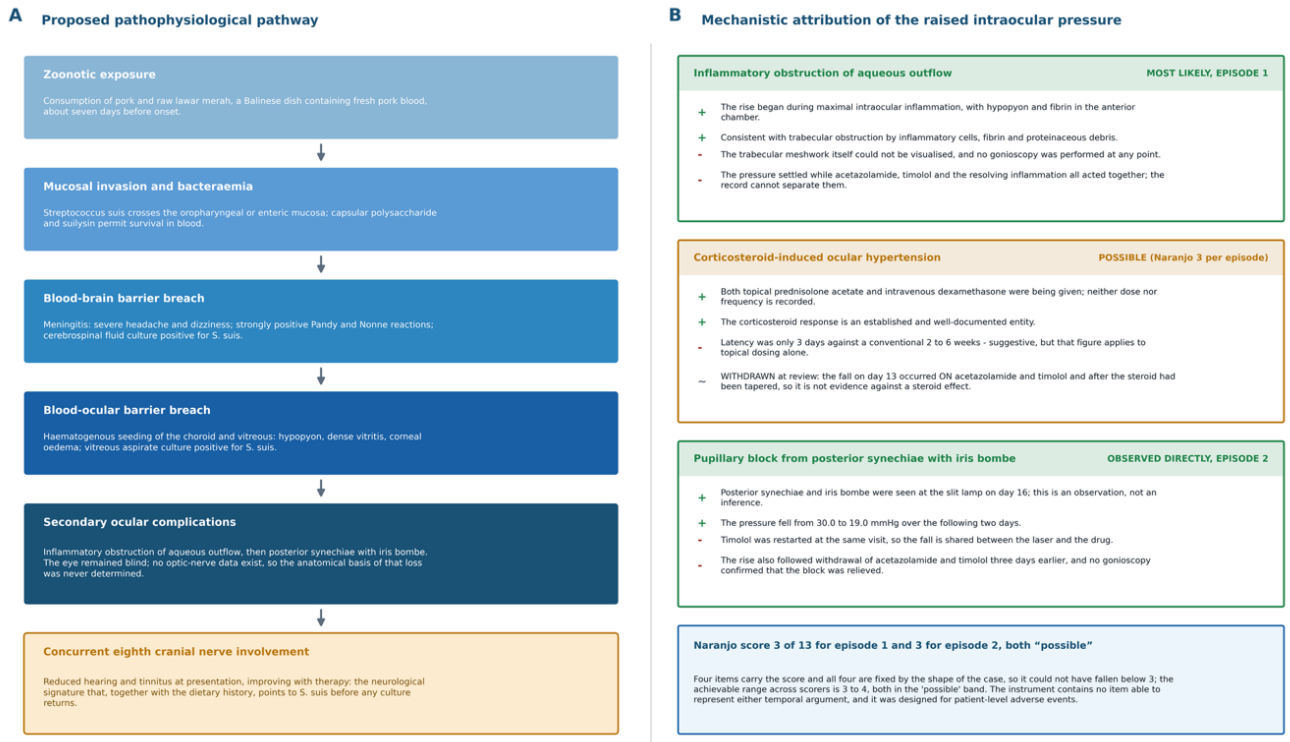

Figure 4. (A) Proposed pathophysiological pathway from zoonotic exposure to irreversible ocular injury, with the concurrent eighth cranial nerve involvement that provided the earliest clue to the organism. (B) Structured attribution of the raised intraocular pressure across three competing mechanisms, with the evidence for and against each, the co-interventions that confound each attribution, and the Naranjo probability score for the corticosteroid hypothesis scored separately by episode.

Attribution of the raised pressure

Corticosteroid causation scored 3 of a possible 13 on the Naranjo scale for the first episode and 3 for the second, placing both in the **possible** band. The score carries less information than it appears to. Four items determine it: the reaction is an established entity, it followed exposure, it was measured objectively, and an alternative cause exists. Those four are fixed by the shape of any case of this kind, so the score could not have fallen below 3, and the only item a different scorer could reasonably move yields 3 to 4, both in the 'possible' band. A further 5 items could not be scored because the corticosteroid was tapered at the same time as pressure-lowering therapy was started and no rechallenge was performed, and one item is a genuine negative rather than a missing value. The instrument also contains no item capable of representing the two temporal observations that bear most directly on the question. The first is the latency: 3 days from the first corticosteroid dose to the first pressure above 21 mmHg, about 4.7 times shorter than the two to six weeks conventionally reported for a topical corticosteroid response. The second is the behaviour of the pressure during the taper, and an earlier version of this analysis read it wrongly: the fall to 12 mmHg on day 13 occurred while the eye was on acetazolamide and timolol, which were withdrawn at that point, and three days after prednisolone acetate had been reduced to four times daily. A corticosteroid response suppressed by two hypotensive drugs and a dose reduction is expected to fall, so that observation is not evidence against a corticosteroid mechanism and is withdrawn as such. What remains is the short latency, which is suggestive rather than decisive, and the direct observation of a mechanism at the second episode that no drug explains. The complete scoring and its justification are given in Figure 4B and summarised in Table 4.

Course of the pressure across the whole admission

Across 44 days of observation, 19 readings were taken in 11 sessions, ranging from 2 to 31 mmHg. In all, 7 of 11 session means (63.6 per cent) lay inside the 10 to 21 mmHg reference band, 3 lay above it and 1 below. Pressure was measured when the clinical situation demanded rather than to a schedule, which is informative sampling: it biases every summary in this paragraph in a direction that cannot be determined, and it is a bias rather than merely a loss of precision. The area above 21 mmHg was 35.0 mmHg.days under the primary assumptions and ranged from 26.0 to 50.0 mmHg.days across the three same-day collapse rules and the three interpolation rules described above. That range understates the difficulty. No interval contributing to the quantity has both endpoints above the threshold, so the whole of it is generated by interpolation across crossings that were never observed, and no published referent exists against which a reader could judge whether it is large or small. It is reported as a descriptive summary of this record

and should not be treated as a comparable exposure measure. The time-weighted mean pressure between day 4 and day 20 was 19.3 mmHg on the same interpolation assumption. These derived metrics are listed with their comparators in Table 4.

