

Mazher Ishaq and Muhammad Khizar Niazi

Department of Ophthalmology, Military Hospital, Rawalpindi

Background: Eales disease is an idiopathic obliterative vasculopathy that commonly affects the peripheral retina of healthy young males characterized by recurrent vitreous haemorrhage. We did this study to evaluate the usefulness of prophylactic scatter photocoagulation in asymptomatic eyes of patients presenting with vitreous haemorrhage due to Eales Disease. **Methods:** Ninety nine patients with Eales' Disease demonstrable on the basis of 3 mirror fundus exam and Florescein Fundus Angiography with vitreous haemorrhage underwent either Pan retinal photocoagulation or vitrectomy in a span of 3 years. Their fellow asymptomatic eyes were grouped on the basis of their visual acuity, fundus findings and FFA picture. Group A comprising of forty-three cases underwent PRP whereas forty-three cases of Group B were not given any treatment. These cases were followed for at least three years (range 38–42 months). **Results:** Out of the forty-three cases of group A, thirty-nine (83.82%) showed visual improvement as compared to only nine cases in Group-B ($p < 0.001$). Vitreous involvement was present in only 16 cases in Group-A as compared to 33 cases in Group-B. Twenty-eight cases of Group-B showed signs of persistence of disease process in the retina compared to 13 cases in Group-A ($p < 0.005$). **Conclusion:** Prophylactic photocoagulation is an effective method of controlling the secondary complications in asymptomatic eyes of Eales disease especially if managed at an early stage. Regular checkup of peripheral retina by triple mirror examination should be performed in all asymptomatic fellow eyes of Eales disease to detect the disease process at an early stage and prevent further complications.

KEYWORDS: Eales disease, Photocoagulation, Retinal neovascularization, Vitreous haemorrhage.

INTRODUCTION

Eales disease is an idiopathic obliterative vasculopathy that commonly affects the peripheral retina of healthy young males characterized by recurrent vitreous haemorrhage¹. Although distributed world wide, it is more common in Southeast Asia². The peak age of onset is between 20–30 years³.

The aetiology is not adequately established⁴, and role of tuberculosis⁵, hypersensitivity to tuberculo-protein⁶, presence of mycobacterium tuberculosis genome in the vitrectomy specimen⁷ and epiretinal membrane⁸, retinal autoimmunity⁹, and presence of class I and II of Human Leucocyte Antigen¹⁰ have all been proposed to be a contributory factor.

Patients present with blurred vision and floaters in one eye due to presence of vitreous haemorrhage and on examination, the other eye is also involved in 50–90% cases¹¹. Initially the patients present with retinal vasculitis and later as retinal ischaemia that may lead to vascular alterations and neovascularization¹². These lesions are particularly seen in periphery of retina and if severe, can involve the posterior pole as well¹³.

Recurrent vitreous haemorrhages occur from these new vessels and they may either resolve spontaneously or lead to multiple vitreo-retinal adhesions and tractional retinal detachment and permanent visual loss¹⁴. The extent and nature of progression in Eales disease is best demonstrated by fundus fluorescein angiography^{12, 15}. In active stages, there is leakage of dye from inflamed vessels, while in later cases there is evidence of ischemia, venous shunts and collaterals.

The management aim is to reduce amount of retinal vasculitis, reduce the chances of vitreous haemorrhage by retinal ablation and surgical removal of vitreous haemorrhage¹⁶. Corticosteroids¹⁷ and photocoagulation^{18, 19} remain the mainstay of therapy in the active stage of Eales disease, whereas vitrectomy alone or combined with other vitreo-retinal surgical procedure is often required in advanced stages²⁰.

We conducted this study to find out the results of prophylactic photocoagulation in fellow asymptomatic eyes of Eales disease presenting with unilateral vitreous haemorrhage.

