Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Etiological Spectrum, Systemic Associations and Visual Outcomes of Vitreous Haemorrhage in Young Adults: A Six-Year Retrospective Cohort Study from a Tertiary Eye Care Centre in India

Pallavi Goel
Dr. Shroff's Charity Eye Hospital
Arjun Bamel
arjunbamel@gmail.com

Dr. Shroff's Charity Eye Hospital
Anju Bansal
Dr. Shroff's Charity Eye Hospital
Manisha Agarwal
Dr. Shroff's Charity Eye Hospital

Research Article

Keywords: Vitreous haemorrhage, young adults, retinal vasculitis, Eales' disease, ocular trauma, visual outcome

Posted Date: November 12th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11182359/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Etiological Spectrum, Systemic Associations and Visual Outcomes of Vitreous Haemorrhage in Young Adults: A Six-Year Retrospective Cohort Study from a Tertiary Eye Care Centre in India

Pallavi Goel[1], Arjun Bamel[2*], Anju Bansal[3] Manisha Agarwal[4]

[1]Dr. Pallavi Goel, DNB, Consultant, Department of Vitreo Retina and Uvea, Dr. Shroff's Charity Eye Hospital, Delhi,India

[2]Dr. Arjun Bamel, DNB, Consultant, Department of Vitreo Retina and Uvea, Dr. Shroff's Charity Eye Hospital, Delhi,India

[3]Dr. Anju Bansal, DNB, Fellow, Department of Vitreo Retina and Uvea, Dr. Shroff's Charity Eye Hospital, Delhi,India

[4]Dr. Manisha Agarwal, MS, Director, Department of Vitreo Retina and Uvea, Dr. Shroff's Charity Eye Hospital, Delhi,India

*Corresponding author: Dr. Arjun Bamel, Consultant, Department of Vitreo Retina and Uvea, Dr. Shroff's Charity Eye Hospital, Delhi,India. Email: arjunbamel@gmail.com

ABSTRACT

Background: Vitreous haemorrhage in young adults is poorly characterised, because most published series describe paediatric or older adult populations. This study aimed to analyse the demographic profile, etiological spectrum, systemic associations and visual outcomes of vitreous haemorrhage (VH) in young adults aged 18–45 years presenting to a tertiary eye care centre in India.

Methods: A retrospective observational study was conducted on patients aged 18 to 45 years who presented with VH between January 2019 and December 2024 and who completed a minimum of six months of follow-up. Demographic and clinical data, etiology, management and best-corrected visual acuity (BCVA) at presentation and final follow-up were recorded. For analysis the cohort was stratified into four mutually exclusive age bands: 18–20, 21–30,

31–40 and 41–45 years. Associations between age band, etiology and visual outcome were assessed using the chi-square or Fisher exact test, as appropriate.

Results: A total of 408 patients (452 eyes) were included. The mean age was 31.6 ± 9.8 years, with a marked male predominance (343 patients, 84.1%). Involvement was unilateral in 364 patients (89.2%). The commonest etiologies were retinal vasculitis and trauma (144 patients each, 35.3%), followed by proliferative diabetic retinopathy (PDR) (60, 14.7%). Etiology varied significantly across age bands ($p < 0.0001$): trauma predominated at 18–20 years (67.2%), retinal vasculitis was the leading cause at 21–30 years (54.5%) and 31–40 years (38.5%), and PDR became the dominant cause at 41–45 years (52.2%). The proportion with BCVA ≥6/9 improved from 10.5% at presentation to 37.7% at final follow-up ($p < 0.0001$), while severe visual impairment (≤5/60) declined from 66.2% to 35.5%. Retinal vasculitis had the highest proportion achieving a final BCVA of ≥6/9 (46.5%). On multivariable logistic regression, presenting BCVA was the only independent predictor of a final BCVA of ≥6/9 (overall $p < 0.001$); neither age band ($p = 0.188$) nor etiology ($p = 0.226$) remained independently associated after adjustment.

Conclusions: Vitreous haemorrhage in young adults shows a distinct, age-stratified etiological profile in which trauma and inflammatory retinal vasculitis, rather than diabetic retinopathy, account for the majority of cases. Visual outcomes were generally favourable, particularly after vasculitis-related and traumatic haemorrhage. Recognition of age- and region-specific etiologies is essential for targeted evaluation and early intervention.

Keywords: Vitreous haemorrhage; young adults; retinal vasculitis; Eales' disease; ocular trauma; visual outcome

INTRODUCTION

Vitreous haemorrhage (VH) is a common cause of sudden, painless visual loss and results from bleeding into the vitreous cavity from retinal, choroidal or ciliary body sources.[1] In unselected adult populations the condition is dominated by a small number of mechanisms: proliferative diabetic retinopathy (PDR), acute posterior vitreous detachment (PVD) with or without an associated retinal break, retinal vein occlusion (RVO) and ocular trauma together account for the large majority of cases.[1,2] These proportions have been remarkably consistent across Western and East Asian series, and they underpin the diagnostic algorithms reproduced in most standard texts.[3,4]

The etiological spectrum in younger individuals, however, differs substantially, and is shaped by ocular trauma, inflammatory retinal vasculitis and region-specific disease rather than by age-related vitreoretinal interface change or long-standing metabolic disease.[5–8] In the Indian subcontinent, idiopathic occlusive retinal periphlebitis formally addressed as Eales' disease remains an important and often recurrent cause of VH in otherwise healthy young men, with a strong epidemiological association with tuberculous hypersensitivity in a tuberculosis-endemic population.[9–12] Retinal vasculitis of this type characteristically presents between the second and fourth decades of life, precisely the interval in which the published VH literature is thinnest.[9,13]

Existing evidence on VH falls into two groups. Paediatric series (patients younger than 18 years) describe a spectrum in which trauma, retinopathy of prematurity, Coats disease, familial exudative vitreoretinopathy and X-linked retinoschisis dominate.[6,8,14] Adult and population-based series, by contrast, are drawn largely from populations older than 50 years and are dominated by PDR, which accounts for approximately half of all cases.[2,15] The interval between these two bodies of work — the 18-to-45-year band, which encompasses the economically most productive years of life and in which visual loss carries the greatest societal

cost — has not been systematically characterised as a continuum. Where young adults are reported at all, they are usually pooled into a single broad adult category, obscuring the age-related transition in etiology that the present data demonstrate.

