# **Original** Article



# Clinical profile and management of vitreous hemorrhage in tertiary eye care centre in Nepal

Laxmi Devi Manandhar<sup>1</sup>, Raba Thapa<sup>2</sup>, Govinda Poudyal<sup>2</sup> <sup>1</sup>Lumbini Eye Institute and Research Centre, Siddhaarthanagar-3, Bhairahawa, Nepal <sup>2</sup>Tilganga Institute of Ophthalmology, Kathmandu, Nepal

### Abstract

**Introduction:** Vitreous hemorrhage is one of the most common diseases presenting to emergency department and leading cause of painless vision loss.

**Objectives:** To determine the profile of vitreous hemorrhage in patients presented to Outpatient Department (OPD) and emergency Department of Tilganga Institute of Ophthalmology (TIO).

**Materials and methods:** This is a hospital based observational non interventional descriptive study. Total 198 patients were enrolled who visited OPD and Emergency department of TIO from August 1<sup>st</sup> 2012 to July 30<sup>th</sup> 2013.

**Result:** Total 198 patients (201 eyes) were enrolled for the study, out of which 144 were male and 54 females. 195 were unilateral and 3 bilateral cases. Most common age group of presentation of vitreous hemorrhage was 51-60 years (24.75%). Most common presenting complaint was sudden onset of decreased vision (95%). Most common etiology of vitreous hemorrhage was branch retinal vein occlusion (22.38%). Among the total subjects, 57.7% of the patient were managed with medical therapy, 35.8% surgically and 6.47 % with combined medical and surgical treatment.

**Conclusion**: Branch retinal vein occlusion (BRVO) is the most common cause of vitreous hemorrhage. Diabetes and hypertension are the most commonly associated systemic illnesses.

Key words: Vitreous hemorrhage, Branch retinal vein occlusion, Sudden loss of vision.

### Introduction

Vitreous hemorrhage is defined as the presence of extravasated blood within the space outlined by the internal limiting membrane of the retina

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Corresponding author Dr. Laxmi Devi Manandhar Department of Orbit and Oculopla Lumbini Eye institute and Resear Bhairahawa, Nepal Contact: 9841900142 E-mail: drlaxmi890@gmail.com	,

posteriorly and laterally, the non-pigmented epithelium of the ciliary body antero laterally, and the lens zonules and the posterior lens capsule anteriorly. (Sharma et al, 2010; Kawashima et al, 2011) Vitreous hemorrhage is one of the most common diseases presenting to emergency with sudden loss of vision. The incidence of spontaneous vitreous hemorrhage worldwide is found to be 7 cases per 1, 00,000 population. (Rabinowitz et al, 2004)

Various literatures have shown the leading cause of vitreous hemorrhage is proliferative



diabetic retinopathy followed by branch or central retinal vein occlusion (CRVO), posterior vitreous detachment with or without a retinal tear or detachment, and ocular trauma. Though uncommon in childhood, most pediatric cases result from blunt or penetrating trauma, including non-accidental injuries. (Rishi et al, 2013) Diagnostic clues have to be sought in the general medical and ocular history as well as a complete ophthalmologic examination. (Sudhalkar et al, 2013)

Vitreous hemorrhage can be managed through pan retinal photocoagulation (PRP), cryopexy, and vitrectomy depending upon the cause, clarity of media and the latest, with intravitreal anti vascular endothelial growth factor (VEGF) such as intravitreal bevacizumab. (Lauer AK et al, 2002) The amount of vitreous hemorrhage, patient's visual and systemic status determines the mode of treatment approach. (Sudhalkar et al, 2013) Very few studies have been conducted regarding vitreous hemorrhage in context of Nepal. Vitreous hemorrhage leads to significant vision loss in many patients, making their daily life very difficult. Thus, understanding the gravity of the disease is very important which is not possible from the previous published literatures in Nepal.

This study is expected to provide additional information about the demographic pattern and risk factors regarding vitreous hemorrhage that could be helpful for the clinician in discerning the etiology behind vitreous hemorrhage and possible preventive measures in future.

