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CASE REPORT

Neglected Total Rhegmatogenous Retinal Detachment with Proliferative Vitreoretinopathy Coexisting with Bilateral Grade IV Hypertensive Retinopathy in Severe Pre-eclampsia: A Case Report from an Indonesian Archipelagic Province

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ABSTRACT

Background: Pre-eclampsia injures the eye through the same endothelial mechanism that injures the kidney and brain, and its retinal signs track systemic severity. Rhegmatogenous retinal detachment arises by an entirely different mechanism, yet where vitreoretinal care is distant the two may present together in one patient, a coexistence not previously described.

Objective: To describe a previously unreported coexistence of chronic rhegmatogenous retinal detachment and grade IV hypertensive retinopathy of severe pre-eclampsia in one patient, and to set out the structural features that distinguish the two mechanisms at the bedside — the distinction that determines whether treatment is medical or surgical.

Case presentation: A 36-year-old woman was examined three weeks after a term vaginal delivery, four months after sudden visual loss in the left eye that began in the fifth month of a pregnancy complicated by pre-eclampsia. She also reported 19 years of unassessed visual loss in the right eye. Blood pressure was 154/114 mmHg. Acuity was hand movement right and 0.1 decimal left. The right eye showed total retinal detachment with a break at 9 o'clock, fixed folds, pigment clumping, grade C posterior proliferative vitreoretinopathy, posterior subcapsular cataract and a 7-degree sensory exotropia; the left eye showed optic disc swelling, peripapillary haemorrhage and a macular star. Both eyes met Scheie grade IV. Left macular tomography gave a central subfield of 182 micrometres and a minimum foveal thickness of 98, with five of nine sectors below the first percentile. No relative afferent pupillary defect was detectable.

Conclusion: Chronic rhegmatogenous and hypertensive retinal disease can coexist in one patient and must be separated structurally, because only one responds to blood pressure control. Sub-percentile macular thinning tempers the expectation of full recovery, and bilateral disease can abolish the relative afferent pupillary defect that would otherwise flag a blind eye.

1. Introduction

Hypertensive disorders of pregnancy are among the most frequent medical complications of gestation and remain a leading contributor to maternal morbidity worldwide. In a retrospective national database analysis of 1,324,424 pregnancies, 29.2% of women had at least one sign or symptom of suspected pre-eclampsia and 14.2% received a formal diagnosis, and confirmed disease carried a three- to fourfold hazard of hypertension-related severe maternal morbidity.[1] Because pre-eclampsia is fundamentally a disorder of maternal endothelium, its consequences are not confined to the kidney, liver and brain. The eye offers uniquely direct access to the same microvascular process, and the retinal findings are neither incidental nor cosmetic. A hospital-based cross-sectional study of 120 women with pre-eclampsia or eclampsia found ocular symptoms in 35.83% and fundus changes in 33.33%,[2] while a cross-sectional study of 216 women attending hospitals in south-western Uganda found retinopathy in 60.2%, with severe hypertension independently associated (adjusted odds ratio 2.8, 95% confidence interval 1.36-5.68).[3] In a prospective cohort of 258 pre-eclamptic women, increasing retinopathy grade was significantly associated with low birth weight and preterm delivery,[4] and in a further prospective series of 94 women retinopathy severity tracked the severity of the hypertensive disorder itself.[5] Funduscopy is therefore a cheap, repeatable index of systemic disease severity that is available wherever an ophthalmoscope is.

The retinal detachment that pre-eclampsia produces is serous. Endothelial dysfunction and intense arteriolar vasospasm compromise the choriocapillaris, the outer blood-retinal barrier fails, and fluid accumulates between the neurosensory retina and the retinal

pigment epithelium. Spectral-domain optical coherence tomography of 30 affected eyes showed subretinal fluid in every eye, macular lesions in 83.33%, ellipsoid zone disruption in 66.67% and Elschnig spots in 66.67%, while intraretinal fluid was present in only 6.67%.[6] The choroid itself is measurably altered: subfoveal choroidal thickness fell from 424.4 to 286.0 micrometres after delivery in a series of 13 eyes with serous detachment,[7] and eyes with retinal change had a thicker choroid than pre-eclamptic eyes without retinal change or normal pregnant controls.[8] The direction of that change is not universally agreed – a comparative study of 90 women found the thinnest subfoveal choroid in the pre-eclamptic group[9] – and the discrepancy is probably one of timing relative to delivery and of the severity of the proteinuric insult, since women with a high protein-to-creatinine ratio have both a thicker antepartum choroid and greater postpartum thinning.[10] Timing matters as much as mechanism: in 40 women imaged within 72 hours of delivery, plane-wave Doppler of the short posterior ciliary arteries correlated moderately with angiographic measures, yet the angiographic parameters themselves no longer differed significantly between pre-eclamptic women and controls at that postpartum time point, which indicates that the choroidal abnormality is dynamic and may already be resolving by the time imaging is obtained after delivery.[11] Clinically, this lesion usually behaves well: serous macular detachment resolved in all 38 affected eyes of a prospective series after delivery,[12] and spontaneous recovery without any ocular intervention has been documented repeatedly.[13,14] Yet recovery is not guaranteed. Permanent concentric field loss has been described after the macular detachment itself had resolved,[15] and massive retinal and choroidal infarction in pre-eclampsia with malignant hypertension left only partial recovery at two months.[16]

Rhegmatogenous retinal detachment shares none of this mechanism. It begins with a full-thickness retinal break, progresses by liquefied vitreous tracking into the subretinal space, and does not respond to blood pressure control. When it is left untreated it acquires the signature of chronicity, as detailed in Table 4: demarcation lines, pigment clumping, fixed folds, secondary cataract and, in eyes blinded early enough, sensory strabismus. Proliferative vitreoretinopathy supervenes in most such eyes. Among 37 eyes repaired after a mean detachment duration of 136.7 days, 70.3% had proliferative vitreoretinopathy of grade C or worse and single-surgery anatomic success was only 54.1%.[17] Presentation at this stage is not evenly distributed across populations. In a cohort of 1,092 adults, macula-off presentation was more likely with public than private insurance and less likely with higher income,[18] and national geospatial mapping has shown that the communities furthest from optometric and ophthalmic services are precisely those with the highest deprivation and the largest indigenous populations.[19] A nationwide register study of 28,320 adults with retinal disease found unilateral blindness in 18% and significantly higher risk in women and in less-developed regions.[20]

These two literatures have developed in parallel and, to the best of our knowledge, have not previously intersected in a single patient. We report a woman in whom a neglected total rhegmatogenous retinal detachment with advanced proliferative vitreoretinopathy in one eye was discovered at the same visit as florid grade IV hypertensive retinopathy of severe pre-eclampsia in the other. The novelty of this report is threefold. First, it documents the simultaneous presence of two mechanistically unrelated forms of retinal disease in the two eyes of one patient and sets out the structural criteria that separate them at the bedside, a distinction that determines whether the correct treatment is an antihypertensive drug or a vitrectomy theatre. Second, it quantifies, using nine-sector macular mapping, a pattern of sub-percentile central and inner-ring retinal thinning in the pre-eclamptic eye that argues against the customary expectation of complete visual recovery and that has not been reported in this context. Third, it draws attention to a pupillary pitfall: a total retinal detachment with hand-movement vision produced no detectable relative afferent pupillary defect because the fellow eye carried its own optic neuropathy. The aim of this report is therefore to describe the clinical, imaging and system-level features of this coexistence; to derive from them a set of discriminating criteria and a practical referral sequence; to situate the case in an archipelagic province without a vitreoretinal service and trace, step by step, the points at which the referral pathway failed; and to argue for routine funduscopy in women with severe features of pre-eclampsia in peripheral settings.