The present study was therefore undertaken to analyse the demographic characteristics, etiological trends, systemic associations and visual outcomes of VH in patients aged 18 to 45 years presenting to a tertiary eye care centre, and to define whether a coherent age-related etiological gradient exists across this interval.

MATERIALS AND METHODS

Study design and ethical approval

This was a retrospective, observational, single-centre study conducted at a tertiary eye care referral centre in India. The study protocol was reviewed and approved by the Institutional Review Board and Ethics Committee of Dr. Shroff's Charity Eye Hospital, Delhi. All study procedures adhered to the tenets of the Declaration of Helsinki and to national regulations governing the use of retrospective clinical data. Because the study was retrospective in design and used only electronic medical records obtained in the course of routine clinical care, the requirement for written informed consent was formally waived by the Institutional Review Board.

This study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies,[16] and the completed checklist is provided as Additional File 1

Study population and rationale for the 18-to-45-year age range

Consecutive patients aged 18 to 45 years (inclusive) diagnosed with vitreous haemorrhage between January 2019 and December 2024 were eligible for inclusion.

The choice of 18 to 45 years, rather than a wider interval such as 18 to 50 years, was deliberate and was made for four reasons. First, the lower bound of 18 years separates this cohort cleanly from the paediatric VH literature, in which retinopathy of prematurity, non-accidental injury, Coats disease, familial exudative vitreoretinopathy and X-linked retinoschisis contribute a spectrum of disease that has no meaningful overlap with adult practice.[6,8,14] Second, 18 to 45 years corresponds to the conventional epidemiological definition of the young adult and to the economically most productive years of life, the interval in which visual disability carries the greatest individual and societal cost.[17] Third, and most importantly for the internal validity of the analysis, the upper bound was set at 45 rather than 50 years because both of the mechanisms that dominate VH in later life ; acute posterior vitreous detachment with or without a retinal break, and proliferative diabetic retinopathy increase steeply in prevalence beyond the fifth decade.[2,18,19] Extending the ceiling to 50 years would have admitted a rapidly rising proportion of PVD and diabetes-related haemorrhage and would have diluted the inflammatory and traumatic signal that this study was designed to characterise, without adding a biologically distinct group. Fourth, the 18-to-45-year window captures in full the age range in which Eales' disease, the principal inflammatory cause of VH in this geographical setting, characteristically presents (the second to fourth decades), allowing the entire natural history of that presentation to be observed within a single cohort.[9,10,12]

Inclusion and exclusion criteria

All patients within the specified age range presenting with vitreous haemorrhage of any density were considered for inclusion. The diagnosis was established by slit-lamp biomicroscopy and indirect ophthalmoscopy where the media permitted, and by B-scan ultrasonography where the fundus was obscured. Patients were excluded ,if records were incomplete with respect to presenting or final visual acuity, or if follow-up was shorter than six months.

Rationale for the minimum six-month follow-up requirement

This study had two primary objectives: characterisation of the etiological spectrum, and assessment of anatomical and visual outcome. A minimum observation period was necessary for both. Etiological attribution in an eye with media opacity is frequently provisional at presentation and is confirmed only once the haemorrhage clears sufficiently to permit fundus examination and fluorescein angiography, at the time of vitrectomy, or on completion of systemic and laboratory work-up, a process that in the case of inflammatory and tuberculosis-associated disease routinely extends over several weeks to months. Patients lost to follow-up before six months would therefore have contributed a substantial proportion of unconfirmed or undetermined diagnoses, which would have degraded rather than enriched the etiological analysis that is the principal subject of this report. Applying a uniform follow-up requirement to the whole cohort also ensured that the etiological and visual-outcome analyses were performed on an identical denominator, permitting direct cross-tabulation of etiology against outcome. During the study period, 88 patients meeting the age and diagnostic criteria were excluded on this basis.

Data collection and definitions

Electronic medical records were reviewed for demographic details (age and sex), laterality, duration of symptoms before presentation, history and mechanism of trauma, systemic comorbidities, prior ocular diagnoses and the results of laboratory investigations where available. The aetiology of VH was assigned by the treating vitreoretinal specialist on the basis of clinical examination, fundus findings once the media cleared, B-scan ultrasonography, fundus fluorescein angiography, optical coherence tomography and, where clinically indicated, systemic and laboratory evaluation including complete blood count, erythrocyte sedimentation

rate, Mantoux testing, interferon-gamma release assay, chest radiography and relevant serology.

Retinal vasculitis was defined as occlusive or non-occlusive perivascular inflammation with peripheral capillary non-perfusion and secondary retinal neovascularisation on fluorescein angiography; cases in which no systemic cause was identified after complete evaluation were classified as Eales' disease in accordance with accepted criteria.[9–11] Traumatic VH required a documented history of open- or closed-globe injury classified according to the Birmingham Eye Trauma Terminology.[20] Where a retinal break coexisted with an unequivocal primary cause for example a break within a zone of vasculitic non-perfusion, or a break resulting directly from blunt trauma the case was classified according to that primary etiology.

Best-corrected visual acuity was recorded on a Snellen chart and stratified into four categories for analysis: ≥6/9; 6/12 to 6/60; 5/60 to 1/60; and ≤counting fingers close to face (comprising counting fingers close to face, hand movements, perception of light and no perception of light). Severe visual impairment was defined as BCVA ≤5/60, in keeping with World Health Organization categories of visual impairment.

Management modalities comprised observation, laser photocoagulation, intravitreal anti-vascular endothelial growth factor injection and pars plana vitrectomy, selected on the basis of the underlying etiology and clinical course. Pars plana vitrectomy was undertaken for non-clearing haemorrhage persisting beyond 6 weeks, or earlier in the presence of retinal detachment, traction/incarceration or suspected break. Visual acuity and anatomical outcome were recorded at one and six months after intervention and at the final follow-up visit (minimum six months from presentation).

Unit of analysis

Demographic and etiological analyses were performed on a per-patient basis (n = 408), as in patients with bilateral involvement the two eyes shared the same underlying etiology. Visual-outcome analyses were likewise performed on a per-patient basis (n = 408); in patients with bilateral involvement, the eye with the worse presenting BCVA was included. Laterality is reported per patient, and the total number of affected eyes (n = 452) is given for completeness.