Table 4. Quantitative outcome analysis: visual acuity, intraocular-pressure metrics, the clinical-significance thresholds and their limits, mechanistic attribution, and the level of evidence supporting each intervention

Measure	Value	Threshold or comparator	Interpretation
Visual acuity (logMAR, clinical-convention scale unless stated)			
Day 4 to day 48, overall	2.70 to 2.00, change -0.70	0.30 logMAR for a clinically important change	A 5.0-fold reduction in the minimum angle of resolution. The threshold is met by construction: this scale places adjacent low-vision categories exactly 0.30 logMAR apart, so any single-category change meets it
Day 4 to day 20, before vitrectomy	2.70 to 2.30, change -0.40	0.30 logMAR	The larger part of the total gain preceded surgery by 14 days
Day 20 to day 35, first postoperative day	2.30 to 2.70, change +0.40	0.30 logMAR	A clinically important deterioration, transient, and reported here because the same framework that credits the improvement must count it
Day 35 to day 48	2.70 to 2.00, change -0.70	0.30 logMAR	Net change across the surgical episode -0.30 logMAR
Psychophysically measured scale (Freiburg test)	2.70 to 1.98, change -0.72	Differs from the clinical-convention scale by a constant 0.02 on two categories	Not an independent check: for any hand-movement to counting-fingers comparison the two scales are algebraically identical
Measured Berkeley scale, overall	2.70 to 2.00, change -0.70	0.30 logMAR	Identical to the clinical-convention scale at both end points, because the two agree on light perception and on counting fingers and differ only at hand movement
Measured Berkeley scale, before and across surgery	-0.10 before, -0.60 across	-0.40 and -0.30 on the clinical-convention scale	The split reverses. Measured hand movement is 0.30 logMAR worse than the convention assumes, which moves most of the gain from before the vitrectomy to across it. Which interval is credited with the improvement is a property of the scale, not of the eye
Day 20 to day 35 on the measured Berkeley scale	change +0.10	0.30 logMAR	The postoperative deterioration falls below the clinically important threshold on this scale, so even the existence of that event is scale-dependent
Snellen-fraction conversion of 1/60	1.78	2.00 on the clinical-convention scale	Defensible: counting fingers at one metre is conventionally handled this way
Snellen-fraction conversion of 1/300	2.48	2.30 on the clinical-convention scale	Invalid. In the Indonesian convention 1/300 denotes hand movement, a category and not a Snellen fraction. Treating it as one inflates the estimated day 20 to day 48 gain 2.33-fold and is an error, not a scale choice
Final acuity in the affected eye	1/60 (0.017 decimal, Snellen 20/1200)	3/60 (0.05) is the World Health Organization threshold for blindness	The eye remains blind by that criterion. The criterion is defined on the presenting acuity of the better eye and does not strictly apply to one eye; no acuity was ever recorded for the right eye
Intraocular pressure (mmHg)			
Readings and sessions	19 readings in 11 sessions over 44 days	-	Measured when the clinical situation demanded, not to a schedule. This is informative, outcome-dependent sampling and biases every summary below in an unknown direction
Range	2 to 31	10–21	7 of 11 sessions (63.6%) lay inside the reference band
Within-session standard deviation	0.913 on 8 degrees of freedom	95% interval 0.617 to 1.749	Estimated from four triplicate sessions. It captures short-term reading variability only: no between-day, between-observer or diurnal component, and no time of day was recorded
Repeatability coefficient for two single readings	2.53	95% interval 1.71 to 4.84	2.77 times the within-session standard deviation. The multiplier

			is an assumed constant, not a derived one
Coefficient for a single reading against a triplicate mean	2.07	The comparison this study actually makes	1.96 times the standard deviation times the square root of 1 + 1/3. The larger figure above is conservative for this comparison but is not the correct estimator for it
Order effect within sessions	The third reading was exactly 1 mmHg below the first in all four triplicate sessions	Sign test p = 0.125	Not significant on four sessions, but perfect consistency is not what random error looks like. Removing an additive position effect gives a within-session standard deviation of 0.816
Episode 1 (inflammatory), days 7 to 11	Peak session mean 29.0	21	Controlled within 4 days of starting acetazolamide, with timolol added the following day. Resolution of the inflammation and two drugs acted at the same time
Episode 2 (pupillary block), days 16 to 18	Peak session mean 30.0	21	Fell by 11.0 mmHg within 2 days of laser peripheral iridotomy and simultaneous reintroduction of timolol; the fall cannot be attributed to the laser alone
Rise from day 13 to day 16	18.0	2.07 (single reading against a triplicate mean)	8.7 times measurement noise, or 4.6 to 12.9 times under the coefficient's own confidence interval. A real change on any of these figures
Ocular-hypertension burden above 21 mmHg	35.0 mmHg.days	26.0 to 50.0 across three same-day collapse rules and three interpolation rules	No interval contributing to this quantity has both endpoints above the threshold: 100% of it is generated by interpolation across crossings that were never observed. No published referent exists, so it is a descriptive summary of this record and not a comparable exposure measure
Time-weighted mean pressure, days 4 to 20	19.3	10–21	Just inside the reference band despite two excursions, on the same interpolation assumption
Postoperative hypotony, day 35	2	Below 6 is hypotony	A complication, not an incident. No pressure and no examination are recorded in the 13 days that followed, so its resolution rests on the single reading of 13 mmHg on day 48. No burden statistic is reported
Mechanistic attribution of the raised pressure			
Naranjo score for corticosteroid causation, episode 1	3 of a possible 13 (range -4 to 13)	1–4 possible, 5–8 probable, 9 or more definite	Category: possible. The score is structurally determined: the four items that carry it are fixed by the shape of any case in which a known reaction follows exposure, is measured objectively and has an alternative cause. The achievable range across scorers is 3 to 4, both in the 'possible' band
Naranjo score, episode 2	3 of 13	As above	Category: possible. Scored separately because the two episodes are mechanistically distinct; a single score across both would mix the evidence
Items that could not be scored	5 unknown or not applicable, 1 a genuine 'no'	of 10 items	The instrument contains no item capable of representing the argument the authors regard as strongest, and it was designed for patient-level adverse events rather than for competing physiological mechanisms of one serial measurement
Latency from first corticosteroid dose to the first pressure above 21 mmHg	3 days	2 to 6 weeks for a conventional topical corticosteroid response	About 4.7 times shorter. The comparator applies to conventional topical dosing; this patient received topical prednisolone acetate and intravenous dexamethasone together, and rises within days are described with intensive and systemic regimens, so the argument is suggestive rather than decisive