2. Case Presentation

History and obstetric course

Table 1 presents the demographic, obstetric and general clinical characteristics of the patient. A 36-year-old woman attended the eye clinic three weeks after delivery with a chief complaint of sudden, painless loss of vision in the left eye that had been present for four months and had begun during the fifth month of her pregnancy. During that pregnancy she had been diagnosed with pre-eclampsia by her obstetrician and had been treated with aspirin, nifedipine and multivitamin supplementation. As detailed in Table 1, she had completed eight antenatal visits, and the pregnancy ended in a term spontaneous vaginal delivery of a live neonate weighing 2,900 g who cried spontaneously at birth. She did not experience seizures at any point, and there had been no headache or alteration of consciousness.

The patient separately reported loss of vision in the right eye of approximately 19 years' duration, for which she had never sought or received medical

evaluation or treatment, together with a progressive outward deviation of that eye. She denied any history of ocular trauma, high myopia, previous intraocular surgery, hypertension antedating the pregnancy, or other systemic disease. The relevant negatives are listed in Table 1, and the interval between the onset of left-eye symptoms and this first ophthalmic assessment was approximately four months.

Table 1. Demographic, obstetric, and general clinical characteristics of the patient at the index ophthalmic visit.

Characteristic	Finding
Age	36 years
Sex	Female
Obstetric status at presentation	3 weeks postpartum, following a term spontaneous vaginal delivery
Antenatal care	8 antenatal visits during the index pregnancy
Hypertensive disorder	Pre-eclampsia, diagnosed by the attending obstetrician during the pregnancy
Antenatal treatment received	Aspirin, nifedipine, multivitamin supplementation
Neonatal outcome	Live-born neonate, 2,900 g, spontaneous cry at birth
Presenting complaint, left eye	Sudden painless visual loss for 4 months, onset in the fifth month of gestation
Presenting complaint, right eye	Painless visual loss for approximately 19 years, never evaluated; progressive outward deviation
Neurological symptoms	None: no headache, no seizure, no alteration of consciousness
Relevant negative history	No ocular trauma, high myopia, previous intraocular surgery, pre-pregnancy hypertension or other systemic disease
Blood pressure at presentation	154/114 mmHg (severe-range diastolic elevation persisting 3 weeks postpartum)
Mean arterial pressure*	127 mmHg
Interval to first ophthalmic assessment	Approximately 4 months from the onset of left-eye symptoms

* Mean arterial pressure calculated as (systolic pressure + 2 x diastolic pressure) / 3.

General and ocular examination

Blood pressure at presentation was 154/114 mmHg, giving a calculated mean arterial pressure of 127 mmHg and a diastolic value within the severe range that persisted three weeks after delivery. The comparative ocular findings of the two eyes are set out in Table 2. Visual acuity was hand movement in the right eye and 0.1 on decimal Snellen testing in the left eye, the latter corresponding to 20/200 or 1.0 logMAR; the left eye was therefore the only eye with any useful form vision, and it was the eye that the hypertensive disorder had damaged. Intraocular pressure was within normal limits in both eyes at 18 mmHg on the right and 19 mmHg on the left. As shown in Table 2, pupillary light reflexes were present bilaterally but sluggish, and no relative afferent pupillary defect could be elicited on swinging-flashlight testing despite the profound asymmetry of acuity.

The acuities recorded above are presenting acuities; a formal refraction was not performed at this visit, so the exclusion of high myopia rests on the history rather than on a measured refractive error. The laboratory parameters that define the severity of pre-eclampsia – quantified proteinuria, platelet count, transaminases and serum creatinine – were not available in the referral documentation and were not repeated at this visit, so the systemic classification rests on the obstetric diagnosis made during the pregnancy together with the persistent severe-range blood pressure recorded here.

Ocular alignment assessment demonstrated approximately 7 degrees of right exotropia on Hirschberg testing, equivalent to roughly 12 prism dioptres, with medial refixation movement observed on both the cover-uncover and the alternating cover test, confirming a manifest deviation. Anterior segment examination showed a posterior subcapsular opacity of the right lens; the left lens was clear. Funduscopy of the right eye revealed a round optic disc with blurred margins, an arteriovenous ratio of 1:3 against a normal ratio of 2:3, a flame-shaped haemorrhage along the superotemporal vascular arcade, and an area of peripheral hyperpigmentation at the 7 o'clock position. An extensive 360-degree retinal detachment was present, with a retinal break of approximately one clock hour at the 9 o'clock temporal meridian, accompanied by fixed retinal folds and clumping of pigmented cells at the 1 to 2 o'clock position. Funduscopy of the left eye demonstrated a round optic disc with blurred margins, a peripapillary haemorrhage, and a complete macular star; the peripheral retina of the left eye was attached, and no retinal break, lattice degeneration or posterior vitreous detachment was documented. Both eyes therefore satisfied Scheie grade IV, the grade defined by the presence of optic disc swelling. The arteriovenous ratio was formally graded only in the right eye, so the assignment of grade IV to the left eye rests on the disc swelling and the peripapillary haemorrhage rather than on a recorded arteriolar calibre; these grading assignments are recorded in the final rows of Table 2.

Table 2. Comparative ocular examination and imaging findings of the right and left eye at the index visit.

Parameter	Right eye	Left eye
Presenting visual acuity*	Hand movement	0.1 decimal (20/200; 1.0 logMAR)
Intraocular pressure	18 mmHg	19 mmHg
Pupillary light reflex	Present, sluggish; no RAPD	Present, sluggish; no RAPD
Ocular alignment	Exotropia, approximately 7 degrees (about 12 prism dioptres); medial refixation on cover-uncover and alternate cover testing	Fixing eye
Lens	Posterior subcapsular opacity	Clear
Optic disc	Round, blurred margins	Round, blurred margins
Arteriovenous ratio	1:3 (normal 2:3)	Not separately graded
Retinal haemorrhage	Flame-shaped, superotemporal arcade	Peripapillary
Macula	Obscured by detached retina	Complete macular star (hard exudates)
Peripheral retina	360-degree detachment; break of approximately 1 clock hour at the 9 o'clock temporal meridian; fixed retinal folds; pigment clumping at 1 to 2 o'clock; hyperpigmentation at 7 o'clock	Attached throughout; no break, lattice degeneration or posterior vitreous detachment documented
Proliferative vitreoretinopathy	Grade C posterior	Absent
B-scan ultrasonography	V-shaped membrane inserting at the disc: total retinal detachment	Not indicated
Macular optical coherence tomography	Not obtainable (total detachment and lens opacity)	Outer retinal hyper-reflective foci; see Table 3
Hypertensive retinopathy, Scheie grade†	Grade IV	Grade IV

RAPD = relative afferent pupillary defect.
* Presenting acuity; a formal refraction was not performed at the index visit.
† Scheie grade IV denotes the changes of grade III together with optic disc swelling.

Multimodal imaging

Figure 1 illustrates the fundus of the right eye. As shown in Figure 1A, the optic disc margins were blurred in an eye whose retina was wholly detached, a combination that on its own would be difficult to attribute to either process. Figure 1B shows the flame-shaped haemorrhage along the superotemporal arcade, marked with an open square, together with the peripheral hyperpigmented area marked with a cross. The causative break is shown in Figure 1C, where a defect of approximately one clock hour lies at the 9 o'clock temporal meridian immediately adjacent to a fixed retinal fold. Figure 1D shows the clumps of pigmented cells lying nasally at the 1 to 2 o'clock position, indicated by arrowheads. Taken together, the features in Figure 1C and Figure 1D are the morphological signature of a detachment of long standing rather than of recent onset.