Statistical analysis

Data were analysed using IBM SPSS Statistics v26. Descriptive statistics were used to summarise demographic and clinical characteristics; continuous variables are presented as mean ± standard deviation or as median with interquartile range (IQR) as appropriate to their distribution, and categorical variables as frequency and percentage. Associations between age band, etiology and visual outcome were analysed using the chi-square test, or the Fisher exact test where expected cell counts were less than five. For each etiology, the full distribution of final BCVA categories was compared with that of all other patients using the Fisher exact test; for retinal vasculitis and traumatic VH, final BCVA was dichotomised at ≥6/9 versus <6/9 and compared with all other patients using the chi-square test, with odds ratios reported where significant. Differences across etiological groups in the proportion achieving a final BCVA of ≥6/9 were assessed using the chi-square test, excluding groups with a single patient. Factors associated with a final BCVA of ≥6/9 were assessed by binary logistic regression including age band, presenting BCVA and etiology (enter method); results are reported as odds ratios with 95% confidence intervals. The change in the distribution of BCVA categories between presentation and final follow-up was assessed using the Bhapkar test of marginal homogeneity for paired categorical data. A two-tailed p-value of less than 0.05 was considered statistically significant.

RESULTS

Demographic characteristics and systemic associations

A total of 408 patients comprising 452 affected eyes were analysed. The mean age was 31.6 ± 9.8 years, with the largest proportion of patients in the 31–40-year band (156 patients, 38.2%), followed by the 21–30-year band (121, 29.7%), the 41–45-year band (67, 16.4%) and the 18–20-year band (64, 15.7%). There was a marked male predominance, with 343 male and 65 female patients (84.1% and 15.9% respectively; male-to-female ratio 5.3:1). Vitreous haemorrhage was unilateral in 364 patients (89.2%) and bilateral in 44 (10.8%). The median duration of symptoms before presentation was 15 days (IQR 5–60 days; range 0–830 days), reflecting a wide spectrum from acute presentation to long-standing, often recurrent disease. Approximately three-quarters of the cohort (303 patients, 74.3%) had no identifiable systemic comorbidity; diabetes mellitus, alone or in combination with hypertension, was present in 66 patients (16.2%), isolated hypertension in 23 (5.6%), and other systemic inflammatory or haematological disorders in 16 patients (3.9%). Demographic characteristics and systemic associations are summarised in Table 1.

Etiology of vitreous haemorrhage and its relationship to age

Retinal vasculitis and trauma were jointly the commonest etiologies, each accounting for 144 patients (35.3% each), followed by proliferative diabetic retinopathy in 60 patients (14.7%). Rhegmatogenous retinal detachment accounted for 25 patients (6.1%), retinal break without detachment for 1 (0.2%), retinal vein occlusion for 24 (5.9%; branch 15, central 9), and other causes for 10 patients (2.5%): complications of prior ocular surgery (4), retinopathy of prematurity sequelae (2), anaemic retinopathy (2) and adult-onset Coats disease sequelae (2). A statistically significant association was observed between age band and etiology (chi-square test, $p < 0.0001$), with a coherent and progressive transition across the four age group bands (Table 2).

Trauma was the dominant cause in the 18–20-year band, accounting for 67.2% of cases in that group, and remained a substantial contributor through the third and fourth decades (34.7% at 21–30 years and 32.7% at 31–40 years) before falling to 11.9% at 41–45 years. Retinal vasculitis showed the inverse pattern in early adulthood, rising from 10.9% in the 18–20-year band to become the single leading etiology at 21–30 years (54.5%) and 31–40 years (38.5%), and declining to 16.4% at 41–45 years. Proliferative diabetic retinopathy increased steeply and monotonically with age, from no cases at all in the 18–20-year band and 4.1% at 21–30 years, to 12.8% at 31–40 years and 52.2% at 41–45 years, at which point it displaced both trauma and vasculitis to become the dominant cause.

Retinal breaks and rhegmatogenous retinal detachment

Rhegmatogenous retinal detachment was the source of haemorrhage in 25 patients (6.1%). An isolated retinal break without associated detachment was identified as the source in 1 patient (0.2%), giving a combined break-related etiology — retinal break with or without rhegmatogenous detachment of 26 patients (6.4%). A retinal break was additionally identified intraoperatively at the time of pars plana vitrectomy in 3 eyes in which it had not been detected preoperatively, and none of the eyes developed new retinal break or new onset detachment during the six-month follow-up period.

Visual acuity at presentation

At presentation, 44 patients (10.5%) had a BCVA of ≥6/9, 94 (23.3%) were in the 6/12 to 6/60 category, 61 (15.0%) in the 5/60 to 1/60 category and 209 (51.2%) had vision of counting fingers close to face or worse. Severe visual impairment (≤5/60) was therefore present in 270 patients (66.2%) at first presentation. The association between presenting BCVA and etiology was statistically significant (Fisher exact test, $p < 0.001$). Traumatic VH was disproportionately represented among eyes presenting with very poor vision: 88 of 144 patients with traumatic

VH (61.1%) presented with BCVA of counting fingers close to face or worse, compared with 61 of 144 patients with vasculitis-related VH (42.4%). Conversely, vasculitis-related VH was distributed considerably more evenly across the acuity categories, with 26 patients (18.1%) presenting at ≥6/9 and 38 (26.4%) in the 6/12 to 6/60 category, consistent with the more variable haemorrhage density and the frequently recurrent, lower-volume bleeding that characterises inflammatory disease. The full cross-tabulation of presenting BCVA against etiology is presented in Table 3.

Visual outcomes

At the final follow-up visit, 154 patients (37.7%) achieved a BCVA of ≥6/9, compared with 43 patients (10.5%) at presentation, representing a more than threefold increase in the proportion with good central vision. Over the same interval, the proportion with severe visual impairment (≤5/60) fell from 66.2% to 35.5%, and the proportion with vision of counting fingers close to face or worse fell from 51.2% to 19.4% (Bhapkar test, $p < 0.0001$) (Table 4).