Level of evidence for each intervention (Oxford 2011; GRADE was not assessed)			
Intravenous ceftriaxone for Streptococcus suis meningitis	Level 4	Oxford Centre for Evidence-Based Medicine 2011	Non-randomised cohorts and case series; no randomised comparison exists for this organism.
Vitreous sampling before intravitreal antibiotic	Level 4	Oxford Centre for Evidence-Based Medicine 2011	Case series and consensus practice. It establishes the microbiological diagnosis and must not delay systemic therapy.
Intravitreal vancomycin 1.0 mg/0.1 mL + ceftazidime 2.25 mg/0.1 mL	Level 4	Oxford Centre for Evidence-Based Medicine 2011	A systematic review of case reports and case series remains Level 4 in this hierarchy, however large.
Pars plana vitrectomy for persistent vitreous opacity	Level 4	Oxford Centre for Evidence-Based Medicine 2011	Non-randomised series with directly conflicting results on timing. The one randomised trial of vitrectomy in bacterial endophthalmitis studied postoperative, not endogenous, disease.
Laser peripheral iridotomy for pupillary block	Level 5	Oxford Centre for Evidence-Based Medicine 2011	Mechanism-based reasoning plus case series. The randomised evidence concerns primary angle closure, which is a different condition.
Topical timolol and oral acetazolamide for secondary ocular hypertension	Level 5 here	Oxford Centre for Evidence-Based Medicine 2011	Level 1 evidence exists in primary open-angle glaucoma. It does not transfer to acute inflammatory ocular hypertension, so in this setting the basis is mechanism-based reasoning.
Topical and systemic corticosteroid for intraocular inflammation	Level 5 here	Oxford Centre for Evidence-Based Medicine 2011	Extrapolated from exogenous endophthalmitis and uveitis practice; no trial in endogenous bacterial endophthalmitis.
What a single case cannot estimate			
Correlation between ophthalmic and neurological measures	Not estimable	Spearman's rank correlation is undefined at n = 1	Replaced by a narrative concordance description, which yields no coefficient, no null hypothesis and no uncertainty and is not an analysis
Exact binomial interval for a 1-of-1 outcome	95% CI 2.5% to 100.0%	Clopper-Pearson	A width of 97.5 percentage points. The proportion it would estimate is the proportion of such eyes gaining at least 0.30 logMAR
One-sample size to distinguish a 40% from a 20% outcome rate	44 cases	Two-sided exact binomial, nominal alpha 0.05, attained size 0.040	Power first reaches 0.80 at n = 38 but falls below it again at n = 40, 42, 43, because the test is discrete; 44 is the smallest size from which it stays above. At n = 1 the rejection region is empty and power is exactly 0 for these parameters
Two-group size for the question the case actually raises	about 82 per arm, 164 in total	Early against delayed vitrectomy, 20% against 40%, 80% power	The one-sample figure above understates the requirement roughly fourfold. These planning parameters are illustrative, were chosen for this report and were not pre-specified

Four conversions are tabulated. The clinical-convention scale assigns counting fingers 2.00, hand movement 2.30 and light perception 2.70 logMAR; the psychophysically measured scale uses 1.98 and 2.28 and retains 2.70 for light perception; the measured Berkeley scale uses the medians obtained with the Berkeley Rudimentary Vision Test in 38 low-vision patients, 2.00 for counting fingers and 2.60 for hand movement; the fourth reads the recorded metric fraction as though it were a Snellen fraction, which is defensible at 1/60 and invalid at 1/300. The 0.10 and 0.30 logMAR thresholds were derived in eyes tested on letter charts; no threshold has been validated for categorical low-vision measurement, and on the clinical-convention scale the 0.30 figure coincides exactly with the spacing between adjacent categories. GRADE certainty was not assessed and letter grades were deliberately not used.

Vitrectomy, hypotony and the final outcome

At the first outpatient review on day 20 the patient reported improved vision. Acuity was 1/300, hand movement equivalent, 2.30 logMAR. The pressure was 12 mmHg and there was residual anterior chamber fibrin and mild inferior corneal haze. Ultrasonography showed persistent vitreous opacity with no detachment demonstrated, and the patient was referred to the vitreoretinal division. Pars plana vitrectomy was performed on day 34, 29 days after the first intravitreal injection, combined with synechiolysis of the fibrotic tissue and posterior synechiae found at surgery and with a second intravitreal dose of vancomycin and ceftazidime. The retina was attached at surgery. On the first postoperative day the pressure was 2 mmHg, below the hypotony threshold, with corneal oedema and Descemet folds, and acuity had fallen to light perception. No Seidel test, anterior chamber depth assessment or choroidal examination is

recorded, and no further examination of any kind appears until day 48, 13 days later; that reading and the gap that follows it are both visible in Figure 3A. Systemic methylprednisolone and clindamycin were given with topical tropicamide, levofloxacin, prednisolone acetate and a lubricant. At day 48, two weeks after vitrectomy, acuity was 1/60, counting fingers at one metre; the pressure was 13 mmHg; the lid spasm and hyperaemia had resolved; and the fundus and optic disc were directly visible for the first time in the illness, although neither disc was described.

Quantified visual outcome, in both directions

From presentation to the final assessment, acuity improved by 0.70 logMAR, a 5.0-fold reduction in the minimum angle of resolution. That total conceals three separate movements, and reporting only the total would have credited the vitrectomy with all of it. On the clinical-convention scale, 0.40 logMAR had already occurred by day 20, fourteen days before surgery. Acuity then **fell** by 0.40 logMAR on the first postoperative day, a deterioration that exceeds this study's own 0.30 logMAR threshold for a clinically important change and that an earlier version of this analysis mentioned only in passing. The net movement across the surgical episode was 0.30 logMAR. Two cautions belong beside every one of those figures. The threshold they are judged against is met by construction, because the conversion scale places adjacent low-vision categories exactly 0.30 logMAR apart; and the presenting value of 2.70 is an upper bound, so each gain is a lower bound. The alternative published scale gives 0.72 logMAR for the total, which is agreement by arithmetic rather than by evidence, since the two scales differ by a constant on the categories concerned. The third conversion is where something real appears. Treating the recorded 1/300 as a Snellen fraction rather than as the hand-movement category it denotes gives a day 20 to day 48 gain of 0.70 logMAR instead of 0.30, an inflation of 2.33-fold. That is not a scale choice with two defensible answers; it is an error, and it is one that a reader of any paper quoting a logMAR change at this level of vision cannot detect unless the conversion is stated. The clinical meaning of the outcome is more sober than the arithmetic suggests: 1/60 corresponds to a decimal acuity of about 0.017, below the 3/60 threshold for blindness, so the eye moved from ICD-10 category 4 to category 3 and remained blind[55]. Because the World Health Organization criterion is defined on the presenting acuity of the better eye, and no acuity was ever recorded for the right eye, no person-level category can be assigned. Both trajectories are plotted in Figure 3B and tabulated in Table 4.