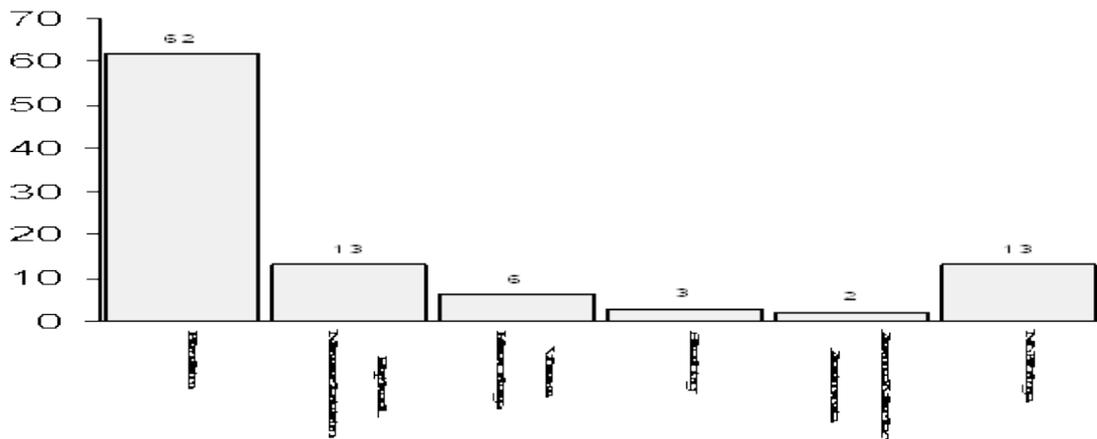
MATERIALS AND METHODS

The study was performed in the Department of Ophthalmology, Military Hospital Rawalpindi during a span of 3 years (1998–2001). All patients reporting with complaints and findings suggestive of Eales disease were taken up in the study including referrals from peripheral hospitals. Patients were excluded if they had a history of Diabetes, Hypertension, Sarcoidosis, Collagen vascular disorder, high myopia, ocular trauma or surgery, or on examination a corneal or lenticular pathology, and advanced disease process in both eyes, e.g., vitreous traction bands, retinal detachment, or macular oedema.

The main symptom was sudden painless deterioration of vision in one eye (due to complication of disease process, vitreous haemorrhage being the most common cause (80% cases), followed by branch retinal vein occlusion (5% cases), tractional retinal detachment (2%) and retinal haemorrhage, (2%). Cases presenting again due to recurrence were also included in the study. Each individual underwent complete ophthalmic examination (including 3-mirror fundus examination) that revealed neovascularization, peripheral sheathing, microaneurysms and areas of leakage or ischaemia (revealed on FFA). Blood ESR, Blood Glucose Fasting, Montoux test, Serum ANA, ELISA for Toxoplasmosis, and C-Reactive protein was done in all the cases to rule out mimicking diseases and to bring the subjects into inclusion criteria. These patients were admitted and given a four weeks course of oral prednisolone with the dose of 1 mg/kg body weight.

After the course of steroid was gradually tapered, the symptomatic eye underwent either Pan-retinal Photocoagulation (in cases where haemorrhage had resolved) or vitrectomy (in cases of organized vitreous haemorrhage or proliferative vitreoretinopathy). The fellow asymptomatic eyes were then included in the study on the basis of the study criteria and the findings in these eyes were recorded as given in Figure-1.

Figure-1: Findings in Asymptomatic Eyes (n=99)



The 86 eyes showing any positive sign of disease process were then grouped into two categories on the basis of randomized sampling.

The patients falling into Group A underwent Argon laser photocoagulation in 2–3 sessions with a gap of 3-4 weeks in between. The details of laser therapy are given as under:

- Total Number of Burns 1500–2500
- Intensity of Burns 350–500 mW
- Size of Burn 500 μ
- Exposure Time 0.1 second

In the first session, laser was applied around involved ischemic retina with neovascularization in an attempt to limit the spread of neovascularization with medium-intensity overlapping burns as shown in Figure-2. In cases of elevated neovascularization, photocoagulation of the feeder vessels was applied beneath the vascular frond. Similar technique was used for aneurysms and shunts. After first session the further laser was applied in a scatter pattern in whole of the affected quadrant as well as in the uninvolved retina as a fill-in procedure.

Figure-2: Technique of Photocoagulation



Group B patients were not given any treatment at the beginning apart from the steroid course. Follow-up was done on six monthly basis for three years. Their follow up was easy because of the condition of fellow complicated eye. On each follow-up, the visual acuity, vitreous involvement, and fundus examination findings were recorded. If any case showed deterioration in its clinical picture, additional shots of laser were applied. The significance of difference between the two groups was tested at the end of study with chi-square test.

RESULTS

The mean age of the patients was 28 ± 12 years. The results of investigations performed in all the case are given in Table-1. A comparison of the two groups done at the end of study is summarized in the Table 2.