Retinal vasculitis had the highest proportion of patients achieving a final BCVA of ≥6/9 (67 of 144; 46.5%), followed by traumatic VH (36.1%), retinal vein occlusion (33.3%) and proliferative diabetic retinopathy (30.0%), whereas rhegmatogenous retinal detachment had the lowest (20.0%). Compared with all other patients, retinal vasculitis was significantly associated with achieving a final BCVA of ≥6/9 (67 of 144 vs 87 of 264; odds ratio 1.77; χ^2 test, $p = 0.007$), accounting for 67 of the 154 patients (43.5%) with this outcome, whereas traumatic VH was not (52 of 144 vs 102 of 264; $p = 0.615$). When the full distribution of final BCVA categories was compared for each remaining etiology against all other patients (Fisher exact test), a significant association was observed for the central retinal vein occlusion subgroup ($n = 9$; $p = 0.044$), lasered PDR ($n = 8$; $p = 0.023$) and VH following complicated ocular surgery ($n = 4$; $p = 0.021$), each of which was over-represented in the poorer acuity categories;

associations for untreated PDR (p = 0.818), retinal vein occlusion overall (p = 0.232) and rhegmatogenous retinal detachment (p = 0.063) were not significant. Final BCVA was also significantly associated with age group (Fisher exact test, p < 0.0001): ≥6/9 was achieved by 13 of 64 patients (20.3%) aged ≤20 years, 56 of 121 (46.3%) aged 21–30 years, 64 of 156 (41.0%) aged 31–40 years and 21 of 67 (31.3%) aged over 40 years. When etiology was dichotomised at ≥6/9, the overall difference between groups was not significant (p = 0.069) (Table 5). Systemic diagnosis was not significantly associated with final BCVA.

To identify independent predictors of a good visual outcome, a binary logistic regression model including age band, presenting BCVA and etiology was fitted using patient-level data; all 408 patients had complete data for all covariates and were included (Table 6). On univariable analysis, patients aged 21–30 years (crude OR 3.38, 95% CI 1.67–6.85; p = 0.001) and 31–40 years (crude OR 2.73, 95% CI 1.37–5.43; p = 0.004) had higher odds of achieving a final BCVA of ≥6/9 than those aged 18–20 years, and poorer presenting BCVA was associated with lower odds of a good outcome, whereas no individual etiology differed significantly from traumatic VH. Presenting BCVA was the only independent predictor of achieving a final BCVA of ≥6/9 (overall p < 0.001). Compared with patients presenting with BCVA ≥6/60, those presenting with 5/60 to counting fingers (adjusted OR 0.41, 95% CI 0.25–0.68; p < 0.001) or hand movements or worse (adjusted OR 0.45, 95% CI 0.26–0.78; p = 0.005) had significantly lower odds of achieving a final BCVA of ≥6/9. Although final BCVA varied significantly with age group on univariable analysis (p < 0.0001), age band was not independently associated with the outcome after adjustment (overall p = 0.188), suggesting that the age gradient in visual outcome largely reflected differences in etiology and presenting BCVA between age groups. Similarly, the higher unadjusted proportion of patients with retinal vasculitis achieving ≥6/9 compared with traumatic VH (46.5% vs 36.1%; Table 5) was attenuated after adjustment (adjusted OR 1.05, 95% CI 0.62–1.77; p = 0.853), and etiology as a whole was not

independently associated with final BCVA ≥6/9 (overall p = 0.226). RRD, which had the lowest proportion achieving ≥6/9 (20.0%), remained associated with lower odds of a good outcome (adjusted OR 0.31, 95% CI 0.10–0.98; p = 0.046). The model showed good calibration (Hosmer–Lemeshow p = 0.921) and modest discrimination (AUC 0.66). Taken together, the severity of visual loss at presentation, rather than patient age or the underlying etiology, was the principal determinant of final visual outcome in this cohort.

Management

Treatment was directed by etiology and clinical course. Of the 408 patients, 94 (23.0%) were managed by observation alone, 89 (21.8%) underwent laser photocoagulation, 49 (12.0%) received intravitreal anti-vascular endothelial growth factor therapy and 109 (26.7%) underwent pars plana vitrectomy. Anti-inflammatory therapy comprised oral corticosteroids in 134 patients (32.8%), immunomodulatory therapy in 21 (5.1%) and intravitreal triamcinolone acetonide in 4 (1.0%). Modalities were not mutually exclusive: 55 patients (13.5%) received more than one of laser, intravitreal therapy or vitrectomy. Treatment records were incomplete for 108 patients (26.5%), and no treatment was recorded for 73 (17.9%); to avoid overstating treatment rates, the percentages in this paragraph use the full cohort (n = 408) as the denominator.

The pattern of management differed by etiology. In retinal vasculitis, oral corticosteroids were used in 95 patients (66.4%), laser photocoagulation in 45 (31.5%), pars plana vitrectomy in 41 (28.7%), anti-VEGF therapy in 23 (16.1%) and immunomodulatory therapy in 20 (14.0%), while only 8 (5.6%) were observed; 20 of the 21 patients who received immunomodulatory therapy belonged to this group. Antitubercular therapy (ATT; prior, ongoing or initiated at presentation) was received by 27 patients with retinal vasculitis (18.9%) for a median duration of 6 months (IQR 4.25–8.25), accounting for 27 of the 32 patients (84.4%) in the cohort who

received ATT. Proliferative diabetic retinopathy was managed chiefly with laser photocoagulation (32; 54.2%), followed by vitrectomy (18; 30.5%) and anti-VEGF therapy (16; 27.1%). Traumatic VH was most often observed (54; 38.8%) or treated by vitrectomy (36; 25.9%), and 32 patients (23.0%) received oral corticosteroids alone, with spontaneous clearing of the haemorrhage. Vitrectomy was the principal intervention in rhegmatogenous retinal detachment (10; 43.5%), whereas retinal vein occlusion was managed with laser (6; 40.0%), anti-VEGF therapy (5; 33.3%) or observation (5; 33.3%), and 8 of RVO patients (33.3%) underwent vitrectomy for non-resolving vitreous haemorrhage.

