# SERUM ELECTROPHORESIS IN PATIENTS OF EALE'S DISEASE

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## ABSTRACT

**Eales disease is an obliterative vasculopathy that usually involve peripheral retina of young adults. It is thought to be a nonspecific tissue response to a number of agents and exact etiology remains unknown. Many diseases reflect some alterations in electrophoretic pattern of serum proteins. This study was carried out to investigate electrophoretic pattern of serum proteins in Eales disease so as to throw some light on possible etiological factors in these patients. Thirty-two patients of Eales disease constituted the subject material for this study. Paper electrophoresis of the sera was performed by cellulose acetate method on Whatman filter paper No.10 using barbitone buffer of pH 8.6. Both patients and controls underwent complete ocular and clinical examination and followed up for one year. This was a descriptive study. Ten patients (30%) had raised total serum proteins out of 32 patients and ten patients (30%) had raised gamma globulin. Serum albumin was decreased in seven patients (20%) out of thirty-two patients of Eales disease. In our study 30% increase in total serum proteins and gamma globulins in Eales disease patients points to a possible role of some immunological process which manifests as retinal perivasculitis in eyes. An increase in erythrocyte sedimentation rate in >85% of cases is consistent with this suggestion.**

**Keywords:** Vitreous haemorrhage, serum electrophoresis, periphlebitis

## INTRODUCTION

In 1880, Henry Eales first described it in healthy young men [1] with abnormal retinal veins and recurrent vitreous haemorrhages [2]. Clinical findings are characterized by avascular areas in retina periphery [3], followed posteriorly by microaneurysm, dilatation of capillary channels, tortuosity of neighbouring vessels and spontaneous chorioretinal scars [4]. It is a diagnosis of exclusion. It is most prevalent in Pakistan, India and Afghanistan affecting healthy young men between third and fourth decade The retinal perivasculitis (Eales disease) is thought to be a non specific response to a number of agents such as hypersensitivity to tuberculoprotein [5-9], as shown by presence of mycobacterium tuberculosis genome in the vitrectomy specimen [10] and epiretinal membranes [11]. An extraneous agent that could result in exposure of normally sequestered pathogenic antigens of immune system, leading to an exuberant immune response in the eye and initiation of the disease [12,13] as the histological changes observed in the perivasculitis of Eales disease resemble the histological changes in blood vessels of collagen vascular diseases [14].

Major cause of visual loss is recurrent vitreous haemorrhage. Severe visual loss occur from complications like persistant vitreous haemorrhage, retinal detachment (tractional or rhegmatogenous or both) & neovascular glaucoma [15].

Systemic steroids have proven beneficial [16]. Laser photocoagulation for non-perfused retina is also useful [17,18]. Pars plana vitrectomy is done for persistant vitreous haemorrhage or retinal detachment [19,20].

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Purpose of study was to investigate electrophoretic pattern of serum proteins in Eales disease so as to throw some light on possible factors which contribute in its etiology.

## PATIENTS & METHODS

It was a descriptive study conducted at Ophthalmology Department, Military Hospital Rawalpindi, between February 2002-April 2003.

Thirty-two patients (fifty-eight eyes) of Eales disease, were considered in this study. Patients were diagnosed as having Eales disease if they had unilateral or bilateral sudden painless loss of vision and obliterative retinal vasculitis. Any systemic disease like Diabetes, sarcoidosis, autoimmune diseases and branch retinal vein occlusion were ruled out before these patients were enrolled. All patients were male serving personnel. Mean age was 30.8 years (range 20-42 years). Disease was bilateral in 26 patients and unilateral in six patients. All patients had a full medical and ophthalmological examination. It included history, physical and ophthalmological examination & laboratory tests including ESR and serum protein electrophoresis.

Ophthalmological examination included determination of visual acuity, intraocular pressure by applanation tonometry, slit lamp examination of anterior segment, direct and indirect ophthalmoscopy, examination of posterior segment & peripheral retina with Goldmann triple mirror (after full pupillary dilatation), fundus photographs and fundus florescein photography in eyes with clear media. Medical & demographic data included patients age, sex, race, medical history, and any medications on presentation. Details of systemic, laser and surgical treatment were also noted.

Sample collected was 5ml whole blood out of which serum was collected. Paper electrophoresis of the sera was performed by cellulose acetate method on Whatman filter paper No. 10 using barbitone buffer of pH 8.6. Quantitative measurement of the relative size of various protein fractions was done in clearing solution which consisted of 85 ml methanol & 15 ml glacial acetic acid.

**Table-1: Percentage values of ESR in Eales disease patients (n=32)**

| Values of ESR (mm at end of 1st hr) | Patients | Percentage |
|---|---|---|
| Upto 20 | 04 | 12.5 |
| Upto 40 | 15 | 47 |
| >40 | 13 | 40.6 |

**Table-2: Age distribution in cases of Eales disease (n=32)**

| Age (years) | Patients | Percentage |
|---|---|---|
| 20-29 | 07 | 21.8 |
| 30-39 | 22 | 68.75 |
| >40 | 03 | 9.4 |

**Table-3: Electrophoretic analysis of Eales disease patients (n=32)**

| Parameters | Mean | Standard Deviation |
|---|---|---|
| ESR (mm fall at 1st hour) | 31.25 | ± 7.45 |
| Total Proteins (g/l) | 75.19 | ± 6.34 |
| S. Albumin (g/l) | 36.84 | ± 3.90 |
| Alpha 1 Globulin (g/l) | 3.50 | ± 0.84 |
| Alpha 2 Globulin (g/l) | 6.47 | ± 1.52 |
| Beta Globulin (g/l) | 8.91 | ± 1.80 |
| Gamma Globulin (g/l) | 17.22 | ± 2.19 |

Follow-up was obtained on thirty patients (fifty-four eyes) because two patients (four eyes) were lost to follow-up as they were boarded out of service. Mean follow-up was 10 months (6 – 24 months). Data was analyzed using SPSS version 10. Descriptive statistics was used to calculate percentages and means alongwith standard deviations of the different fractions of electrophoresis.