Table-1: Mean Results of Investigations (n=86)

S. No	Tests (S.I .units)	Mean Results
1.	Blood ESR (mm fall at 1hr)	12 ± 10 mm fall
2.	Blood glucose–fasting (mMol/l)	4.8 ± 3.0 mMol/l
3.	Montoux test	Positive in 73% Negative in 27%
4.	Serum ANA factor	Positive in 36% Negative in 64%
5.	ELISA for Toxoplasmosis	Positive in 10% Negative in 90%
6.	C-reactive protein	Positive in 22% Negative in 78%

Table-2: Comparative Results at the End of Study (n=86)

Groups	VA		Persistent Vitreous Haemorrhage		Fundus changes	
	Improvement*	No improvement	Present	Absent	Regressive	Progressive
A: Laser (43)	39	04	16	27	30	13

B: No Laser (43)	09	34	33	10	15	28
------------------	----	----	----	----	----	----

The results showed marked improvement in the visual acuity in Group-A as compared to Group-B ($p<0.005$), persistent vitreous haemorrhage and vitreous cells were less marked in Group-A ($p<0.005$), and fundus picture had also improved in this group ($p<0.005$). The complications of the procedure were few. Fifteen cases developed Post laser pain that were managed by topical NSAIDs, vitreous re-bleed occurred in six cases that gradually cleared itself over passage of time. Choroidal effusion was seen in two cases, damage to lens, iris or cornea occurred in eight cases, and Choroidal Neovascular membrane formation occurred in two cases. Frequency of complications is summarized in Table-3.

Table-3: Frequency of Complications in Group-A (n=43)

S. No.	Complications	No. of cases	%
1.	Post laser pain	15	34.88
2.	Preretinal haemorrhage	07	16.27
3.	Vitreous re-bleed	06	13.90
4.	Choroidal Neovascular Membrane	03	6.97

DISCUSSION

Vitreous haemorrhage is the most common cause of visual loss in patients of Eales Disease ^{1, 21}. Various methods have been adopted to treat the secondary complications of this disorder. So far no preventive measure or disease source has been found to curb the primary lesions ²¹. The different methods used to control the secondary stage of disease include systemic steroids ¹³, photocoagulation of involved ischaemic areas, cryotherapy with laser therapy ¹⁴ and vitrectomy, especially in unresponsive cases. Favourable results have been demonstrated with light intensity, full scatter Argon laser photocoagulation to the non-perfused retina ^{18, 19}.

In longstanding non-resolving vitreous haemorrhage, vitrectomy is the only option available and sometimes even in spite of prior photocoagulation, haemorrhage occurs in these patients ¹. With repeated episodes of bleeding, the chances of spontaneous absorption of blood decreases and complications of

organized long-standing blood in vitreous cavity increase. In these cases, the benefits of Pars Plana vitrectomy are well documented ²⁰.

In our study, we tried to emphasize on the point that asymptomatic eyes of Eales disease also need monitoring, as this point was not documented before and previously laser treatment was limited to the symptomatic eyes only.

After an initial detailed evaluation of the fundus and involvement of the vitreous in both eyes, the findings showed that disease was bilateral in 77 cases (86.87%).

Results of the study were analyzed after three years of follow-up. The results regarding visual outcome of Group A were very encouraging. It showed that improvement of visual acuity of at least two Snellen's lines occurred in 39 out of 43 cases as compared to only 9 cases in Group-B. Vitreous involvement persisted in only 16 cases in Group-A as compared to 33 cases in Group-B. Twenty-eight cases of Group-B showed signs of persistence of neovascularization or sheathing in the retina compared to only 13 cases in Group-A. The comparison between our results with similar studies abroad is shown in table-4:

Table-4: Comparison of Results

S. No.	Studies	No of Eyes Treated	No. of Eyes With Positive Results
1.	Meyer-Schwickerath ²²	143	124
2.	Pahwa and Garg ²³	124	80
3.	Spitznas ²⁴	224	205
4.	Own	43	39

The complications of the procedure were minimal and did not adversely affect the out come to result in any case. Vitreous re-bleed occurred in six out of 43 cases that resolved slowly over a passage of four months.

CONCLUSION

Early intervention of cases of Eales Disease by laser therapy to the involved retina gives good control of disease process. We recommend mandatory triple mirror examination in both of the eyes of Eales Disease patients to detect and manage early changes of disease process and limit its complications.

REFERENCES

1. Biswas J, Sharma T, Gopal L, Madhavan HN, Sulochana KN, Ramakrishnan S. Eales disease-An Update. *Surv Ophthalmol* 2002; 47 (3): 197-214.
2. Das T, Biswas J, Kumar A. Eales disease. *Indian J Ophthalmol* 1994; 42: 3-18.
3. Gadkari SS, Kamdar PA, Jehangir RP. Pars plana vitrectomy in vitreous haemorrhage due to Eales disease. *Indian J Ophthalmol* 1992; 10: 35-37.