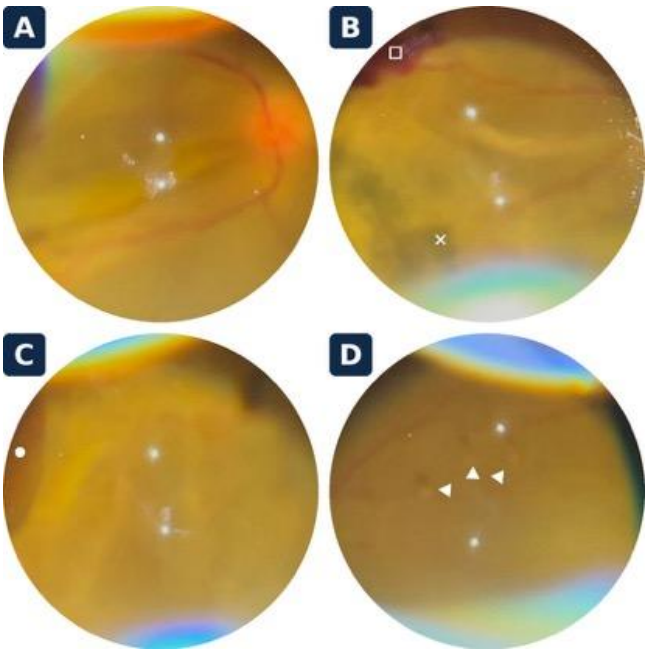

Figure 1. Fundus photographs of the right eye at the index visit. (A) Optic disc with blurred margins in an eye with total retinal detachment. (B) Flame-shaped retinal haemorrhage along the superotemporal vascular arcade (open square) and an area of peripheral hyperpigmentation (cross). (C) Retinal break of approximately one clock hour at the 9 o'clock temporal meridian with an adjacent fixed retinal fold (filled circle). (D) Clumps of pigmented cells nasally at the 1 to 2 o'clock position (arrowheads). Images were acquired through a condensing lens and are reproduced at the native resolution of the source photographs.

Figure 2 presents the panoramic fundus photograph of the left eye. The optic nerve head is swollen with indistinct margins and a peripapillary haemorrhage, and a dense, complete macular star of hard exudates radiates outward from the fovea. As shown in Figure 2, the retinal arterioles show no cotton-wool spots and no exudative detachment was present at this visit, so that the exudate seen here represents the residue of a process that had already run its course rather than an active one.

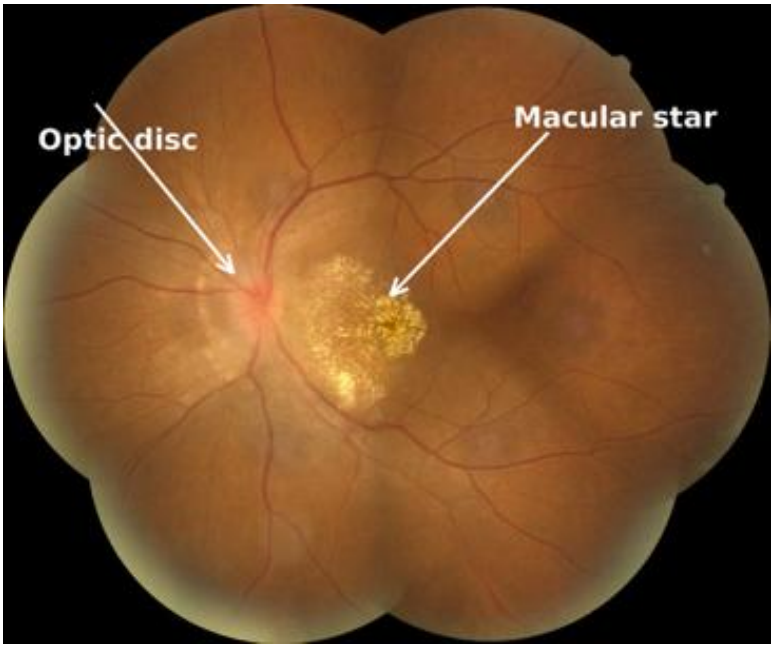

Figure 2. Panoramic fundus photograph of the left eye showing swelling of the optic nerve head with blurred margins, a peripapillary haemorrhage, and a complete macular star of hard exudates radiating from the fovea along the fibres of the Henle layer. No subretinal fluid was present at this visit.

Figure 3 shows B-scan ultrasonography of the right eye. A highly reflective V-shaped membrane inserts at the optic nerve head and extends anteriorly toward the ora serrata in both meridians, the configuration of a total retinal detachment. As demonstrated in Figure 3, no intraocular mass, no choroidal thickening and no posterior scleral nodule were identified, which excluded the exudative causes that would otherwise enter the differential diagnosis of a totally detached retina in an eye with opaque or partly opaque media.

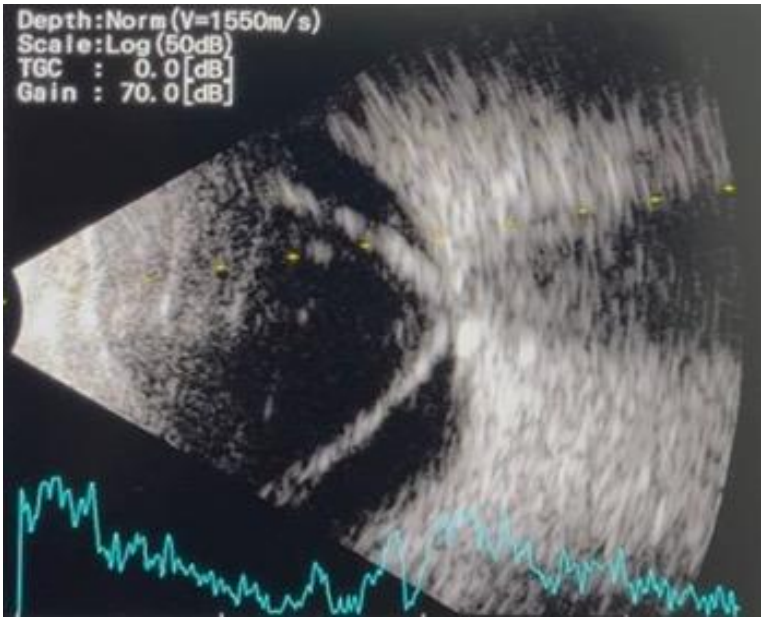

Figure 3. B-scan ultrasonography of the right eye demonstrating a highly reflective V-shaped membrane inserting at the optic nerve head and extending anteriorly, consistent with total retinal detachment. No intraocular mass, choroidal thickening or scleral nodule was identified. Acquisition settings are displayed in the upper left corner of the image, which is reproduced at the native resolution of the instrument printout.