Ophthalmological and neurological findings, and how they relate

Spearman's rank correlation is undefined at a sample size of one, so no coefficient is reported. What follows is a description of how the two sets of findings relate to a single hypothesis, haematogenous dissemination from a systemic focus, and it is a structuring device rather than an analysis: it has no null hypothesis, no uncertainty, and its items were chosen by the authors who hold the hypothesis. Of the 4 findings considered, the foundational one is the isolation of the same organism from the cerebrospinal fluid and from the vitreous, which is the premise of the whole study and is not counted again. Two further pairs are concordant with it: barrier breach demonstrated in both compartments, by hypopyon and dense vitritis on one side and strongly positive Pandy and Nonne reactions on the other; and resolution in both compartments after targeted therapy, the hypopyon clearing five days after the intravitreal injection and the headache absent by the first outpatient review. The remaining finding, the reduced hearing with tinnitus, has no ocular counterpart at all. It is nonetheless the most useful of them clinically, because an eighth-nerve deficit in a patient with meningitis and a red eye points to this organism before any culture returns. An earlier version of this analysis also counted a fourth concordant pair and one informatively discordant finding; both have been withdrawn, the first because no serial inflammatory markers exist, and the second because it rested on a description of the fellow optic disc that the record does not contain.

What this single case could not estimate

A single favourable observation out of one gives an exact Clopper-Pearson interval of 2.5 to 100.0 per cent, a width of 97.5 percentage points; the proportion it would estimate is the proportion of such eyes gaining at least 0.30 logMAR. For a two-sided exact binomial test of one proportion against a known 20 per cent, power against 40 per cent first reaches 0.80 at 38 cases but falls below it again at 40, 42, 43 cases, because the test is discrete; 44 is the smallest size from which it stays above. At a sample size of one the rejection region of that test is empty and its power is exactly 0, which is a property of these parameters rather than of single cases in general. The question this case actually raises, early against delayed vitrectomy, is a comparison between two groups, and for a 20 against 40 per cent difference that needs about 82 cases per arm, 164 in total – roughly four times the one-sample figure. The 5 eye-based series in Table 5 contain 567 eyes between them, drawn from 4 countries over overlapping calendar periods; the Cochrane review in the same table counts 420 randomised participants in exogenous disease and the database study counts 8,255 hospital admissions, so the units are not interchangeable and no pooled count is offered. These figures are reported because they define precisely what a reader should and should not take from what follows.

Table 5. Contemporary evidence on the management and visual prognosis of endogenous bacterial endophthalmitis, and the position of the present case among it

Study	Design and size	Question examined	Reported finding	Bearing on this case
Muqit et al., 2022	Cochrane systematic review; one eligible randomised trial of 420 participants	Immediate vitrectomy against vitreous tap and intravitreal antibiotic	No difference in acuity at 3 or 9 months, at very low certainty; one eligible trial in four decades	The only randomised evidence in the field concerns exogenous, postoperative disease and transfers to this eye by analogy alone
Uppuluri et al., 2023	National inpatient database, United States, 2002–2014; 8,255 admissions	Setting, causative category and procedures performed	Bacterial in 93.2 per cent; vitrectomy in 11.5 per cent of urban and 2.9 per cent of rural admissions	Establishes that vitrectomy is performed in a minority of admissions and that access depends on the setting, which is the constraint this pathway operates under
Dedieu et al., 2024	12-year cohort, two tertiary centres, France; 64 eyes of 53 patients	Microbiological yield and visual outcome	Confirmed microbiologically in 81 per cent; final acuity worse than 20/400 in 49 per cent; structural loss of the globe in 28 per cent	This eye was confirmed from two compartments and was retained, placing it in the better half on structure and the worse half on acuity
Sromicki et al., 2025	Single-centre series of early vitrectomy; 92 eyes, predominantly exogenous, with endogenous cases inside a 30-eye mixed subgroup	Visual outcome and complication rate after early vitrectomy, stratified by aetiology	78.9 per cent of post-cataract eyes presenting better than light perception reached 20/40 or better; complications were commonest outside that group	This eye presented at light perception and was not post-cataract, the stratum in which early vitrectomy performed least well
Chuang et al., 2025	10-year single-centre retrospective review, northern Taiwan; 99 eyes of 91 patients	Pathogen distribution, visual outcome and mortality	Bacterial in 61.5 per cent; visual prognosis poor irrespective of pathogen; mortality 15.4 per cent	Matches the outcome here: the infection was controlled and the patient survived, and the eye remained blind
Weng et al., 2026	Retrospective cohort, 8 tertiary centres, United States; 262 eyes of 231 patients	Predictors of visual gain and of loss of the globe	Eyes presenting at counting fingers or hand movement gained more acuity than eyes at light perception or worse; 23 of the 28 eyes removed (82.1 per cent) had presented at light perception or worse	This eye presented at light perception with poor projection and was retained, which is the favourable outcome within an unfavourable stratum
Zhang et al., 2026	Culture-proven series, single centre, United States, 2013–2024; 50 eyes of 41 patients	Ocular and systemic features and outcome of culture-proven disease	Fungal in 46 per cent; final acuity 20/400 or better in 52 per cent; death in 7 per cent	The final acuity here, 1/60 or about 20/1200, lies below that threshold and therefore in the worse half of this series
The present case				
This report	Observational analytic case study; 1 eye, 48 days of follow-up	Intravitreal antibiotic on day 5, vitrectomy on day 34, an interval of 29 days	Acuity 0.40 logMAR better before surgery and 0.30 logMAR better across it on the clinical-convention scale, but 0.10 and 0.60 logMAR respectively on the measured Berkeley scale, with a transient 0.40 logMAR loss on the first postoperative day	One observation cannot adjudicate between these studies; it can only be placed among them

All 7 comparator studies were published between 2022 and 2026 and each figure quoted here is taken from the abstract of the primary paper. The units differ and are not interchangeable: the database study counts hospital admissions, the Cochrane review counts randomised participants in exogenous disease, and the 5 eye-based series count eyes (567 in total, drawn from 4 countries over overlapping calendar periods). No pooled count is offered and the totals must not be added. There is no randomised trial of vitrectomy in endogenous endophthalmitis; that is a gap in the literature and not an omission from this table.