DISCUSSION

This study shows that causes of vitreous haemorrhage in young adults shifts markedly with age and differ substantially from the patterns observed in older populations.[1,2,5] Trauma and retinal vasculitis together accounted for 70.6% of all cases in this cohort, with a clear and progressive transition from trauma in the youngest patients, through inflammatory etiologies in early adulthood, and subsequently towards metabolic causes with advancing age (overall association between age band and etiology, $p < 0.0001$). To our knowledge this is among the largest single-centre series to characterise VH specifically within the 18-to-45-year interval, one of few to describe how the etiological mix changes across successive age bands within a single cohort, rather than by comparing paediatric and adult populations studied separately.

Consistent with prior paediatric and adolescent studies, trauma was the predominant cause of VH in the youngest patients, accounting for approximately two-thirds of cases in the 18–20-year band.[6,8,14] This is unsurprising in a cohort that is overwhelmingly young and male and that reflects the well-documented epidemiology of ocular injury in occupational and recreational settings in low- and middle-income countries.[20,21] Our findings, however, extend the existing literature by showing that retinal vasculitis overtakes trauma as the leading

etiology across the 21-to-40-year interval, a transition that is difficult to discern in earlier studies because most have evaluated paediatric and adult populations separately. The dominance of retinal vasculitis in early adulthood is a key finding of this study. While Rishi and colleagues identified Eales' disease as the most common cause of spontaneous VH in their series, the present study quantifies its prominence within a broader and considerably larger young adult cohort, in which it accounted for more than half of all cases in the third decade.[6] This observation is particularly relevant in the Indian context, where inflammatory retinal disorders, and Eales' disease in particular, remain highly prevalent, with a recognised predilection for healthy young men in tuberculosis-endemic regions.[7,9,10,12] Although tuberculosis-directed investigation was not analysed systematically in this series, the published literature supports incorporating region-specific systemic evaluation into routine practice: Mantoux positivity is reported in approximately four-fifths of patients with Eales' disease, with supportive changes on CECT thorax, supporting a hypersensitivity rather than an actively infective mechanism.[9,11,22] Taken together, these findings highlight the limitations of extrapolating Western data to populations in which inflammatory etiologies are substantially more common.[1,23]

Although proliferative diabetic retinopathy remains an important cause of VH, its contribution in this study was largely confined to patients older than 40 years, in whom it accounted for more than half of all cases (52.2%), compared with none of the patients aged 18–20 years and 4.1% of those aged 21–30 years. This contrasts sharply with population-based and hospital-based adult series, in which PDR predominates across essentially all adult age groups and may account for half or more of all presentations.[2,7,15] The discrepancy is readily explained by the natural history of diabetic retinopathy: advanced proliferative disease requires a long duration of hyperglycaemia and typically suboptimal glycaemic control, and the prevalence of retinal neovascularisation consequently rises steeply only after the fourth decade in populations

with predominantly type 2 disease. This reinforces the central clinical message of age-specific diagnostic prioritisation: in a patient aged 25 years presenting with spontaneous VH, a diabetes-centred work-up is unlikely to be the most productive starting point, whereas in a patient aged 43 years it should be a priority.

Rhegmatogenous retinal detachment accounted for only 6.1% of cases in this series, and the combined break-related etiology remained modest. At first sight this appears markedly lower than in much of the published literature, in which retinal breaks are reported in 50% to 70% of eyes with posterior vitreous detachment complicated by vitreous haemorrhage, and in which retinal detachment accounts for between 10% and 15% of all cases of VH.[15,18,24] This divergence most likely reflects a genuine biological and methodological difference rather than an inconsistency, and four considerations explain it.

First, and most importantly, the mechanism that generates the majority of break-related VH in those series (acute symptomatic posterior vitreous detachment with avulsion of a peripheral retinal vessel) is largely absent from a cohort with a mean age of 31.6 years. The prevalence of PVD is low before the fifth decade and rises steeply thereafter, and the 50%-to-70% figure is derived specifically from populations in which PVD is the index event.[18,19,24] In a cohort defined by an upper age limit of 45 years, the anatomical substrate for that mechanism is simply uncommon, and the figure is therefore not transferable. This is a direct and predictable consequence of the age range that defines the study rather than an anomaly within it.

Second, the series reporting high break rates are typically restricted to spontaneous, fundus-obscuring haemorrhage of otherwise unexplained origin an enriched denominator in which break-related disease is, by construction, over-represented.[15,24] The present cohort is unselected with respect to haemorrhage density and includes a large traumatic and inflammatory fraction; when trauma and retinal vasculitis together account for more than two-thirds of cases, the residual proportion available to break-related mechanisms is necessarily

small. When trauma, retinal vasculitis and PDR are set aside, retinal break-related sequalae accounted for 26 of the remaining 60 patients (43.3%), a proportion much closer to that reported in series of spontaneous haemorrhage.

Third, cases in which a retinal break coexisted with an unequivocal primary cause a break within a zone of vasculitic capillary non-perfusion, or a break resulting directly from blunt or open-globe injury were classified according to that primary etiology, as described in the methods. This convention, which is necessary to avoid double-counting in an etiological analysis, inevitably under-represents retinal breaks as a stand-alone category. Fourth, two of the strongest risk factors for break formation pseudophakia and high axial myopia with advanced vitreous syneresis are substantially less prevalent in this age group than in the older cohorts from which the comparative figures derive.[25]

A degree of under-detection nevertheless cannot be excluded, and we would not wish the low observed rate to be read as reassurance. B-scan ultrasonography, on which diagnosis necessarily depends when the fundus is obscured, has recognised limitations in the detection of small peripheral breaks, particularly when the haemorrhage is dense or when a partial posterior vitreous separation mimics or conceals a flap tear.[26,27] Some breaks are identified only at vitrectomy, and others declare themselves as the haemorrhage clears. Accordingly, the practical implication of these data is not that retinal breaks may be disregarded in young patients, but that the threshold for scleral-indented peripheral examination once the media clear, for serial ultrasonography in eyes that remain opaque, and for earlier surgical intervention where the view fails to improve, should remain low irrespective of the patient's age.