Figure 4 presents macular optical coherence tomography of the left eye, acquired on a Nidek RS-3000 Advance spectral-domain instrument as a 9.0 × 9.0 mm macular map with a 512 × 128 scan pattern and segmented from the internal limiting membrane to the retinal pigment epithelium and Bruch membrane complex. As shown in Figure 4, discrete hyper-reflective foci are present within the outer retinal layers, corresponding to the hard exudates of the macular star seen ophthalmoscopically in Figure 2. The quantitative output of that scan is reproduced in Table 3. The central subfield measured 182 micrometres and the minimum foveal thickness 98 micrometres, and as detailed in Table 3 the central subfield together with the inner nasal, inner temporal and inner inferior sectors and the outer nasal sector all fell below the first percentile of the instrument's normative database, while the inner superior sector lay between the first and fifth percentiles and the three remaining outer sectors were within normal limits. The nine sector volumes listed in Table 3 sum to the reported total macular volume of 7.87 mm^3. The deviation map in the lower right panel of Figure 4 confirms that the negative deviation is centred on the fovea rather than being a segmentation artefact of the peripapillary region.

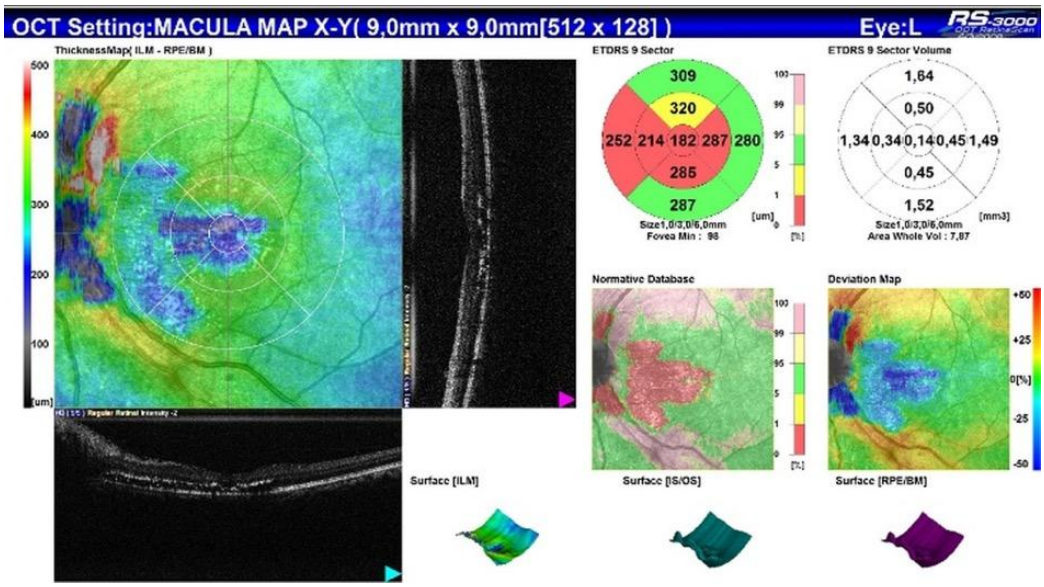

Figure 4. Macular optical coherence tomography of the left eye. The thickness map (upper left) and the accompanying horizontal and vertical B-scans show hyper-reflective foci within the outer retinal layers, corresponding to the hard exudates of the macular star. The nine-sector ETDRS grid (upper middle) gives a central subfield thickness of 182 µm and a minimum foveal thickness of 98 µm; sectors shown in red lie below the first percentile of the normative database, the sector in yellow between the first and fifth percentiles, and sectors in green within normal limits. The corresponding sector volumes are shown in the upper right panel and total 7.87 mm^3. The deviation map (lower right) localises the negative deviation to the fovea.

Table 3. Nine-sector ETDRS macular thickness, sector volume and normative significance of the left eye.*

ETDRS sector	Thickness (µm)	Volume (mm^3)	Normative significance†
Central subfield (1 mm)	182	0.14	Below the 1st percentile
Inner superior	320	0.50	1st to 5th percentile
Inner nasal	214	0.34	Below the 1st percentile
Inner temporal	287	0.45	Below the 1st percentile
Inner inferior	285	0.45	Below the 1st percentile
Outer superior	309	1.64	Within normal limits
Outer nasal	252	1.34	Below the 1st percentile
Outer temporal	280	1.49	Within normal limits
Outer inferior	287	1.52	Within normal limits
Total macular volume‡	—	7.87	—
Minimum foveal thickness	98	—	—

* Nidek RS-3000 Advance spectral-domain instrument; macular map 9.0 × 9.0 mm, 512 × 128 scan pattern, segmentation from the internal limiting membrane to the retinal pigment epithelium and Bruch membrane complex.
† Significance as displayed against the instrument's built-in normative reference database.
‡ The nine sector volumes (0.14 + 0.50 + 0.34 + 0.45 + 0.45 + 1.64 + 1.34 + 1.49 + 1.52) sum to the reported total macular volume of 7.87 mm^3.

Diagnosis, management and disposition

The patient was diagnosed with a long-standing rhegmatogenous retinal detachment of the right eye with proliferative vitreoretinopathy grade C posterior, posterior subcapsular cataract and sensory exotropia; bilateral hypertensive retinopathy of Scheie grade IV; and severe pre-eclampsia with persistent severe-range hypertension three weeks postpartum. The diagnostic

elements are summarised across Table 1, Table 2 and Table 3, and the sequence in which they were assembled is set out in Figure 5. The examination findings behind each element of that list are given in Table 2 and the quantitative macular data behind it in Table 3, while Figure 1, Figure 2, Figure 3 and Figure 4 supply the imaging on which the two diagnoses rest.

Because the systemic disorder was the immediate threat, the patient was referred to an internist at another hospital for further blood pressure management before any ocular intervention was contemplated. Referral to a vitreoretinal service at a tertiary referral centre in a neighbouring province was planned in parallel for further evaluation of the right eye, no vitreoretinal surgical service being available in the province at the time this patient was managed. The left eye required no ocular treatment; its management was the management of the blood pressure. No ocular intervention of any kind was performed at the reporting institution before referral. As shown in Figure 5, the two referral arms were initiated at the same visit, which was also the first occasion on which either eye had been examined by an ophthalmologist. Written informed consent for publication of this case report and the accompanying images was obtained from the patient.