## RESULTS

Thirty two patients of Eales disease were taken. Mean ESR was 34 mm at the end of 1st

hour (range 18 - 50). Percentage values of ESR in Eales disease patients is shown in (Table-1) and (Table-2) indicates the age distribution of Eales patients. (Table-3) shows the electrophoretic analysis of patients of Eales disease.

## DISCUSSION

In 1880, Eales described the occurrence of recurrent intraocular haemorrhages associated with enlarged and tortuous retinal vessels in young men [1] with concurrent constipation, epistaxis and headache [2]. Eales disease refers to idiopathic obliterative vasculopathy [4] and phlebitis in young males whose peripheral retina develops sheathing, non-perfusion and neovascularisation [3].

Our findings differ from those of Rengarajan, Namperumalsamy [21] and Hogan who did not observe any consistant abnormal protein patterns in the electrophoretograms of serum proteins in five patients of Eales disease studied by them but in their study. Sample size was very small (five patients only) and it might be due to chance that they did not find any consistant abnormal protein pattern. However, in another study done on twenty four patients of Eales disease and twenty four age and sex matched controls, they used same procedure (cellulose acetate method) of serum electrophoresis and use of Whatman filter paper no. 10. V. B. Pratap, M. K. Mehra and R. K. Gupta [22] found a rise in alpha-1 and alpha-2 globulins in most and in gamma globulins in about 33% of patients in Eales disease. These findings are consistant with our findings.

## CONCLUSION

In the present study, the elevation in serum alpha and gamma globulin fractions in 30% of Eales disease cases points to a possible role of an immunological process, which manifests as retinal perivasculits. An increased value of erythrocyte sedimentation rate in over eighty five percent of these cases is consistent with this suggestion.

## REFERENCES


1. Eales H. Primary retinal haemorrhage in young men. **Ophthalmol Rev 1882;1: 41- 4**
2. Eales H. Causes of retinal haemorrhage with epistaxis and constipation. **Birm Med Rev 1880; 9: 262-3.**
3. Gieser AS, Murphy RP. Eales disease. **Retina 1994; 2: 1503-7.**
4. Patnaik. Eales disease. Clinical features, pathophysiology, etiopathogenesis. Ophthalmol **Clin North Am 1998; 11(4): 601-604.**
5. Madhavan HN, Biswas J. PCR for detection of Mycobacterium TB in epiretinal membranes of patients in eales disease. **Invest Ophthalmol 2000; 41 (3): 822-825.**
6. Hermans PWM, Soolingen V. Specific detection of Mycobacterium TB complex strains by PCR. **J Clin Microbiol 1990; 28: 1204-1213.**
7. Eisenach KD, Cave MD, Bates JH. PCR amplification of a repetitive DNA sequence specific for Mycobacterium.TB. **J Infectious disease. 1990; 161: 977-981.**
8. Abraham C, Baig SM, Badrinath SS. Eales disease. **Proc All India Ophthalmol Soc 1977; 33: 223-229.**
9. Donders PC. Eales disease. **Doc Ophthalmol 1958; 12: 1-105.**
10. 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.**
11. Kimura SJ, Carriker FR, Hogen MJ. Retinal vasculitis with intraocular haemorrhage. Classification and results of special studies. **Arch Ophthalmol 1956; 56: 361-365.**
12. Muthukkaruppan VR, Chakkalatha HR. Immunological status of patients with eales disease. **Indian J Med Res 1989; 90: 351-9.**

13. Biswas J, Narain S, Roy S, Madhavan HN. HLA Association in Eales disease. **Invest Ophthalmol Vis Sci 1996; 36: S363.**

14. Saxena S, Kumar D. Cellular immune response to retinal S-antigens and interphotoreceptor retinoid binding protein fragments in eales patients. **Pathobiology 1999; 67 (1): 39-44.**

15. Kolsi R, Patnaik B. The developing features of phlebitis retinae. **Ind J Ophthalmol 1929; (3): 27-29.**

16. Gastand P, Taieb JC, Laserre L. Eales disease: Therapeutic considerations. **Bull Soc Ophthalmol 1989; 89 (8-9): 1099-1103.**

17. Magargal LE, Walsh AW. Treatment of Eales disease with scatter laser photocoagulation. **Ann Ophthalmol 1989; 21(8): 300-302.**

18. Ishaq M, Niazi MK. Usefulness of laser photocoagulation in managing asymptomatic eyes of Eales disease. **J Ayub Med Coll Abbottabad 2002; 14 (4): 22-25.**

19. Wu N, Zhang J, Yu S. Application of endolaser photocoagulation in vitrectomy. **Chin Ophthal Res 2000; 18 (3): 261-269.**

20. Shanmugam MP,Gopal L,Badrinath SS. Long term visual results of vitrectomy for Eales disease. **Int Ophthalmol 1998; 22 (1)**

21. Rengarajan K, Namperumalsamy P. Biochemical analysis of serum proteins in Eales disease. **Current Eye Res 1989; 8: 1259-1269.**

22. Pratap VB, Mehra MK, Gupta RK. Electrophoresis pattern of serum proteins in Eales disease. **Ind J Ophthalmol 1976; 23:14-16.**