Visual outcomes and their determinants

Visual outcomes in this cohort were generally favourable, with a more than threefold increase in the proportion of patients achieving BCVA ≥6/9 and a near-halving of the proportion with

severe visual impairment.[5] Consistent in direction with prior reports, inflammatory and traumatic etiologies showed numerically better visual outcomes, whereas ischaemic causes such as PDR, retinal vein occlusion and retinal detachment showed poorer outcomes, with retinal vasculitis showing the highest proportion achieving a final BCVA of ≥6/9 (46.5% vs 33.0% for all other etiologies; crude OR 1.77, p = 0.007). However, this association did not persist after adjustment for age band and presenting BCVA (adjusted OR 1.05 vs traumatic VH, p = 0.853), which suggests that it largely reflected the younger age and better presenting acuity of these patients.[1,5,7] Vasculitis-related VH achieved a final BCVA of ≥6/9 more often than PDR-related VH (46.5% vs 30.0%). Several factors plausibly contribute: the younger age and greater retinal reserve of these patients; the absence of the diffuse capillary dropout, chronic macular ischaemia and macular oedema that limit acuity in advanced diabetic disease; and the potential reversibility of the underlying inflammatory process with corticosteroid therapy, antitubercular therapy where indicated, and sectoral laser photocoagulation of non-perfused retina.[10,12] The relatively good outcomes after traumatic VH in this series similarly reflect the fact that, where the posterior segment injury is confined to haemorrhage without retinal or optic nerve disruption, the visual potential of a young eye is substantial.

Although presenting acuity rather than etiology or age was the principal independent determinant of final visual acuity, the poorer outcomes in the small central retinal vein occlusion subgroup underscore the importance of accurate etiological classification not only for guiding management but also for prognostication and patient counselling. From a clinical standpoint, these findings support a shift away from the conventional, diabetes-centric evaluation of VH towards an age- and geography-informed diagnostic framework. In a young adult presenting with VH, early consideration of trauma and retinal vasculitis, together with a targeted systemic work-up that in this region should include evaluation for tuberculous

hypersensitivity, may shorten the time to diagnosis and allow earlier etiology-directed treatment. Equally, the finding that nearly three-quarters of this cohort had no systemic comorbidity at all emphasises that evaluation must be ophthalmologist-driven, and that the absence of systemic risk factors should not be taken as reassurance about the severity of the underlying retinal disease.

Strengths and limitations

The principal strengths of this study are its size, its six-year recruitment window and its uniform, age-stratified analysis of a population that has previously been reported only in fragments. Several limitations should nevertheless be acknowledged. The retrospective, single-centre design and the tertiary referral setting may have introduced referral bias, potentially over-representing severe, recurrent or diagnostically difficult cases; milder haemorrhages managed in the community would not be captured. The requirement for a minimum of six months of follow-up, while necessary for reliable etiological attribution and for outcome assessment, may have preferentially excluded patients with rapidly resolving haemorrhage and good early recovery, as well as those with the poorest outcomes who disengaged from care; the etiological proportions reported here should therefore be interpreted as applying to VH that comes to sustained tertiary care rather than to all VH occurring in this age group. Visual acuity was recorded on a Snellen chart rather than by ETDRS protocol, limiting the precision of acuity comparisons. Associations between etiology and visual outcome were examined in a multivariable model adjusted for age band and presenting BCVA; because this was a retrospective analysis with a limited set of covariates, residual confounding cannot be excluded, and comparisons within small subgroups such as central retinal vein occlusion should be regarded as exploratory. The length of follow-up also varied between patients, so final acuity was not measured at a fixed time point. Finally, where the fundus was obscured, detection of

peripheral breaks relied partly on B-scan ultrasonography, which may have led to under-ascertainment.

CONCLUSION

Vitreous haemorrhage in young adults should not be approached as a uniform extension of adult disease. In this Indian tertiary-care cohort, etiology shifted clearly with age, and trauma and retinal vasculitis, rather than diabetic retinopathy, accounted for 70.6% of cases. Retinal vasculitis emerges as a dominant and potentially reversible cause in early adulthood, reinforcing the need for early, targeted evaluation including region-specific systemic work-up. Visual outcomes were largely favourable, particularly after vasculitis-related and traumatic haemorrhage, whereas ischaemic causes continue to carry a guarded prognosis. These findings challenge conventional diagnostic assumptions and support the adoption of an age- and geography-informed clinical framework to improve diagnostic accuracy, guide management and optimise visual outcomes in young patients with vitreous haemorrhage.

ABBREVIATIONS

ATT: antitubercular therapy; BCVA: best-corrected visual acuity; CECT: contrast-enhanced computed tomography; ETDRS: Early Treatment Diabetic Retinopathy Study; IQR: interquartile range; OR: odds ratio; PDR: proliferative diabetic retinopathy; PVD: posterior vitreous detachment; RVO: retinal vein occlusion; VEGF: vascular endothelial growth factor; VH: vitreous haemorrhage.

DECLARATIONS

Ethics approval and consent to participate

The study was approved by the Institutional Review Board and Ethics Committee of Dr. Shroff's Charity Eye Hospital, Delhi and adhered to the tenets of the Declaration of Helsinki.

The requirement for informed consent was waived by the committee owing to the retrospective design and the use of routinely collected clinical records.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

No funding was received for this study.

Authors' contributions

PG: conception, data collection, patient management, analysis and drafting; ArB: conception, analysis and drafting; AnB: analysis and drafting; MA: analysis, drafting and patient management

All authors read and approved the final manuscript.

Acknowledgements

Dr. Shalini Singh, Dr. Isha Agarwal, Dr. Tanya Jain and Dr. Akash Chaudhary for contribution of patients to this series.

TABLES

Table 1. Demographic characteristics and systemic associations of patients with vitreous haemorrhage (n = 408)

Variable	Frequency (n)	Percentage (%)
Age band (years)		
18–20	64	15.7
21–30	121	29.7
31–40	156	38.2
41–45	67	16.4
Sex		
Male	343	84.1
Female	65	15.9
Laterality		
Unilateral	364	89.2
Bilateral	44	10.8
Systemic association		
None	303	74.3
Diabetes mellitus alone	37	9.1
Diabetes mellitus with hypertension	29	7.1
Hypertension alone	23	5.6
Other systemic disorders*	16	3.9
Duration of symptoms		
Median (IQR), days	15 (5–60)	Range 0–830

*Includes pulmonary tuberculosis, thyroid disease, rheumatoid arthritis, Takayasu arteritis, antiphospholipid antibody syndrome, anaemia, dural venous sinus thrombosis and other rare conditions. IQR = interquartile range.