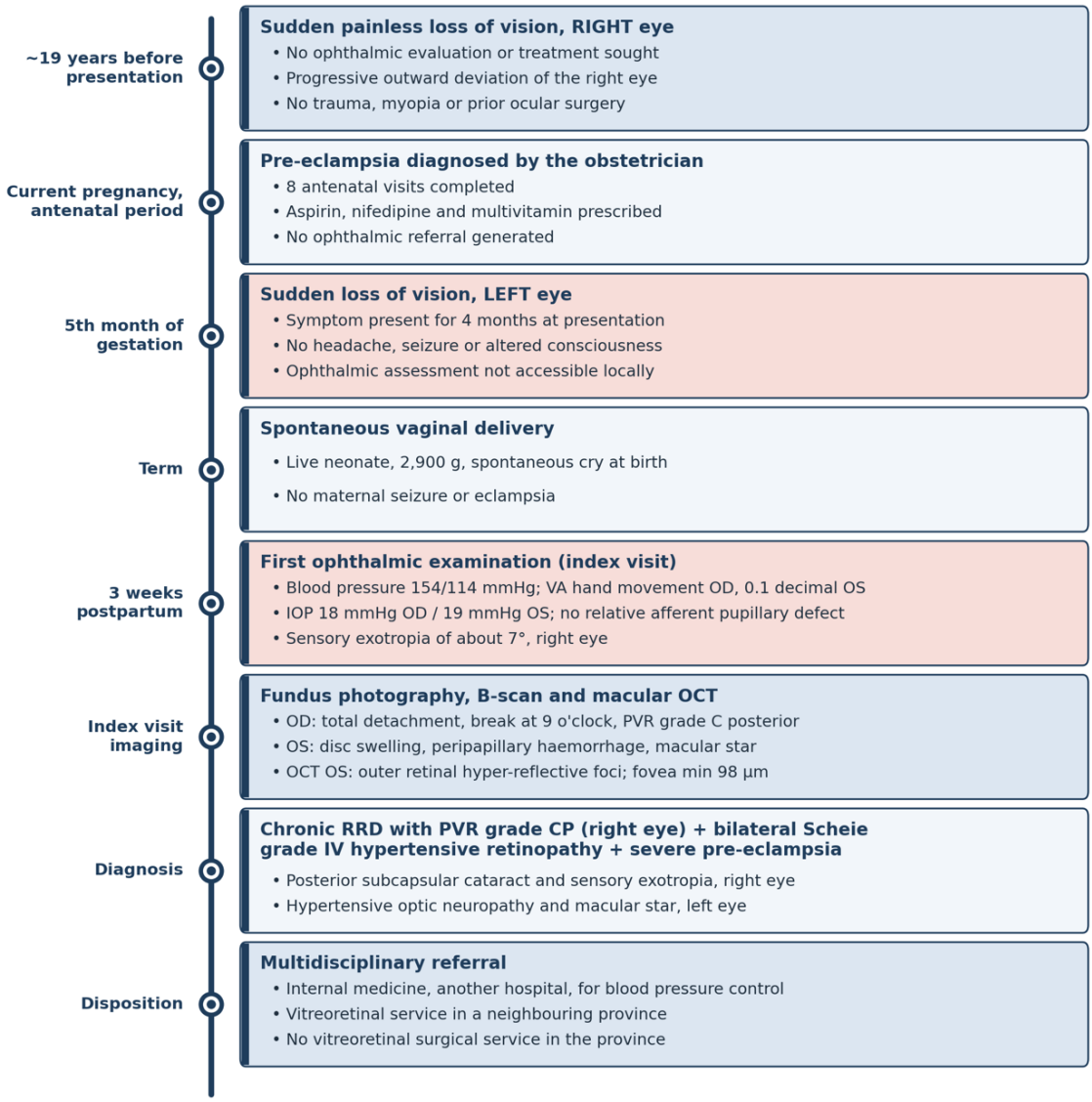

Figure 5. Clinical timeline from the first symptom in the right eye, approximately 19 years before presentation, through the index pregnancy and delivery, to the multidisciplinary referral made at the first ophthalmic examination three weeks postpartum.

3. Discussion

Two detachments, two mechanisms, one patient

The central clinical problem posed by this patient was not the diagnosis of either disease in isolation but the recognition that two unrelated diseases were present at once and that the signs of one were being written on top of the other. Severe pre-eclampsia produces serous retinal detachment through choroidal ischaemia and failure of the outer blood-retinal barrier, and the choroidal circulation appears to be affected before the retinal vasculature because the choroidal arteries are short, relatively unbranched and poorly autoregulated,

so that systemic pressure is transmitted more directly to the choriocapillaris; the measurable consequence is a choroid that is thickest in precisely those pre-eclamptic eyes that also show retinal change.[8] Optical coherence tomography of pre-eclamptic eyes shows subretinal fluid in essentially all affected eyes with intraretinal fluid in fewer than one in fifteen,[6] and the choroid thickens acutely before thinning after delivery.[7,8,10] None of these features describes the right eye of the present patient. What the right eye showed instead was a full-thickness retinal break at the 9 o'clock meridian, fixed retinal folds, pigment clumping and proliferative vitreoretinopathy of grade C posterior, in an eye that had been blind for approximately 19 years and had developed a secondary cataract and a sensory deviation. Table 4 sets out the discriminating features of the two entities and applies them to this patient.

The discriminators set out in Table 4 are worth stating explicitly because they are all obtainable with a condensing lens and an indirect ophthalmoscope. A causative break, fixed rather than shifting subretinal fluid, retinal folds, pigment clumping or a demarcation line, secondary lens opacity and a sensory strabismus each point to a rhegmatogenous process; bilaterality, gravity-dependent fluid, an absent break, Elschnig spots and prompt resorption after blood pressure control point to a hypertensive serous process. As demonstrated in Table 4, the present patient displayed the rhegmatogenous set in the right eye and the hypertensive set in the left, and it is the coexistence rather than either individual pattern that is novel. The clinical stake is high, because the two require entirely different responses. The hypertensive lesion is treated by the physician who lowers the blood pressure; series of pre-eclamptic serous detachment show complete resolution in every affected eye after delivery,[12] both patients of a recent two-case series recovered vision completely within two weeks on medical treatment alone,[14] and a woman whose postnatal acuity was finger counting at one to two metres reached 8/10 in both eyes at one month with no ocular treatment whatever.[13] The rhegmatogenous lesion is treated in an operating theatre and, in this eye, in a different province.

One feature deserves separate comment because it is genuinely ambiguous. The right eye also showed blurred optic disc margins, an arteriovenous ratio of 1:3 and a flame-shaped haemorrhage along the superotemporal arcade, illustrated in Figure 1A and Figure 1B. These are hypertensive signs, not rhegmatogenous ones. The most parsimonious interpretation is that the systemic hypertensive insult was superimposed on an eye whose retina was already structurally compromised, so that the right eye carried both diseases and the left carried only one. This matters for grading, since the assignment of Scheie grade IV to the right eye rests on optic disc swelling that is at least partly attributable to the hypertensive process rather than to the detachment. It matters more for what could not be seen: a total detachment with advanced proliferative vitreoretinopathy would conceal any serous component that pre-eclampsia might have added, so the absence of documented serous detachment in the right eye is uninformative rather than reassuring.

Table 4. Discriminating features of chronic rhegmatogenous and hypertensive serous retinal detachment, applied to the present patient.

Feature	Chronic rhegmatogenous detachment	Hypertensive serous detachment	Present patient
Causative retinal break	Present, by definition	Absent	Present: approximately 1 clock hour at 9 o'clock, right eye
Onset	Often insidious, unnoticed	Acute, with a systemic hypertensive crisis	Right eye approximately 19 years; left eye in the fifth gestational month
Laterality	Usually unilateral	Typically bilateral	Detachment unilateral (right); hypertensive signs bilateral
Subretinal fluid	Non-shifting; fixed folds develop	Shifting and gravity-dependent	Fixed retinal folds, right eye
Proliferative vitreoretinopathy	Characteristic of chronicity	Not a feature	Grade C posterior, right eye
Pigmentary change	Demarcation line, pigment clumping	Absent acutely; Elschnig spots later	Pigment clumping at 1 to 2 o'clock; hyperpigmentation at 7 o'clock
Anterior segment sequelae	Secondary cataract, iris atrophy, hypotony	None	Posterior subcapsular cataract, right eye
Ocular alignment	Sensory strabismus when long-standing	Normal	Sensory exotropia of approximately 7 degrees, right eye
Response to blood pressure control	None	Resorption of subretinal fluid expected	Applicable to the left eye only
Definitive treatment	Vitreoretinal surgery	Systemic antihypertensive therapy	Both required, in different eyes

Hypertensive optic neuropathy, choroidopathy and the macular star

The left eye showed the classical triad of accelerated hypertensive injury to the posterior pole: optic disc swelling with blurred margins, peripapillary haemorrhage, and a complete macular star. As shown in Figure 2, the exudate radiates from the fovea in the spoke pattern imposed by the obliquely oriented fibres of the Henle layer, which is the anatomical explanation for the star configuration rather than a separate disease process. The sequence is well described in the imaging literature on malignant hypertension: disc swelling, flame haemorrhages, cotton-wool spots and Elschnig spots accompany disruption of the superficial and deep capillary plexuses,[21] and a focal choriocapillaris non-perfusion defect can be the sole manifestation, producing a strictly unilateral exudative detachment; over nine months of follow-up in that patient the blood pressure normalised, visual function returned and choriocapillaris perfusion was completely restored.[22] Lipid-rich fluid that has leaked during the acute phase is resorbed more slowly than water, and its residue precipitates as hard exudate in the outer plexiform layer during the resolution phase. A macular star seen months after the acute event is therefore best read as a footprint of a serous detachment that has already resolved, not as evidence of ongoing leakage. That interpretation is consistent with what was found in this patient: as shown in Figure 2 there was no residual subretinal fluid, yet the exudate was dense and complete.