4. Discussion

Principal findings

This study documents a culture-proven endogenous endophthalmitis caused by *Streptococcus suis* in a patient whose only identified exposure was a traditional Balinese dish containing raw pork blood, and whose eye and central nervous system yielded the same organism. Three findings go beyond what a descriptive report would have produced, and one of them is a correction of this study's own earlier reading. First, the raised intraocular pressure was not one entity but two, separated by nine days and by mechanism: an inflammatory obstruction of aqueous outflow during the acute phase, and a pupillary block from posterior

synechiae with iris bombé once the inflammation had begun to organise, the second seen directly at the slit lamp. Second, the contribution of corticosteroid to the first episode, which is the explanation most readily reached for in an eye receiving frequent prednisolone acetate, scores 3 of 13 on a validated instrument – a score that could not have come out much differently, and which is therefore worth less than it looks. Third, the visual outcome is a case study in how a large measured improvement and a poor clinical result coexist, and in how easily the improvement is misattributed: acuity improved by 0.70 logMAR overall, but 0.40 of that preceded the vitrectomy, acuity fell by 0.40 logMAR on the first postoperative day, and the eye is blind.

The organism, the exposure and the Indonesian context

Human *S. suis* infection is concentrated in populations with close contact with pigs or with traditions of eating raw or undercooked pork, and the great majority of reported cases come from East and Southeast Asia[8,9,10]. Meningitis dominates the clinical spectrum and hearing loss is its signature sequela[12,40,56]. The sociocultural dimension of this exposure has been examined explicitly, and the conclusion relevant here is that the dish, not the occupation, is often the vehicle[11], which is why the exposure recorded in Table 1 is a dietary one. Bali provides the clearest illustration: *S. suis* meningitis has been documented on the island in association with the consumption of raw pork preparations[17]. For a clinician in Denpasar this changes the prior probability of the diagnosis materially. A patient with meningitis, hearing loss and a dietary history of raw pork should have *S. suis* requested specifically, and the request should be operational rather than general: the organism is readily reported as a viridans streptococcus by biochemical identification panels, so what a district laboratory needs to be asked for is confirmation by mass spectrometry or 16S ribosomal RNA sequencing[8,19]. The present case reached its microbiological diagnosis quickly enough for that not to matter; elsewhere it might not have.

Comparison with previous ocular reports and with contemporary endophthalmitis series

The published ocular experience of this organism consists of isolated reports. Endogenous endophthalmitis complicating *S. suis* meningitis has been described in a patient managed in Vietnam[18], and a severe endophthalmitis with bilateral deafness has been reported with genomic characterisation of the causative sequence type[19]. The present case is strictly unilateral, and that is the point at which it differs from the bilateral report. A handful of cases cannot establish a distribution, and the honest statement is that the ocular phenotype of this organism is described by a literature of individual eyes, which is why any account of what *S. suis* 'usually' does in the eye should be treated with suspicion. What the reports share is the association with hearing loss, and that association is worth more clinically than the laterality, as Figure 4A indicates. Against the broader endogenous endophthalmitis literature, summarised in Table 5, the case is unremarkable in its presentation and its outcome. Presenting acuity of light perception is at the poor end of the range reported in tertiary series, and in the largest recent multicentre cohort it is the stratum from which eyes gained least and from which 23 of the 28 eyes eventually removed had come[3,4,5]. The absence of any conventional predisposing factor is less usual: most endogenous cases occur in a host with an identifiable vulnerability, and in culture-proven series a systemic source is identified in the majority[1,6]. Here no such factor was found, but neither was one systematically sought, and the suspected immunocompromised state recorded by the treating team was never characterised. That is a gap in the record rather than a negative finding, and it should not be read as evidence that this organism attacks the immunocompetent eye. The comparison also has a boundary worth naming: the series in Table 5 are adult series, and the organism spectrum and prognosis of endogenous endophthalmitis differ in children, so none of them would transfer to a paediatric presentation of the same infection[57].

Two mechanisms, two treatments: the central clinical message

The most transferable finding in this case is that two pressures a single millimetre of mercury apart, 29.0 and 30.0 mmHg, meant two different things nine days apart. On day 7 the pressure rose while the anterior chamber was full of inflammatory cells and fibrin and the pupil could not be seen; it settled as aqueous suppression, beta-blockade and the resolution of the inflammation acted together, and the record cannot separate their contributions. On day 16 a pressure indistinguishable from it, and well inside the 2.07 mmHg measurement limit derived here, arose from a pupil sealed by posterior synechiae with the iris bowed forward – a mechanism seen directly, not inferred – and the pressure fell by 11.0 mmHg over the following 2 days after laser peripheral iridotomy **and the simultaneous reintroduction of timolol**. The numerical attribution of that fall is therefore shared between the two, and it would be wrong to present the laser as having done it alone. What is not shared is the diagnosis. Had the second episode been treated as a simple recurrence of the first, the iris configuration might have gone unexamined and the block unrelieved. The mechanisms of raised pressure in inflamed eyes are individually well described[23,24,27], but they are usually presented as a list rather than as a sequence, and the sequence is what a clinician encounters. The randomised evidence for laser peripheral iridotomy concerns primary angle closure and does not extend to secondary inflammatory pupillary block, where the basis is mechanism and case series[27,58]. The practical rule that follows is embedded in Figure 5: in an

inflamed eye whose pressure rises, examine the pupil and the iris contour before escalating drops, and perform gonioscopy where the cornea permits it. In the acute phase it may not, which is precisely why the examination must be repeated as the media clear rather than recorded once as impossible. A second, less comfortable lesson follows from the same sequence: the day 16 rise came three days after acetazolamide and timolol were withdrawn, and withdrawal of therapy is a competing explanation for it that this record cannot exclude. The block is established by direct observation, not by the timing.