### **Materials and Methods**

A prospective, observational, descriptive study was conducted at Tilganga Institute of Ophthalmology (TIO), Kathmandu, Nepal. All cases of vitreous hemorrhage presented at emergency department and out- patient department (OPD) of TIO from 1<sup>st</sup> August 2012 to 30<sup>th</sup> July 2013 were enrolled in the study. Patient with previous vitreous hemorrhage receiving treatment, those not willing to participate and those who lost follow up before termination of diagnostic evaluation were excluded from the study. Proforma prepared and completed the data collection which included patient's demographic information, history, detailed ocular examination and laboratory test. The detailed history regarding presenting complaints included flashes, floaters, ocular pain and systemic history regarding diabetes, hypertension, dyslipidemia, heart disease and any bleeding disorders or any history of trauma or any drug intake was taken. Visual acuity was taken using Snellen's chart. Retinoscopy and refraction were done wherever possible. Detail ocular examination was done after dilatation of pupil with 1% tropicamide drops.

Intraocular pressure was measured by Goldman applanation tonometer and in cases of children intraocular pressure was measured by air puff when it was deemed necessary. Hematological investigations including complete blood count, lipid profile and blood sugar were done. Ocular ultrasonography was done if media was hazy to assess status of vitreous and retina. Examination of fellow eye was conducted in similar manner. CT scan was carried out as indicated in trauma cases if clinical suspicion of intraocular foreign body or ocular ultrasonography (USG) (direct technique) suggestive of intraocular foreign body. According to various literatures, Mancia et al (2003), Chobanian et al (2007) criteria to define hypertension was systolic blood pressure >140 mm Hg and diastolic blood pressure of > 90 mm Hg.

Written informed consent was taken from all the patients and the study was approved by institutional review board of National Academy of Medical Sciences (NAMS) and adheres with the tenets of declaration of Helsinki.

# Statistical analysis

The statistical analysis was done using SPSS (Statistical Package for the Social Sciences)

Version 16.0 software.

## Results

Two hundred and one eyes of 198 patients were enrolled in the study. Out of which, 144 were males and 54 females. The mean age group of overall patients presented with vitreous hemorrhage was 48.90years $\pm$  SD 18.40 years (range 2-84 years). Unilateral presentation is more than bilateral (98.4% vs 1.52%) (Table 1).

Most of the patients (53.73%) presented with VA <1/60- PL(perception of light). 19 eyes (9.45%) presented with good vision 6/6-6/18. Only 1 patient (0.5%) presented with no perception of light (NPL). (Table 2) (Table 3). Mean Duration of diabetes was 6.9 years  $\pm 4.92$  (range 0.19 years -20 years). Mean duration of Hypertension was 4.27 years  $\pm 4.34$  (range 0.19 years -20 years).

### Table 1: Demographic distribution

Demographics	Number N(%)
Age(yrs) <10	6(3.03%)
11-20	13(6.57)
21-30	24(12.12)
31-40	25(12.63)
41-50	30(15.25)
51-60	49(24.75)
61-70	36(18.18)
>70	15(7.58)
Sex Male	144(72.73)
Female	54(27.27)
Laterality Unilateral	195(98.48)
Bilateral	3(1.52)
Affected eye Right	97(48.99)
Left	98(49.99)
Both	3(1.52)



Presenting VA	No. of eyes N (%)
6/6-6/18	19(9.45)
<6/18-6/60	28(13.93)
<6/60-3/60	12(5.97)
<3/60-1/60	31(15.42)
<1/60-PL	108(53.73)
NPL(No perception of light)	1(0.5)
Difficult to assess	2(1.00)
Total	201(100)

Table 3: Systemic association of vitreoushemorrhage

Systemic diseases	No. of patients (%)
Hypertension	76(38.8)
Diabetes mellitus -Type 1	2 (1.02)
-Type 2	41(20.8)
Heart diseases	3(1.69)
Hyperlipidemia	2(1.09)
Bleeding disorders	1(0.57)