The systemic context supports this reading. Blood pressure remained at 154/114 mmHg three weeks after delivery, which is not an unusual finding; in a prospective cohort of 190 women with pre-eclampsia, persistent hypertension affected 37.4% at six weeks postpartum and still affected 16.9% at six months.[23] Persistent postpartum hypertension is not merely a residue of the pregnancy either. Six years after delivery, women with previous pre-eclampsia had measurably reduced endothelial glycocalyx thickness together with higher blood pressure and greater blood pressure variability than controls,[24] which is why the referral of this patient to an internist rather than a return obstetric visit was the correct disposition.

What the optical coherence tomography adds: exudate above, thinning beneath

The most informative single investigation in this patient was the macular scan of the left eye, and its quantitative output is the finding most likely to have been passed over. Two things are shown in Figure 4, and they point in opposite directions. The first is expected: hyper-reflective foci within the outer retinal layers, the tomographic correlate of the hard exudates seen ophthalmoscopically. The second is not. As detailed in Table 3, the central subfield measured 182 micrometres and the minimum foveal thickness 98 micrometres, and five of the nine ETDRS sectors - the central subfield and the inner nasal, inner temporal and inner inferior sectors, together with the outer nasal sector - lay below the first percentile of the instrument's normative reference database, with a sixth sector between the first and fifth percentiles. Only the outer superior, outer temporal and outer inferior sectors were within normal limits. In other words, beneath a macula that looked florid and exudative on colour photography, the retina was measurably thin.

The limitation must be stated before the inference. This is a single time point with no pre-morbid baseline, the patient is her own only control, and hard exudates are hyper-reflective structures that can shadow underlying layers and bias automated segmentation of the retinal pigment epithelium boundary. A single sub-percentile sector would prove nothing. What is harder to dismiss is the pattern: the thinning is centred on the fovea, it involves the whole inner ring rather than one quadrant, the outer ring is largely spared, and the deviation map in Figure 4 localises the negative deviation to the fovea rather than to the peripapillary area where segmentation errors cluster. Exudate-related shadowing would be expected to produce artefactual thickening or focal irregularity, not a symmetrical, fovea-centred deficit with a preserved outer ring. A second caveat concerns the reference standard rather than the measurement: the percentile assignments are made against the built-in normative database of the instrument, whose reference population is not matched to this patient, and ethnic variation in macular thickness is well recognised. The argument therefore rests on the internal pattern of the deficit as much as on the percentile labels themselves.

Read that way, the finding carries a prognostic consequence. The prevailing expectation is that the visual disturbance of pre-eclampsia recovers once the hypertensive disorder is treated, and the published trajectories support it in most patients.[12,13,14,25] But recovery is a function of outer retinal integrity, not of subretinal fluid clearance. The outcome column of Table 5 makes that point empirically: the reports in which vision recovered completely are those in which the insult was purely serous and brief, whereas the two reports of permanent deficit are those in which infarction or a more prolonged insult intervened. Ellipsoid zone disruption was demonstrable in two thirds of pre-eclamptic eyes with serous detachment even at the time of diagnosis,[6] and in 187 eyes imaged six weeks after retinal detachment repair the relative reflectivity of the ellipsoid zone was significantly associated with the acuity ultimately achieved.[26] Macular exudate in another disease of raised pressure has been shown to thicken the outer retinal layers and to be associated with worse acuity independently of the disc swelling itself,[27] which establishes the outer retina as the layer where exudate and vision meet. A history of pre-eclampsia has itself been associated with thinner outer retinal subfields a decade or more after delivery, and in that cohort thinner inner nasal and inner

superior quadrants predicted worse executive performance,[28] which is a striking parallel to the specific sectors affected here. The clinical action that follows is concrete: this patient's left eye should not be counselled as certain to recover, her acuity of 0.1 decimal at four months should be treated as a possible endpoint rather than a way-station, and structural macular imaging should be repeated at defined intervals rather than replaced by acuity alone. Cases in which the macular pathology resolved and the visual deficit did not[15,16] are precisely the cases this reasoning predicts.

The absent relative afferent pupillary defect: a bilateral-disease pitfall

As shown in Table 2, no relative afferent pupillary defect was detectable in an eye with a total retinal detachment and hand-movement vision. That combination ordinarily produces an obvious defect, and its absence would, if taken at face value, argue against serious unilateral pathology. The explanation is that the swinging-flashlight test measures a difference between the two afferent pathways, not the absolute integrity of either. The left eye of this patient carried its own hypertensive optic neuropathy with a swollen disc and peripapillary haemorrhage, as shown in Figure 2, so afferent input was reduced on both sides and the interocular difference was correspondingly compressed. The relationship is quantitative: in 52 patients the graded defect correlated with the interocular difference in visual field index ($r = 0.55$, $p < 0.001$), the association following the approximate relation that the interocular visual field index difference equals 16.75 times the grade minus 7.53.[29] When the fellow eye is also damaged, the difference term shrinks and the measured defect shrinks with it.

The practical lesson generalises well beyond this patient. In screening settings where a non-ophthalmologist performs the pupil examination - which is exactly the setting in which women with severe features of pre-eclampsia are first seen - a negative swinging-flashlight test must not be allowed to override a history of monocular visual loss or an abnormal fundus. Bilateral disease is common in hypertensive emergency, and in that context the pupil is an insensitive instrument. The corollary is that direct visualisation of the fundus, not the pupil reflex, is the screening test that should be insisted upon.

Reading chronicity: what the right eye tells us about elapsed time

Several independent features dated the right-eye detachment, and their concordance is what makes the chronology secure. As shown in Figure 1C and Figure 1D, fixed retinal folds and clumping of pigmented cells were present, and Figure 1B shows peripheral hyperpigmentation at the 7 o'clock position of the kind that forms at the margin of a detachment that has been static long enough for the retinal pigment epithelium to react. The lens showed a posterior subcapsular opacity, the anterior segment consequence of a long-standing posterior segment disorder rather than of age in a 36-year-old woman. Proliferative vitreoretinopathy had reached grade C posterior. Intraocular pressure was 18 mmHg rather than the hypotony that a recent and extensive detachment often produces, again suggesting an equilibrium of long standing. As demonstrated in Figure 3, the detached retina had assumed the fixed, funnel-like V configuration on ultrasonography rather than the mobile, undulating membrane of a fresh detachment, and the ultrasonographic appearance of chronic total detachment, including peripheral macrocyst formation, is well documented.[30]