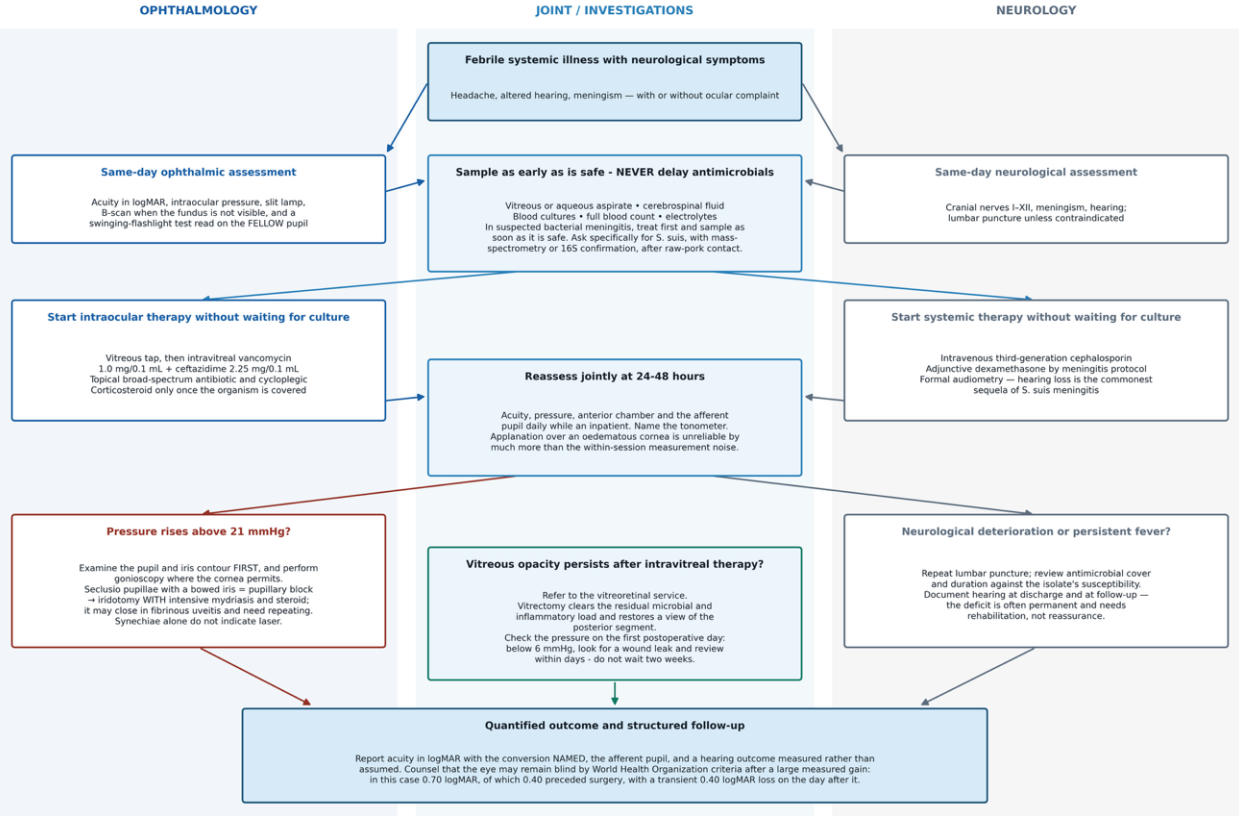

Figure 5. Proposed interdisciplinary pathway for suspected endogenous endophthalmitis complicating bacterial meningitis, showing the ophthalmological, joint and neurological responsibilities at each step. Antimicrobial therapy is never delayed for a specimen.

Was it the steroid? A worked attribution, and what the working shows

Corticosteroid-induced ocular hypertension is real, common and mechanistically well understood: glucocorticoids increase outflow resistance through accumulation of extracellular matrix in the trabecular meshwork, reorganisation of the cytoskeleton, increased myocilin expression and reduced phagocytic activity[25,26]. In an eye receiving frequent topical prednisolone acetate and intravenous dexamethasone it is the first explanation any clinician reaches for. Scoring it formally returns 3 of 13, the **possible** band, for each episode separately. The more useful result of the exercise is what it reveals about itself. Four items carry the score, and all four are fixed by the shape of the case rather than by its evidence, so the total could not have fallen below 3 and could not plausibly have exceeded 4. An instrument that cannot vary with the evidence is not adjudicating anything, and the honest report of such an exercise is to say so. The instrument is also being used outside its design: it estimates the probability that a drug caused an adverse event in a patient, not that a drug rather than a competing physiological mechanism produced one serial measurement, and it contains no item that could represent either of the temporal observations that matter here. Of those two, one survives and one does not. The latency of 3 days is genuinely short against the two to six weeks conventionally reported[25,59,60], but that figure applies to conventional topical dosing, and this patient received a topical and a systemic corticosteroid together. Systemic dexamethasone is standard adjunctive therapy in bacterial meningitis and is supported by long-run cohort data as well as by trial evidence[61], so its administration here was not discretionary and could not have been withheld to protect the eye. Rises within days are described for intensive regimens; the argument is suggestive, not decisive. The second observation, that the pressure fell while prednisolone acetate continued, is withdrawn: it fell on two hypotensive drugs and after a dose reduction, and is exactly what a suppressed steroid response would also do. The wider point is methodological, and it is the reason for printing every item in Figure 4B. A causality claim in a case report is checkable only if the instrument, the items and the justification for each score are shown; and showing

them, in this instance, is what revealed that the instrument had nothing to add.

Antimicrobial strategy: why the eye needs its own dose

Systemic antibiotic penetration into the vitreous is limited by the blood-ocular barrier, which is why an eye with established endophthalmitis needs a dose delivered into the vitreous cavity rather than relying on the systemic regimen that is treating the source[1,62]. The empirical intravitreal combination of vancomycin for Gram-positive and ceftazidime for Gram-negative organisms, at 1.0 mg and 2.25 mg in 0.1 mL respectively, is the standard against which alternatives are judged[20,62]. In retrospect the choice was fortunate as well as correct: *S. suis* is generally susceptible to beta-lactams, so the systemic ceftriaxone directed by the neurology service and the intravitreal ceftazidime were both active, and the confirmation of the organism on day 10 required no change of agent[9,10]. Three cautions follow for practice. The vitreous tap was taken one day after systemic ceftriaxone had already begun and still grew the organism, which is fortunate rather than reproducible; specimens should be taken as early as is safe, and **antimicrobial therapy in suspected bacterial meningitis must never be delayed for them**. The hourly fortified drops given here achieve negligible vitreous concentrations on an intact globe and carry a real risk of epithelial toxicity in an already oedematous cornea. And the hearing deficit, which is the sequela this organism is known for and which persisted at the last review, is not prevented by getting the antimicrobial right, so it must be quantified and followed rather than assumed to resolve with the infection[39,56,63].