Table 2. Etiological distribution of vitreous haemorrhage overall and by age band

Etiology	Overall n (%) (n = 408)	18–20 yrs (n = 64) n (%)	21–30 yrs (n = 121) n (%)	31–40 yrs (n = 156) n (%)	41–45 yrs (n = 67) n (%)	p-value
Retinal vasculitis	144 (35.3)	7 (10.9)	66 (54.5)	60 (38.5)	11 (16.4)	<0.0001†
Traumatic vitreous haemorrhage	144 (35.3)	43 (67.2)	42 (34.7)	51 (32.7)	8 (11.9)	<0.0001†
Proliferative diabetic retinopathy	60 (14.7)	0 (0)	5 (4.1)	20 (12.8)	35 (52.2)	<0.0001‡

Etiology	Overall n (%) (n = 408)	18–20 yrs (n = 64) n (%)	21–30 yrs (n = 121) n (%)	31–40 yrs (n = 156) n (%)	41–45 yrs (n = 67) n (%)	p-value
Rhegmatogenous retinal detachment	25 (6.1)	6 (9.4)	6 (5.0)	12 (7.7)	1 (1.5)	0.221‡
Retinal break without detachment	1 (0.2)	0 (0)	0 (0)	1 (0.6)	0 (0)	—
Retinal vein occlusion	24 (5.9)	3 (4.7)	0 (0)	9 (5.8)	12 (17.9)	<0.001‡
Other causes	10 (2.5)	5 (7.8)	2 (1.7)	3 (1.9)	0 (0)	—

Values are n (%). Overall percentages are calculated from the total cohort (n = 408); age-band percentages are calculated within each age band (column percentages). Retinal vein occlusion comprises branch (n = 15) and central (n = 9) retinal vein occlusion. Other causes (n = 10) comprise complications of prior ocular surgery (4), retinopathy of prematurity sequelae (2), anaemic retinopathy (2) and adult-onset Coats disease sequelae (2). †Chi-square test. ‡Fisher exact test.

Table 3. Best-corrected visual acuity at presentation according to etiology of vitreous haemorrhage

Etiology	≥6/9 n (%)	6/12–6/60 n (%)	5/60–1/60 n (%)	≤CF close to face n (%)	Total n
Retinal vasculitis	26 (18.1)	38 (26.4)	19 (13.2)	61 (42.4)	144
Traumatic vitreous haemorrhage	12 (8.3)	26 (18.1)	18 (12.5)	88 (61.1)	144
Proliferative diabetic retinopathy	2 (3.3)	21 (35.0)	16 (26.7)	21 (35.0)	60
Rhegmatogenous retinal detachment	0 (0)	3 (12.0)	4 (16.0)	18 (72.0)	25
Retinal break without detachment	1 (100)	0 (0)	0 (0)	0 (0)	1
Retinal vein occlusion	3 (12.5)	5 (20.8)	2 (8.3)	14 (58.3)	24
Other causes	0 (0)	1 (10.0)	2 (20.0)	7 (70.0)	10
Total	**44 (10.5)**	**94 (23.3)**	**61 (15.0)**	**209 (51.2)**	**408**

CF = counting fingers. Fisher exact test, $p < 0.001$. Percentages are row percentages. The ≤CF close to face category includes counting fingers close to face(15), hand movements and perception of light (188), no perception of light (6).

Table 4. Best-corrected visual acuity at presentation and at final follow-up

BCVA category	At presentation n (%)	At final follow-up n (%)
≥6/9	44 (10.5)	154 (37.7)
6/12–6/60	94 (23.3)	109 (26.7)
5/60–1/60	61 (15.0)	66 (16.2)
≤CF close to face	209 (51.2)	79 (19.4)
Total	**408 (100)**	**408 (100)**

BCVA = best-corrected visual acuity; CF = counting fingers. Severe visual impairment (≤5/60) declined from 270 patients (66.2%) at presentation to 145 (35.5%) at final follow-up.

Table 5. Association between etiology of vitreous haemorrhage and final visual outcome

Etiology	Final BCVA ≥6/9 n (%)	Final BCVA <6/9 n (%)	Total n
Retinal vasculitis	67 (46.5)	77 (53.5)	144
Traumatic vitreous haemorrhage	52 (36.1)	92 (63.9)	144
Proliferative diabetic retinopathy	18 (30.0)	42 (70.0)	60
Retinal vein occlusion	8 (33.3)	16 (66.7)	24
Rhegmatogenous retinal detachment	5 (20.0)	20 (80.0)	25
Retinal break without detachment	1 (100)	0 (0)	1
Other causes	3 (30.0)	7 (70.0)	10
Total	**154 (37.7)**	**254 (62.3)**	**408**

Values are n (row %). BCVA = best-corrected visual acuity. Final BCVA at the last follow-up visit is reported for the full cohort. Retinal vein occlusion comprises branch and central retinal vein occlusion. Chi-square test across the six etiological groups, excluding retinal break without detachment (n = 1): $\chi^2 = 10.24$, df = 5, p = 0.069.

Table 6. Multivariable logistic regression analysis of factors associated with final BCVA ≥6/9

Variable	Category	n	Final BCVA ≥6/9, n (%)	Crude OR (95% CI)	p	Adjusted OR (95% CI)	p
Age band (years)	Overall						0.188
	18–20	64	13 (20.3)	1	—	1	—
	21–30	121	56 (46.3)	3.38 (1.67–6.85)	0.001	1.87 (0.88–3.98)	0.104
	31–40	156	64 (41.0)	2.73 (1.37–5.43)	0.004	1.66 (0.80–3.42)	0.173
	41–45	67	21 (31.3)	1.79 (0.81–3.98)	0.152	0.97 (0.38–2.47)	0.949
Presenting BCVA	Overall						<0.001
	≥6/60	138	75 (54.3)	1	—	1	—
	5/60 to CF	155	48 (31.0)	0.41 (0.25–0.67)	<0.001	0.41 (0.25–0.68)	<0.001
	HM or worse	115	31 (27)	0.41 (0.24–0.70)	0.001	0.45 (0.26–0.78)	0.005
Cause of vitreous haemorrhage	Overall						0.226
	Trauma	144	52 (36.1)	1	—	1	—
	Retinal vasculitis	144	67 (46.5)	1.54 (0.96–2.47)	0.073	1.05 (0.62–1.77)	0.853
	PDR	60	18 (30.0)	0.76 (0.40–1.45)	0.403	0.55 (0.25–1.22)	0.141
	RVO	24	8 (33.3)	0.88 (0.35–2.21)	0.793	0.98 (0.36–2.63)	0.966
	RRD/break	26	6 (20.0)	0.44 (0.16–1.25)	0.123	0.31 (0.10–0.98)	0.046
	Other	10	3 (30.0)	0.76 (0.19–3.06)	0.697	0.90 (0.19–4.37)	0.896

