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# Editorial

n this issue, an article by Puri et al on 'Diseases and drug related ocular complications of tuberculosis' is presented [1]. One must be aware of these side effects and also of ocular tuberculosis. Tuberculosis is the most common single cause of morbidity and mortality worldwide, causing nearly 3 million deaths each year [2]. Most of the patients of intraocular tuberculosis fall in the category of uveitis. The most common clinical presentation appears to be posterior uveitis, followed by anterior uveitis, panuveitis and intermediate uveitis. Serpiginous choroiditis, neuroretinitis and sub-retinal abscesses are new entities considered to be cause of intraocular tuberculosis [3]. Diagnosing intraocular tuberculosis is not straightforward as there are variations in clinical presentations and no clear cut diagnostic criteria. Several diagnostic tests are ordered before we reach to the exact diagnosis. Gupta et al [4] have proposed guidelines for the diagnosis of intraocular tuberculosis in which they have divided the cases into two groups, confirmed cases and presumed intraocular tuberculosis. Apart from the clinical presentation mentioned above, ocular investigations like demonstration of AFB or positive PCR from ocular fluid. Systemic investigations like Mantoux test, evidence of healed or active tubercular lesion on X-ray chest or confirmed active extra-pulmonary tuberculosis are required. Tests for exclusion of other uveitic entities like serology for syphilis,

toxoplasmosis etc. should be done. A positive therapeutic response to 4 drugs ATT (isoniazid, rifampicin, ethambutol and pyrazinamide) over a period of 4 to 6 weeks may be tried. The guidelines suggest that therapeutic trial with single drug isoniazid should be avoided due to risk of development of resistance. In nut shell, we have to remember that there should be a high index of suspicion of ocular tuberculosis in entities mentioned above to clinch the diagnosis.

# References

- 1. Paper by Puri from Ophthalmology and Allied Sciences.
- Dye C, Scheele S, Dolin P et al: Consensus statement. Global burden of tuberculosis: estimated incidence, prevalence and mortality by country. WHO Global Surveillance and Monitoring Project.JAMA282: 677-86, 1999.
- 3. Gupta V, Gupta A, Rao Narsing A. Intraocular tuberculosis: An update. Surv Ophthalmol.2007; 52: 561-587.
- 4. Gupta A, Gupta V: Tubercular posterior uveitis. Int. Ophthalmol Clin.2005; 45: 71-88.

# Kamaljeet Singh

Editor-in- chief Ophthalmology and Allied Sciences Professor and Head Department of Ophthalmology MLN Medical College, Allahabad. Email id: kamaljs2@rediffmail.com

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# Correlation between Altered Serum Lipid Profile and Spectral Domain Optical Coherence Tomography based Macular Thickness Parameters in Diabetic Retinopathy

Sandeep Saxena<sup>1</sup>, Shivani Sinha<sup>1</sup>, Khushboo Srivastav<sup>1</sup>, Vinod Kumar B. M.<sup>1</sup>

Authors Affiliation: 1Retina Service, Department of Ophthalmology, King George's Medical University, Lucknow, India.

Abstract

Aim: To study the correlation between altered serum lipid profile and spectral domain optical coherence tomography (SD-OCT) based macular thickness parameters in diabetic retinopathy. Method: Study subjects included 60 cases of type 2diabetes mellitus (DM): no diabetic retinopathy (No DR, n=20); non proliferative DR (NPDR, n=20); proliferative DR (PDR, n=20) and 20 healthy controls. Best corrected visual acuity (BCVA) was measured on logMAR scale. Cube average thickness (CAT) and central subfield thickness (CST) was assessed using SD-OCT. Serum lipid profile was analyzed using standard protocol. Data was analyzed statistically. Result: Decrease in BCVA positively correlated with increased CAT (r=0.25, p=0.028), increased CST (r=0.28, p=0.04), increased serum cholesterol (r=292, p=0.01) and decreased high density lipoprotein (r=-0.714, p=0.01). Statistically significant positive correlation was found between increase in CAT with increase in serum cholesterol (r=0.403, p=0.00) and also with increase in low density lipoprotein (r=0.343, p=0.02.). Conclusion: Deranged lipid profile correlates with the progression of diabetic retinopathy. Further, this study demonstrates the correlation of deranged lipid profile and decreased visual acuity with increased CAT.

**Keywords:** Diabetic Retinopathy; Lipid Profile; Spectral Domain Optical Coherence Tomography; Cube Average Thickness; Central Subfield Thickness.