4. Patnaik B, Nagpul PN, Namperumalsamy P, Kalsi R. Eales disease-clinical features, pathophysiology, etiopathogenesis. *Ophthalmol Clin North Am* 1998; 11: 601-617.
5. Abraham C, Baig SM, Badrinath SS. Eales disease. *Proc All India Ophthalmol Soc* 1977; 33: 223-229.
6. Donders PC. Eales disease. *Doc Ophthalmol* 1958; 12: 1-105.
7. Biswas J, Therese L, Madhavan HN. Use of PCR in detection of mycobacterium tuberculosis complex DNA from vitreous sample of Eales Disease. *Br J Ophthalmol* 1999; 83: 994-996
8. Kimura SJ, Carriker FR, Hogen MJ. Retinal vasculitis with intraocular haemorrhage. Classification and results of special studies. *Arch Ophthalmol* 1956; 56: 361-365.
9. Saxena S, Rajasingh J, Biswas S. Cellular immune response to retinal S-antigen and interphotoreceptor retinoid-binding protein fragments in Eales disease patients. *Pathobiology* 1999; 67: 39-44.
10. Biswas J, Narain S, Roy S, Madhavan HN. HLA Association in Eales disease. *Invest Ophthalmol Vis Sci* 1996; 36: S363.
11. Atmaca LS, Idli A, Gunduz K. Visualisation of retinal vasculitis in Eales disease. *Ocul Immunol Inflamm* 1993;1: 49-54.
12. Renie WA, Murphy RP, Anderson KC. The evaluation of patients with Eales disease. *Retina* 1983; 3: 243-248.
13. Nagpal PN, Sharma RK, Joshi BS, Patel AM. Management of Eales disease-analysis of 800 cases (1,214 eyes). *Asia Pac J Ophthalmol* 1998; 10: 11-17.
14. Badrinath SS, Gopal L, Sharma T. Vitreoschisis in Eales disease: pathogenic role and significance in surgery. *Retina* 1999; 19: 51-54.
15. Theodossiadis G Fluorescein angiography in Eales disease *Am J Ophthalmol* 1970; 69: 271-277.
16. Jalali S, Das T. Eales disease. In: Dutta LC, ed. *Modern Ophthalmology*. New Delhi: Jaypee Brothers Medical Publishers, 2000; 2: 704-705.
17. Howe LJ, Stanford MR, Edelsten C, Graham EM. The efficacy of systemic corticosteroids in sight-threatening retinal vasculitis. *Eye* 1994; 8: 443-447.
18. Magargal LE, Walsh AW, Magargal HO, Robb-Doyle E. Treatment of Eales Disease with scatter laser photocoagulation. *Ann Ophthalmol* 1989; 21: 300-304.
19. Obana A, Miki T, Matsumoto M. An experimental and clinical study of chorioretinal photocoagulation using a frequency-doubled Nd: YAG laser. *Nippon Ganka Gakai Zasshi* 1993; 97: 1040-1046.
20. Gadkari SS, Kamdar PA, Jehangir RP, Shah NA, Adrianwala SD. Pars plana vitrectomy in vitreous haemorrhage due to Eales` disease. *Indian J Ophthalmol* 1992; 40: 35-37.
21. Stephen CG, Robert PM. Eales` Disease. In : Jakobiec FA, Albert DM, eds. *Principles and Practice of Ophthalmology: Clinical practice*. Philadelphia: WB Saunders Company, 1994; 58: 791-795.
22. Meyer SG. Eales disease, treatment with light coagulation. *Mod Probl Ophthalmol* 1966; 4: 10-14.
23. Pahwa JM, Garg MP. Eales disease-its clinical course and treatment by photocoagulation-a review of 100 cases. *Eye, Ear, Nose, Throat Monthly*; 1968; 47: 174-178.
24. Spitznas M. Eales` disease: clinical picture and treatment with photocoagulation. In L'Esperance FA, ed. *Current diagnosis and management of chorioretinal diseases*. St Louis: CV Mosby Company, 1977; 4:12-16.

Address for correspondence:

Dr. Mazhar Ishaq, FRCS, FRCOphth., Eye Department, Military Hospital, Rawalpindi.

E-mail: khizar_aleem@yahoo.com