The location of the break is consistent with the slow evolution that this history implies. The defect lay at the 9 o'clock temporal meridian of the right eye, that is, in the horizontal rather than the superior retina. Detachments arising from non-superior breaks progress more slowly because gravity does not assist the spread of subretinal fluid, which delays the moment at which the patient notices a change and, in a patient with no access to examination, delays it indefinitely. The anatomical explanation should not be given more weight than it can carry: a detachment does not remain untreated for nineteen years because of where its break lies, but because no one ever looked. Break location plausibly explains why the patient did not present in the first weeks; the complete absence of examination explains the remaining two decades. Proliferative vitreoretinopathy is the predictable end state of that delay. In a database of 57,264 eyes examined within a month of detachment diagnosis, poor baseline acuity was among the factors independently associated with proliferative vitreoretinopathy,[31] and grade C disease at presentation is what defines the modern surgical problem. Even in expert hands the results are sobering: among 370 eyes with grade C proliferative vitreoretinopathy in an international multicentre study, single-surgery anatomic success reached 86.6% with internal limiting membrane peeling and 73.2% without at three months, falling to 75.2% and 65.3% at six months,[32] and in 112 eyes managed by retinectomy without lensectomy final reattachment reached 99.1% only after a mean follow-up of 29 months, with single-surgery success of 65.2% at six months.[33] Outer retinal folds, a marker of the remodelling that accompanies these eyes, were present in 44.4% of grade C eyes one month after vitrectomy with a mean height of 199.25 micrometres.[34] The 37-eye chronic macula-off series cited earlier reported single-surgery success of 54.1%.[17] Anatomical reattachment in an eye blind for 19 years would therefore be a realistic surgical goal; restoration of useful vision would not.

Sensory exotropia as a chronological witness

The exotropia recorded in Table 2 is not an incidental finding but an independent marker of how long the right eye has been visually useless. Sensory strabismus develops when one eye loses the acuity required to maintain fusion, and in horizontal sensory deviations

the exotropic form predominates: of 1,017 patients with sensory horizontal strabismus identified from 13,252 records, 70.5% were exotropic and 29.5% esotropic, and severe amblyopia was present in 71.5%.[35] A deviation of approximately 7 degrees, equivalent to about 12 prism dioptres, is a modest angle by the standards of that series and is compatible with a deviation that developed in adulthood, after binocular sensory development was complete. This is congruent with a history of visual loss beginning at approximately 17 years of age.

The deviation also has management implications that are easy to overlook when attention is on the retina. Should the right eye eventually be reattached without visual recovery, the exotropia will persist and will remain the patient's most visible disability. Surgical correction of sensory exotropia is effective but its durability is limited: successful alignment was achieved in 51 of 83 patients (61%) at one month, 19 of 32 (59%) at one year and 8 of 16 (50%) at five years,[36] so realistic counselling about drift and the possible need for further surgery belongs in the same conversation as the retinal prognosis. As detailed in Table 2, the deviation was manifest on both cover-uncover and alternate cover testing, which distinguishes it from a latent deviation and confirms that it is a genuine sensory tropia.

Why she presented late: geography, literacy and a broken referral chain

Two separate failures of access are visible in this history, and Figure 5 makes the interval between them explicit. The first spans approximately 19 years, during which a total retinal detachment progressed to grade C proliferative vitreoretinopathy without a single examination. The second spans four months, from the onset of sudden visual loss in the better eye during a pregnancy that was already under medical supervision, to the first funduscopic examination three weeks after delivery. As shown in Figure 5, the second failure is the more instructive one, because the patient was not outside the health system at all: she attended eight antenatal visits, her pre-eclampsia was diagnosed and treated, and she still reached an ophthalmologist only after the acute phase had passed. A new visual symptom in a woman with pre-eclampsia is a severe feature; the referral it should have generated was never made.

The structural determinants are well characterised. Macula-off presentation of rhegmatogenous detachment is significantly more likely among patients with public insurance and significantly less likely as income rises,[18] and national mapping of eye services against population distribution shows that the communities with the longest travel times are those with the highest deprivation and the largest indigenous populations.[19] Even where services exist, uptake is not automatic: in an Indonesian urban population survey, spectacle coverage after cataract surgery was 69.8% overall but 88.2% in the highest income group, and a physician's explicit recommendation raised the odds of uptake roughly twenty-six-fold (odds ratio 25.99, 95% confidence interval 2.59-260.10).[35-37] That last figure is the most actionable in this discussion, because it quantifies something clinicians control directly: an explicit instruction from a treating physician changes patient behaviour by an order of magnitude. The archipelagic geography of this province compounds the problem. Basic ophthalmic care is concentrated in the provincial capital, inter-island travel requires sea or air transport, and no vitreoretinal surgical service existed in the province when this patient was managed, so that even a correctly recognised emergency would have required transfer to another province.

Two feasible remedies emerge. The first is that funduscopy should be embedded in the antenatal care of women with severe features of pre-eclampsia rather than being left to referral, since the examination requires only a dilated pupil and a condensing lens and yields a grading that has been shown to track both maternal severity and fetal outcome.[4,5] The second is asynchronous teleophthalmology. A traumatic hyphema in a child on an island roughly 1,000 km from the mainland was diagnosed by teleconsultation from portable slit-lamp images captured by a general practitioner, transfer was arranged, and acuity recovered from hand movement to 20/20 within six days.[38] The same architecture - image capture by a non-specialist, remote grading by an ophthalmologist, triage of transfer priority - would have identified this patient's macular star in the fifth month of pregnancy, and would have identified her right eye at any time in the preceding two decades.

Management sequence and counselling

The management of this patient can be stated as an explicit ordering. Blood pressure control came first and was delegated to an internist, because a diastolic pressure of 114 mmHg three weeks postpartum is a systemic emergency in its own right and because it is the only treatment the left eye required. The left eye needed no ocular intervention, a point worth emphasising because the florid appearance in Figure 2 invites the opposite conclusion. The right eye was referred for vitreoretinal assessment with the explicit understanding that the realistic goal is anatomical reattachment and the preservation of a comfortable, cosmetically acceptable eye rather than visual rehabilitation, an expectation supported by the grade C outcome data cited above.[32,33] Where retinal detachment surgery must be undertaken during pregnancy, technique can be adapted to the obstetric situation: a foldable capsular buckle scleral buckling procedure performed under subconjunctival anaesthesia in a woman at 28 weeks of gestation avoided general anaesthesia and prolonged supine positioning and achieved 20/20 acuity.[39] That option did not apply here, since the detachment was chronic and the patient already delivered, but it is relevant to

any future pre-eclamptic patient in whom a fresh rhegmatogenous detachment is discovered antenatally. Counselling deserves its own paragraph because this patient's functional situation is more precarious than her diagnosis list suggests, and more precarious than that of any patient listed in Table 5. Her better eye sees 0.1 decimal, which is 20/200. She is the mother of a three-week-old infant. She is, functionally, a patient with one eye, and that eye has been injured by a disease that carries a substantial risk of recurrence in subsequent pregnancies and a documented long-term cardiovascular and microvascular legacy.[24] Counselling should therefore address contraception and the planning of any future pregnancy, sustained blood pressure follow-up beyond the traditional six-week postpartum visit, and the practical safety of an infant carer with severely reduced vision. The risk of recurrent pre-eclampsia in a subsequent pregnancy is the specific hazard being counselled against, and in a woman whose only seeing eye has already been injured by that disease a recurrence would threaten the eye as well as the pregnancy; hypertension persisting months after delivery, which this patient may prove to have,[23] identifies precisely the group in whom that conversation is most urgent. Table 5 compares the present case with recently reported cases of ocular complications of pre-eclampsia and related hypertensive disorders, and the contrast is instructive: as detailed in Table 5, in every published case the ocular problem was unilateral or bilateral but singular, and it usually resolved.