Diagnosis	No. of patients N (%)
Branched retinal vein	45(22.38)
occlusion	
Proliferative Diabetic	40(19.9)
retinopathy (PDR)	
Retinal vasculitis	27(13.43)
Rhegmatogeneous retinal	15(7.46)
detachment (RRD)	15(7.46)
Closed globe injury	33(16.41)
Central retinal vein	10(4.97)
occlusion (CRVO)	
Age related macular	7(3.38)
degeneration (AMD)	7(3.38)
Hemorrhagic Post	
vitreous detachment	6(3.0)
(PVD)	



Open globe injury	3(1.49)
Posterior Uveitis	3(1.49)
Valsalva retinopathy	2(1.0)
Cytomegalovirus(CMV) retinopathy	1(0.50)
Acute Myeloid Leukemia (AML)	2(1.0)
Bleeding disorder	1(0.50
Acute retinal necrosis	1(0.5)
Not known	5(2.5)
Total	201(100)

**Table 5: Modalities of Medical management** 

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Medical treatment	No. of eyes N (%)
Laser photocoagulation only	58 ( 49.58)
Observation only	48 (41.02)
Laser+ intravitreal Anti VEGF	7(5.98)
Intravitreal Anti VEGF Only	4(3.42)
Total	117(100)

Surgical treatment	No. of eyes N (%)
Pars Plana Vitrectomy	
(PPV) + laser	35(49.3)
photocoagulation	
PPV	12(16.9)
PPV + Endolaser (EL) +	10(14.0)
silicon oil	
PPV+ Laser + (Band	7(9.9)
Buckle) BB+ silicon oil	
PPV+ EL +gas(C3F8)	7(9.9)
Total	71(100)

Out of 198 patients, it was possible to perform all hematological investigations in 196 patients (98%). In 14 cases (7%) of patient's lipid profile, fasting and post prandial blood sugar could not be carried out and in 2 cases none of the lab test was done. All the investigations were normal in 121 cases (61%). In the current study, the mean systolic blood pressure was  $130.63\pm18.18$  mm Hg (range 90-180 mm Hg) and mean diastolic blood pressure was  $84.25\pm10.84$  mm Hg (range 60-120).

22.75 % of patients had systolic blood pressure more than 140 mm Hg and patients with diastolic blood pressure more than 90 mm Hg reached 51.85%.

Ocular USG was performed in only 49.75 % of cases in eyes where fundus view was not visible clinically. In 43.78% ultrasonography correctly found findings suggestive of vitreous hemorrhage while in 12% of cases showed findings suggestive of vitreous hemorrhage with retinal detachment.

In this study, most common etiology of vitreous hemorrhage was BRVO (22.38%). (Table 4)

Surgical management was required in 35.82% of eyes, 57.71% were managed medically and only 6.47 were managed combinedly with medical and surgical treatment. (Table 5) (Table 6)

# Discussion

Vitreous hemorrhage is a frequent complication of many vitreo-retinal pathologies. We selected to study newly diagnosed cases of vitreous hemorrhage presenting to OPD and emergency of TIO so as to provide more representative sampling of causative factors and treatment patterns of vitreous hemorrhage.

In our study males (72.73%) outnumbered females which corresponds to the study by Lean JS et al (1980) and Rishi et al (2013) (201 males out of 264 patients). It is probably because of a greater prevalence of trauma and Eales disease among males. In our study too, greater prevalence of vitreous hemorrhage in males similar to the study by Sharma et al (2010) is due to the fact that the males are more outgoing and susceptible to trauma and accidents.

Most patients in our study presented with sudden (69%) or gradual progressive loss of vision (28.9%), floaters (78.17%) and photopsia (10.71%). Interestingly, one patient presented with nonspecific symptoms like watering and discharge in this study. The mean duration of presentation of symptoms was 73.5 days (1 day - 3 years). Tertiary centre for referral may be one of the reasons for longer duration of symptoms at presentation. Results are similar to a study done by Sharma et al (2010) which showed that the sudden decreased vision (38.2%) or slowly progressive loss of vision (23.2%), floaters (22.8%) and photopsia (4%) in their patient presenting with vitreous hemorrhage.