Timing of vitrectomy: where this case sits, and what it cannot settle

The timing of pars plana vitrectomy in endogenous endophthalmitis is unsettled, and Table 5 sets the contemporary evidence side by side. The most important entry in that table is a negative one: a Cochrane review identified a single eligible randomised trial in four decades, enrolling 420 participants, which found no difference between immediate vitrectomy and vitreous tap with intravitreal antibiotic at three or nine months at very low certainty, and which studied **exogenous, postoperative** disease[21]. There is no randomised evidence at all in endogenous disease, so every recommendation in this area rests on observational data. Those data are consistent on prognosis and silent on timing. A multicentre cohort of 262 eyes found that presenting acuity, not the procedure performed, predicted visual gain, and that eyes presenting at light perception or worse supplied 82.1 per cent of the eyes eventually removed[5]. A twelve-year two-centre cohort of 64 eyes reported final acuity worse than 20/400 in 49 per cent and loss of the globe in 28 per cent[3]. A ten-year series of 99 eyes found the visual prognosis poor irrespective of pathogen[4]. A series of 92 eyes undergoing early vitrectomy found good outcomes concentrated in post-cataract eyes presenting better than light perception, with endogenous cases confined to a small mixed subgroup[22]. Across a national database of 8,255 admissions, vitrectomy was performed in 11.5 per cent of urban and 2.9 per cent of rural admissions, so the procedure is a minority event whose availability depends on the setting[2]. The eye reported here was operated 29 days after the first intravitreal injection and falls in the later group under every published cut-off. It did respond, but the response must be stated in full: the vitreous cleared, the fundus became visible, acuity fell to light perception on the first postoperative day and finished 0.30 logMAR better than its pre-operative value on the clinical-convention scale, or 0.60 logMAR better on the measured Berkeley scale. Whether earlier surgery would have produced a better result is not answerable from this case and is not claimed. What can be said is arithmetical: a single favourable observation supports an interval two orders of magnitude wider than any difference worth detecting. The two-group comparison this question actually requires would need about 164 eyes, which is fewer than the 567 eyes already contained in the 5 eye-based series of Table 5. The trial is therefore feasible in arithmetic and absent in fact: those 567 eyes are spread across 4 countries, seven centres and overlapping calendar periods, none of them randomised and none of them restricted to endogenous disease, which is precisely the configuration a registry with a mandatory minimum dataset would replace.

Interpreting the visual outcome honestly

The temptation in reporting an eye that improves from light perception to counting fingers is to describe a 5.0-fold reduction in the minimum angle of resolution and leave it there. Four cautions belong beside that number. First, on the clinical-convention scale most of the gain preceded the operation it is easily attributed to, and the operation was followed by a clinically important transient loss – but that split is a property of the conversion and not of the eye. The clinical-convention and measured Berkeley scales agree exactly at both ends of the illness, placing light perception at 2.70 and counting fingers at 2.00, so the total gain is identical on either. They disagree only at hand movement, the single intermediate point that separates the pre-operative from the post-operative interval, where the measured value is 0.30 logMAR worse than the convention assumes[43]. Substituting it moves the gain from 0.40 logMAR before surgery and 0.30 across it to 0.10 before and 0.60 across, reversing which interval carries the improvement, and it reduces the postoperative deterioration from 0.40 to 0.10 logMAR, below the threshold at which this study would call it clinically important. Both arrangements appear in Table 4. Nothing in this record adjudicates between the two scales, so the correct report of the split is that it is scale-dependent, which is a weaker and more accurate

claim than either version alone. Second, the presenting acuity was light perception with **poor** projection, worse than light perception with accurate projection, and no published conversion distinguishes them; 2.70 logMAR is an upper bound and the reported gain a lower one[41,43]. Third, the thresholds the change is judged against, 0.10 and 0.30 logMAR, were derived in eyes tested on letter charts, and on the scale used here adjacent low-vision categories sit exactly 0.30 logMAR apart, so the threshold is met by any single-category change whatever the eye does. Fourth, and most important clinically, the final acuity of 1/60 is a decimal acuity of about 0.017, below the 3/60 threshold for blindness, so the eye that improved by seven notional lines moved from ICD-10 category 4 to category 3 and remained a blind eye[55]. The counselling implication is direct: a patient told that their vision has improved five-fold, who then discovers that the eye is registered as blind, has been misled by an arithmetically correct statement. The conversion question makes the same point from another direction. Treating the recorded 1/300 as a Snellen fraction rather than as the hand-movement category it denotes inflates the day 20 to day 48 gain 2.33-fold, as Figure 3B shows. A logMAR change quoted at this level of vision without its conversion stated is not a measurement.

The interdisciplinary contribution, stated concretely

Joint assessment is easy to praise and hard to evidence. In this case the neurological contribution can be named at three specific points. The diagnosis of bacterial meningitis and the decision to perform lumbar puncture produced the cerebrospinal fluid isolate, which was the first microbiological identification of the organism and which allowed the intravitreal regimen to be read as targeted rather than empirical once the vitreous culture agreed. The systemic antimicrobial and corticosteroid strategy was directed by the neurology service under a meningitis protocol. And the recognition of reduced hearing with tinnitus as a probable eighth-nerve deficit, rather than a coincidental symptom, is the observation that with the dietary history points to this organism specifically. An earlier version of this analysis claimed a fourth contribution, an inference about intracranial pressure drawn from the fellow optic disc; it has been withdrawn, because the record contains no description of that disc. Two places where genuine joint reasoning was needed and is not documented deserve naming, because they are where such a collaboration earns its keep. Adjunctive intravenous dexamethasone is given in bacterial meningitis precisely because it reduces the hearing loss this organism causes, and it is simultaneously the systemic limb of the corticosteroid exposure whose contribution is assessed here; no dose, route or taper appears anywhere in the record. And acetazolamide was added on day 7 to a patient with bacterial meningitis, a sodium of 134 mmol/L and a platelet count of 119 x 10^3/uL, with foreseeable metabolic consequences and no follow-up electrolytes recorded.

Implications for practice in Indonesian tertiary centres

Three practical points follow. The first is that a febrile patient with neurological symptoms and any ocular complaint needs an ophthalmic examination on the day of admission, not when the systemic illness has settled. Here the interval from ocular symptom onset to ophthalmic assessment was between 1 and 5 days, which was fast, and the eye still ended blind; the whole sequence of decisions and the service responsible for each is displayed in Figure 1. The second is that specimens should be obtained as early as is safe from both compartments, without delaying antimicrobial therapy in a patient with suspected bacterial meningitis, where delay costs lives; the vitreous tap here was performed on day 5, one day after systemic ceftriaxone had begun, and grew the organism nonetheless. The third concerns resources, and it has changed since the first draft of this analysis. Optical coherence tomography, automated perimetry and electrophysiology were unavailable in this eye at every point during the acute illness, and it is tempting to present that as the constraint. It was not. The measurement this case most needed was a swinging-flashlight test read on the fellow pupil, which requires a torch, works through an opaque cornea, and is the only bedside measure of retinal and optic-nerve function available in such an eye. It was not performed, and the absence of any optic-nerve data is the reason this study can say nothing about why the eye remained blind. The analytic framework demonstrated here needs no equipment a provincial Indonesian eye department does not already have; the same is true of the examination it most conspicuously lacked. Figure 5 renders the resulting pathway as a practical algorithm.