Differential diagnosis and alternative explanations

Three alternatives were considered and can be set aside. First, that both eyes carried hypertensive serous detachment and the right eye simply looked different because it was worse. This fails because a serous detachment has no retinal break, and a break of approximately one clock hour was directly visualised, as shown in Figure 1C, and because every rhegmatogenous discriminator listed in Table 4 was satisfied by that eye; it also fails because serous fluid does not produce proliferative vitreoretinopathy, fixed folds or a secondary cataract, and because a 19-year history antedates the pregnancy by nearly two decades. Second, that the right eye harboured an exudative detachment from an intraocular tumour or an inflammatory cause. As demonstrated in Figure 3, ultrasonography showed no mass, no choroidal thickening and no scleral nodule, and the presence of a break together with pigment clumping is not the morphology of an exudative process. Third, that the visual loss in pregnancy had a non-hypertensive cause. Acute persistent vision loss in pregnancy can arise from mechanisms entirely unrelated to pre-eclampsia; Purtscher-like retinopathy has been described in a woman at 20 weeks with pancreatitis and hypercalcaemia whose blood pressure was normal and who had no proteinuria.[39,40] In the present patient the blood pressure was 154/114 mmHg at examination, pre-eclampsia had been diagnosed independently by the obstetrician during the pregnancy, and the fundus appearance in Figure 2 was that of accelerated hypertension, so the hypertensive attribution is secure. The value of the comparison is that it identifies the question a clinician must ask before making that attribution.

Table 5. Comparison of the present case with recently reported cases of ocular complications of pre-eclampsia and related hypertensive disorders of pregnancy.

Report	Age and obstetric status	Presenting acuity	Principal ocular findings	Reported outcome
Calisir et al., 2022[13]	29 years, day 1 postpartum	Finger counting at 1 m (right) and 2 m (left)	Bilateral bullous serous retinal detachment	8/10 in both eyes at 1 month, no ocular treatment
Munsell et al., 2023[41]	37 years, 38 weeks	20/800, left eye	Exudative detachment, ciliochoroidal effusion, angle closure; pressure 26 mmHg	20/60 at 1 month after delivery
Abdouli et al., 2024[15]	43 years, delivered at 33 weeks	Reduced, both eyes	Bilateral serous macular detachment with HELLP syndrome	Permanent concentric field defect despite macular resolution
Li and Qu, 2025[25]	26 weeks, pregnancy unrecognised	Light perception, both eyes	Exudative detachment with hypertensive choroidopathy, suspected HELLP	0.8 in both eyes at 4 months
Viswanath et al., 2025[16]	Primigravida, third trimester	Sudden loss	Hypertensive retinochoroidopathy with massive retinal and choroidal infarcts	Partial recovery only, at 2 months
Oumaiouf et al., 2026[14]	Two patients, severe pre-eclampsia	Severely reduced, both eyes	Bilateral serous retinal detachment	Complete visual recovery within 2 weeks in both patients
Wu et al., 2026[39]	36 years, 28 weeks	20/40, left eye	Macula-involving rhegmatogenous detachment during pregnancy	20/20 after capsular buckle scleral buckling; healthy infant at 38 weeks
Present case	36 years, 3 weeks postpartum	Hand movement and 0.1 decimal	Chronic total rhegmatogenous detachment, PVR grade C posterior (right eye); grade IV hypertensive retinopathy with macular star (left eye)	Referred; outcome not available

HELLP = haemolysis, elevated liver enzymes and low platelet count.

Strengths and limitations

The principal strength of this report is that both eyes were documented at the same visit with colour fundus photography, ultrasonography and quantitative optical coherence tomography, so that the two disease processes can be compared within one patient and one imaging session rather than across separate reports. The quantitative macular data reproduced in Table 3 permit an inference that qualitative description would not support.

The limitations are substantial and should temper every inference drawn above. The laboratory parameters that define the severity of pre-eclampsia - quantified proteinuria, platelet count, transaminases and creatinine - were not available in the referral documentation, so the systemic classification rests on the obstetric diagnosis made during the pregnancy together with the persistent severe-range blood pressure recorded at the index visit. Formal perimetry, fundus autofluorescence and angiography were not obtainable in this setting, so the functional extent of the macular damage in the left eye was not mapped. The right eye could not be imaged tomographically because of the total detachment and the lens opacity, so no direct structural comparison between the two maculae is possible. The macular thickness measurements represent a single time point without a pre-morbid baseline and without a normative comparison drawn from a matched local population, and automated segmentation in the presence of dense hard exudate has known limitations. The 19-year duration of the right-eye detachment is a patient report and cannot be corroborated by any record. Follow-up beyond the index visit is not available, since the patient was referred out of the province and no ocular intervention was performed before that referral, so the eventual anatomical and visual outcomes of both eyes are unknown; no mechanism existed to retrieve the outcome from a centre in another province, although the authors intend to report it should it become available. Finally, this is a single patient; the comparison presented in Table 5 is with published reports rather than with a matched series, and the associations proposed between sub-percentile macular thinning and limited visual recovery, and between bilateral optic neuropathy and an abolished relative afferent pupillary defect, are mechanistic hypotheses generated by this case and require prospective confirmation in a cohort with serial imaging.

4. Conclusion

This patient carried two retinal diseases that shared nothing but a single patient and a single clinic visit. A neglected total rhegmatogenous retinal detachment with grade C posterior proliferative vitreoretinopathy, secondary cataract and sensory exotropia occupied the right eye and had done so for approximately 19 years; florid grade IV hypertensive retinopathy of severe pre-eclampsia, with disc swelling, peripapillary haemorrhage and a complete macular star, occupied the left. Separating them at the bedside required no technology beyond a condensing lens, and the separation determined everything that followed, because only one of the two responds to an antihypertensive drug. Three lessons generalise. First, a macular star months after the acute event marks a serous detachment that has already resolved, and the sub-percentile central and inner-ring macular thinning found beneath it in this patient argues that recovery in such eyes should be verified structurally rather than assumed. Second, the absence of a relative afferent pupillary defect does not exclude a blind eye when the fellow eye is also diseased, and in hypertensive emergency the fundus, not the pupil, is the screening test to insist upon. Third, this patient was never outside the health system; she attended eight antenatal visits and still reached an ophthalmologist four months after losing vision in her only useful eye. Embedding funduscopy in the antenatal care of women with severe features, and building asynchronous teleophthalmology into island referral pathways, would have changed the trajectory of both eyes.

Declarations

Ethical approval

This case report was prepared in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. Formal ethical approval is not required for the publication of a single de-identified case report at the reporting institution.

Informed consent

Written informed consent for the publication of this case report and the accompanying clinical images was obtained from the patient. No information that could identify the patient is included in this report.

Data availability

All data supporting the findings of this report are contained within the article. The de-identified source images are available from the corresponding author on reasonable request.

Conflict of interest

The authors declare that they have no competing interests relevant to the content of this article.

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Author contributions

D.J.S. and I.W.A.P. examined the patient and acquired the clinical photographs, the ultrasonogram and the macular tomography. W.Y. reviewed the imaging, assembled the clinical data and drafted the manuscript. A.A. provided the vitreoretinal interpretation, the grading of proliferative vitreoretinopathy and the

surgical prognostication. All authors critically revised the manuscript for intellectual content and all have read and approved the final version.

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Reference base
All 41 references cited in this article report original patient or participant data. None is a narrative review, systematic review, meta-analysis, guideline or editorial. All were published between 2022 and 2026, and every digital object identifier was verified against the Crossref registry and the publisher record before submission.

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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