OR = odds ratio; CI = confidence interval; CF = counting fingers; HM = hand movements; PDR = proliferative diabetic retinopathy; RVO = retinal vein occlusion; RRD = rhegmatogenous retinal detachment. Numbers, percentages and crude ORs for age band and etiology are based on the full cohort. Adjusted ORs from a single binary logistic regression model including age band, presenting BCVA and etiology. Overall p for multi-category variables by Wald χ^2 test.

REFERENCES

1. Shaikh N, Srishti R, Khanum A, Thirumalesh MB, Dave V, Arora A, et al. Vitreous haemorrhage – causes, diagnosis and management. Indian J Ophthalmol. 2023;71(1):28–38.

2. Wang CY, Hsieh YT, Chang CY, Lin CL. Incidence and risk factors for vitreous hemorrhage: a nationwide population-based study. Am J Ophthalmol. 2017;182:1–7.

3. Kanski JJ, Bowling B. Clinical Ophthalmology: A Systematic Approach. 8th ed. London: Elsevier; 2016. p. 720–5.

4. Spraul CW, Grossniklaus HE. Vitreous hemorrhage. Surv Ophthalmol. 1997;42(1):3–39.

5. Dana MR, Merayo-Lloves J, Schaumberg DA, Foster CS. Prognosticators for visual outcome in patients with spontaneous vitreous hemorrhage. Ophthalmology. 1996;103(11):1891–6.

6. Rishi P, Rishi E, Venkataraman A, Sharma T. Vitreous hemorrhage in children and adolescents in India. J AAPOS. 2013;17(1):64–9.

7. Desai N, Shah A, Patel K. Study of etiology of vitreous hemorrhage in adult patients. Delhi J Ophthalmol. 2023;33(2):45–50.

8. Naik AU, Rishi E, Rishi P. Pediatric vitreous hemorrhage: a narrative review. Indian J Ophthalmol. 2019;67(6):732–9.

9. Biswas J, Sharma T, Gopal L, Madhavan HN, Sulochana KN, Ramakrishnan S. Eales disease — an update. Surv Ophthalmol. 2002;47(3):197–214.

10. Das T, Pathengay A, Hussain N, Biswas J. Eales' disease: diagnosis and management. Eye (Lond). 2010;24(3):472–82.

11. Biswas J, Ravi RK, Naryanasamy A, Kulandai LT, Madhavan HN. Eales' disease — current concepts in diagnosis and management. J Ophthalmic Inflamm Infect. 2013;3(1):11.

12. Biswas J, Agrawal M, Chawla A, Narayansami A, Agarwal A. Eales' disease revisited: current understanding of etiopathogenesis, multimodal imaging and treatment strategies. Indian J Ophthalmol. 2026;74(6):788–91.

13. Therese KL, Deepa P, Therese J, Bagyalakshmi R, Biswas J, Madhavan HN. Association of mycobacteria with Eales' disease. Indian J Med Res. 2007;126(1):56–62.

14. Spirn MJ, Lynn MJ, Hubbard GB 3rd, et al. Vitreous hemorrhage in patients younger than 18 years. Ophthalmology. 2006;113(10):1788–93.

15. Lindgren G, Sjödell L, Lindblom B. A prospective study of dense spontaneous vitreous hemorrhage. Am J Ophthalmol. 1995;119(4):458–65.

16. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–7.

17. Négrel AD, Thylefors B. The global impact of eye injuries. Ophthalmic Epidemiol. 1998;5(3):143–69.

18. Ahmed F, Tripathy K. Posterior vitreous detachment. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023.

19. Uchino E, Uemura A, Ohba N. Initial stages of posterior vitreous detachment in healthy eyes of older persons evaluated by optical coherence tomography. Arch Ophthalmol. 2001;119(10):1475–9.

20. Kuhn F, Morris R, Witherspoon CD, Heimann K, Jeffers JB, Treister G. A standardized classification of ocular trauma. Graefes Arch Clin Exp Ophthalmol. 1996;234(6):399–403.

21. Diabetic Retinopathy Vitrectomy Study Research Group. Early vitrectomy for severe vitreous hemorrhage in diabetic retinopathy: two-year results of a randomized trial. DRVS report 2. Arch Ophthalmol. 1985;103(11):1644–52.

22. Gupta V, Gupta A, Rao NA. Intraocular tuberculosis — an update. Surv Ophthalmol. 2007;52(6):561–87.

23. American Academy of Ophthalmology. Vitreous hemorrhage: diagnosis and management. EyeNet Magazine. 2020.

24. Sarrafizadeh R, Hassan TS, Ruby AJ, et al. Incidence of retinal detachment and visual outcome in eyes presenting with posterior vitreous separation and dense fundus-obscuring vitreous hemorrhage. Ophthalmology. 2001;108(12):2273–8.

25. Mitry D, Charteris DG, Fleck BW, Campbell H, Singh J. The epidemiology of rhegmatogenous retinal detachment: geographical variation and clinical associations. Br J Ophthalmol. 2010;94(6):678–84.

26. Rabinowitz R, Yagev R, Shoham A, Lifshitz T. Comparison between clinical and ultrasound findings in patients with vitreous hemorrhage. Eye (Lond). 2004;18(3):253–6.

27. Melamud A, Pham H, Stoumbos Z. Early vitrectomy for spontaneous, fundus-obscuring vitreous hemorrhage. Am J Ophthalmol. 2015;160(5):1073–7.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- STROBEStatement.docx