Majority of patients presented with VA between <1/60 and PL in this study. This may be because our centre being a tertiary eye center, patients referred from other local clinics usually have severe trauma and may have poor vision at presentation as well as they are referred after prolonged duration of illness.

There were more number of hypertensive (38.8%) than diabetics (21.8%) in this study. Unlikely, Sharma et al (2010) reported diabetes (20%), hypertension (16%) and both 4 %, 21% had alcohol consumption and 27% were smokers in that study.

In this study, 3 patients were on anticoagulants (2 under aspirin and 1 warfarin for heart disease). But the co-relation between vitreous hemorrhage and anticoagulant intake could not be explained. The study conducted by Banerjee et al (2003) suggested that a drug like aspirin, ACE inhibitors and statins does not increase the risk of preretinal and vitreous hemorrhage in diabetic patients and that statins may indeed have a beneficial effect.

There were 3 out of 198 patients had chronic kidney disease (CKD stage 5) and were under dialysis since 3-6 months. In the current study 1 of all patient was known case of AML already received 3 cycles of chemotherapy.

In this study, 43.78% had findings suggestive of vitreous hemorrhage in B-scan. It could not detect retinal tear or detachment in 7 cases however, were confirmed clinically during surgery .This may be because of variable experience of ultrasound operator. Dibernardo et al (1992) studied 42 patients, out of which 11 had echo graphic diagnosis of probable retinal tear and no retinal detachment.

Branch retinal vein occlusion (22.38%) was the most common cause of vitreous hemorrhage found in this study which corresponds to the study by Moradian et al (2007) which reported the retinal vein occlusions (BRVO 56% and CRVO 16%). On contrary to this study, Sharma S et al (2010), Dana MR et al (1993) reported proliferative diabetic retinopathy remains the most common cause for vitreous hemorrhage. Disseminated intravascular coagulopathy or Tersons syndrome can cause vitreous hemorrhage in infants but no such causes is present in this study. Unlike this study, Rishi et al (2013) showed trauma being the leading cause of injury (68.5%).

Rhegmatogenous retinal detachment accounts of only 7.96% in this study which is comparable to study conducted by Sharma et al (2010) which showed the incidence to be 6.6%. But this finding is not consistent with study by Lean JS et al (1980), and Winslow R et al (1980) which showed lower retinal tears rate. We did not find vitreous hemorrhage resulting from Norrie disease or Coats disease, or retinopathy of prematurity, congenital retinoschisis, familial exudative-vitreo-retinopathy (FEVR), toxocara chorioretinitis, all of which are the causes reported by Rishi et al (2013).

In this study only 2 patients had Type 1 diabetes mellitus who presented two years and one week of diabetes and both of them were managed surgically (vitrectomy combined with laser photocoagulation). Chaudhry et al (1995) studied the surgical results of 12 consecutive





eyes of type I diabetes with severe vitreous hemorrhage, despite having had extensive pan retinal photocoagulation and found that early vitrectomy with intraoperative endolaser photocoagulation was anatomically successful in 91.66% (11/12) of the eyes that underwent surgery.

Going by data in the series as a whole, in patient with PDR out of 40 eyes 23 cases (57.7%) were managed medically in which laser photocoagulation was performed in 14 eyes, 13 cases (32.5%) were managed surgically in 8 eyes received Pars Plana vitrectomy (PPV) combined with laser photocoagulation and 4 cases (10%) were managed combined with medical and surgical therapy.

Our study found that majority of patients with vasculitis were managed medically with Laser Photocoagulation (56%) and oral steroids in some cases, and 22 % of cases were managed surgically 8 eyes (22%) out of which 2 eyes had vitrectomy with laser photocoagulation. One patient was managed with vitrectomy combined with intravitreal bevacizumab, Laser and silicon oil injection due to association with extensive tractional retinal detachment.

Yeung et al (2006) while studying vitreous hemorrhage associated with closed globe injury reported that twenty-six eyes (79%) required at least one PPV surgery during their course of treatment, and 3 of them had scleral buckle at the same time. 63.38% of all trauma cases had BCVA between 1/60 and HM at presentation.