Strengths

The study has three strengths. The microbiological foundation is unusually secure: the same organism was isolated from a systemic and from an intraocular specimen, which moves the account of endogenous seeding from impression to strong support. The measurement model is explicit and reproducible, and its own uncertainty is reported rather than concealed: the repeatability coefficient carries 8 degrees of freedom and a confidence interval of 1.71 to 4.84 mmHg, the exposure metric is reported as a range across its analytic choices rather than as a point estimate, and the fraction of it generated by unobserved interpolation is stated. And the limits of the design are quantified rather than gestured at, including an explicit list of every parameter of the assessment framework that was not obtained, each with its reason, in Table 2 and Table 4. A fourth claim made in an earlier version, that the causality instrument strengthened the attribution, has been withdrawn; what the instrument

in fact showed is that it could not discriminate here, and reporting that is more useful than the score.

Limitations

The limitations are substantial. This is one eye of one patient, so no estimate generalises and no comparison is tested; every inferential statement in this manuscript is external. The record is retrospective and the measurement schedule was clinically driven, which is informative sampling and therefore a source of bias and not merely of imprecision. **The study holds no data of any kind on optic-nerve function**: no relative afferent pupillary defect, no field, no nerve fibre layer imaging and no description of the disc even after it became visible, so the contributions of optic neuropathy, macular damage and persistent media opacity to the final acuity cannot be separated, and the diagnosis recorded by the treating team as secondary glaucoma is supported here only as secondary ocular hypertension. No gonioscopy was performed, so the pupillary block was neither formally distinguished from peripheral anterior synechial closure nor confirmed relieved. The tonometer is not named and central corneal thickness was not measured, so an applanation bias that varies in step with the corneal oedema, and therefore with the trajectory under analysis, cannot be estimated; no time of day was recorded, and diurnal variation exceeds the measurement limits derived here. The corticosteroid exposure whose contribution is assessed here is itself unquantified: neither the frequency of the topical drug before its taper nor any detail of the systemic dexamethasone appears in the record. Cerebrospinal fluid opening pressure, cell counts, protein and glucose were not recorded and no neuroimaging was documented, so no statement about intracranial pressure is made. Hearing was never quantified. Blood cultures were not taken and the suspected immunocompromised state was never characterised. The Gram stain reported both Gram-positive and Gram-negative organisms while culture grew only *S. suis*, and that discrepancy is unresolved. The eye was not examined for 13 days after a postoperative pressure of 2 mmHg, so the resolution of the hypotony rests on a single later reading. Every conversion scale, threshold and instrument was chosen after the clinical course had closed. Follow-up ended 48 days after onset, which is far too short to describe the final refractive, retinal or pressure status of this eye; secondary glaucoma and hypotony maculopathy after an episode of this kind may both declare themselves much later[47,50,64]. Finally, the reporting unit of this study is a single case; whatever apparatus is applied to it, readers and editors may reasonably regard the case-report category as the more accurate label.

5. Conclusion

In this structured re-analysis of a single culture-proven case of *Streptococcus suis* endogenous endophthalmitis, the raised intraocular pressure proved to be two mechanistically distinct entities rather than one: an inflammatory episode peaking at 29.0 mmHg on day 7, which settled as two hypotensive drugs and the inflammation itself acted together, and a pupillary block from posterior synechiae with iris bombé peaking at 30.0 mmHg on day 16, seen directly at the slit lamp, which fell 11.0 mmHg after laser peripheral iridotomy and the simultaneous reintroduction of timolol. Corticosteroid causation scored 3 of 13 on the Naranjo scale for each episode, a score determined largely by the temporal sequence rather than by the evidence weighed, and the 3-day latency is suggestive against a corticosteroid mechanism without settling it. Acuity improved by 0.70 logMAR overall, a figure identical on the clinical-convention and the measured Berkeley scales because the two agree at both ends of the illness; how that gain divides across the vitrectomy does not survive the change of scale, being 0.40 logMAR before and 0.30 after on the first and 0.10 before and 0.60 after on the second, so the attribution of the improvement to the operation is a property of the conversion rather than of the eye. A clinically important transient loss followed surgery on the clinical-convention scale but not on the measured one; the eye moved one World Health Organization category and remained blind. For Indonesian tertiary practice the operative recommendations are that *S. suis* be requested specifically, with confirmatory identification, when meningitis, hearing loss and a history of raw pork coincide; that intraocular and cerebrospinal specimens be obtained as early as is safe and without ever delaying antimicrobial therapy; and that the pupil and iris contour be examined, and gonioscopy performed where the cornea permits, before pressure-lowering therapy is escalated in any inflamed eye. The measurement this case most needed was none of the technology it lacked but a swinging-flashlight test read on the fellow pupil, and it was not performed. A single case cannot settle the timing of vitrectomy, and this one does not claim to; the next step is a multicentre Indonesian registry large enough to do so.

Declarations

Ethical approval and consent

This study was approved by the Ethical Committee of CMHC Research Center, Indonesia (Approval number 2026/207). Written informed consent for the publication of clinical details was obtained from the patient after recovery from the acute illness. The study was conducted in accordance with the Declaration of Helsinki.

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Conflict of interest

The authors declare that they have no competing interests.

Data availability

The analysis script, the verification harness and the derived results file that generated every number in this manuscript are available from the corresponding author on reasonable request. The clinical record itself cannot be shared, because a single case with this combination of features cannot be de-identified against a reader who knows the referral pathway.

Author contributions

DAID and IGAMJ conceived the study and drafted the manuscript. DAID, IGAMJ, IGARS and NMAS performed the ophthalmological assessment and management. AAASI performed the neurological assessment, directed the systemic antimicrobial and corticosteroid strategy and interpreted the cerebrospinal fluid findings. IGAMJ supervised the analysis. All authors reviewed and approved the final manuscript.

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Generative AI disclosure

The authors declare that no generative artificial intelligence tools were used in the design, analysis, or writing of this manuscript.

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