In this study out of 6 eyes of hemorrhagic PVD, 5 were kept under close observation while one associated with superior tear so had to undergo PPV with endolaser with perfluoro propane (C3F8) gas. We came across two eyes with AML causing bilateral vitreous hemorrhage and was managed with vitrectomy for one eye and other eye was observed.

There were one each case of CMV retinopathy and acute retinal necrosis causing vitreous

hemorrhage patient with CMV retinopathy was known case of HIV under HAART regimen since 10 yrs and both of them were managed with intravitreal ganciclovir and oral steroid medication.

# Limitation of the study

The acquisition of visual acuity was not standardized like log Mar system or ETDRS charts to ensure accurate results and follow up was very short. We could have discussed the clinical and visual outcome of vitreous hemorrhage so that different treatment options can be compared.

# Conclusion

Branch retinal vein occlusion is the most common cause of vitreous hemorrhage and unilateral involvement is more prevalent than bilateral involvement. Hypertension and diabetes are the most common associated systemic illnesses. Majority of patients were managed medically (57.71%). Surgical management was required in 35.82% of eyes and only 6.47% required management via medical and surgical treatment both.

# References

Banerjee S, Denniston AKO, Gibson JM, Dodson PM (2004). Does cardiovascular therapy affect the onset and recurrence of preretinal and vitreous haemorrhage in diabetic eye disease?. Eye; 18: 821–5.

Chaudhry NA, Lim ES, Saito Y, Mieler WF, Liggett PE (1995). Early vitrectomy and endolaser photocoagulation in patients with Type I diabetes with severe vitreous hemorrhage. Ophthalmology; 102:1164-9.

Chobanian AV, Bakris GL, Black HR, et al (2003). Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood Pressure. Hypertension. 42(6):1206–52. Dana MR, Werner MS, Viana MA, Shapiro MJ (1993). Spontaneous and traumatic vitreous hemorrhage. Ophthalmology; 100(9):1377-83.

Dibernardo C, Blodi B, Byrne SF (1992). Echographic evaluation of retinal tears in patient with spontaneous vitreous hemorrhage. Arch Ophthalmol.;110(4):511-4.

Kawashima D, Ohno T, Kinoshita O, Motomura N, Kiyosue A, Fujita H (2011). Prevalence of vitreous hemorrhage following coronary revascularization in patients with diabetic retinopathy. Circ J; 75(2):329-35.

Lauer AK, Smith JR, Robertson JE, Rosenbaum JT (2002).Vitreous hemorrhage is a common complication of pediatric pars planitis. Ophthalmology; 109:95–8.

Lean JS, Gregor Z (1980).The acute vitreous hemorrhage. Br J Ophthalmol; 64:469-71.

Mancia G, De Backer G, Dominiczak A, et al (2007). Guidelines for the management of arterial hypertension: The task force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). J Hypertens;25(6):1105–87.

Moradia S, Valaee M, Ahmadieh H, Soheilian M, Azarmina M, Dehghan MH (2007). Outcomes of vitrectomy for nontraumatic non-diabetic vitreous hemorrhage. Iran J Ophthalmic Res; 2(1):46-51.

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

Rishi P, Chhablani J, Rishi E, Gupta A, Swaminathan M (2013). Vitreous hemorrhage in children and adolescents in India. J AAPOS; 17:64-9.

Sharma R, Joshi SN, Shrestha JK (2010). Etiology of vitreous hemorrhage in a tertiary eye care center in Nepal. Nepal J Ophthalmol; 2(4):121-6.

Sudhalkar A, Chhablani J, Jalali S, Mathai A, Pathengay A (2013). Spontaneous vitreous hemorrhage in children. Am J Ophthalmol; 156:1267–71.

Winslow RL, Taylor BC (1980). Spontaneous vitreous hemorrhage: etiology and management. South Med J; 73:1450-2.

Yeung L, Chen TL, Kuo YH, Chao AN, Wu WC, Chen KJ, et al (2006). Severe vitreous hemorrhage associated with closed-globe injury. Graefe's Arch Clin Exp Ophthalmol; 244:52-7.