# Introduction

Diabetic retinopathy (DR) is a micro vascular complication of diabetes mellitus (DM) and is a leading cause of morbidity in people with DM [1]. The prevalence of DR is 18% in urban population older than 40 years with DM [2]. Although the pathogenesis of DR is not completely understood, several risk factors have been established. These include poor glycemic control, hypertension, increasing age, dyslipidemia, serum urea, serum creatinine and duration of DM [3, 4, 5, 6, 7].

Lipoproteins play an indirect role in DR by affecting the integrity of the blood retina barrier (BRB). In retina with an intact BRB, plasma lipoproteins may be largely irrelevant but when BRB is impaired in diabetes, it leads to lipoprotein extravasation and subsequent modification, hence causes toxicity to the neighbouring retinal cells [8]. The external limiting membrane (ELM) is a part of the retinal barrier that is disrupted by pathological conditions contributing to fluid accumulation in the macula, hence affecting the macular thickness [9,10].

In a previous study it was found that high low density lipoprotein (LDL) was found to be associated with increased central subfield macular thickness (CSMT) and central subfield macular volume (CSMV) in diabetic patients without diabetic macular edema (DME) [11]. The present study was undertaken to explore the association of deranged lipid profile with central subfield thickness (CST) and cube average thickness (CAT) in DR.

Reprint Request: Prof. Sandeep Saxena Department of Ophthalmology, King George's Medical University, Lucknow, India. 226003. E-mail: sandeepsaxena2020@yahoo.com

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### Method

The study was conducted according to the tenets of the Declaration of Helsinki after approval from the institutional review board. An informed voluntary consent was obtained from all the study subjects. This was a tertiary care centre based cross sectional study. Sixty consecutive cases of type 2 DM and twenty healthy controls, between age group 45-70 years, were included. Based on the fundus photography and fluoresce in angiography, cases were divided into three groups: patients of diabetes without retinopathy (No DR) (n = 20), non proliferative diabetic retinopathy (NPDR) (n= 20) and proliferative diabetic retinopathy (PDR) (n = 20) according to the ETDRS classification. Cases with ocular or systemic diseases affecting the retinal vascular pathology, end stage renal disease, cases with history of any previous intra-vitreal injection(s), ophthalmic surgical or laser interventions and cases with media haze at any level giving signal strength of less than 5 on OCT were excluded. Cases on lipid lowering medications were also excluded. Best corrected visual acuity (BCVA) was documented on logMAR scale. All the study subjects underwent detailed fundus evaluation using stereoscopic slit lamp bio-microscopy and indirect ophthalmoscopy. Digital fundus photography and flourescein angiography was done using Zeiss fundus camera

FF 450 Plus with pixel width of 0.0054 and image size 2588  $\times$  1958.

All study subjects underwent macular thickness analysis using three dimensional spectral domain optical coherence tomography (SD-OCT) (Carl Zeiss Meditec Inc.,CA, U.S.A). Macular cube analysis 512 × 128 protocol was used figure 1. Blood samples were collected from all the study subjects by aseptic venepuncture.

Total cholesterol (CHO) and triglycerides (TGs) were measured by enzymatic method. High density lipoprotein (HDL) was analysed using phosphate tungsten method. All tests were performed using standard protocol. Very low density lipoprotein (VLDL) and LDL were calculated using the above values [VLDL=TG/5, LDL= (VLDL+HDL) - cholesterol].

Data has been summarized as Mean  $\pm$  SE. The continuous variables of the study groups were compared by one factor analysis of variance (ANOVA). The discrete (categorical) variables were compared by chi-square ( $\pm$ 2) test. For pair wise comparison between the groups, Tukey's test for multiple comparisons was used. The logMAR vision score of two groups (NPDR and PDR) was compared by independent Student's t test. Pearson correlation analysis was used to assess association between the variables. A p<0.05 was considered statistically significant. All analyses were performed STATISTICA 6.0 software package (StatSoft, 2001).



Fig. 1: Spectral domain optical coherence tomography showing macular thickness analysis on a macular cube using 512× 128 protocol in diabetic macular edema.

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	Central Subfield	Cube Volume	Cube Average
	Thickness (µm)	(mm³)	Thickness (µm)
ILM-RPE	453	12.2	340

# Results

Mean age (in years) of the four groups was  $53.6 \pm 8.06$  in controls,  $54.00 \pm 6.05$  in No DR,  $57.6 \pm 4.64$  in NPDR and  $55.3 \pm 8.14$  in PDR groups. No significant

difference in the age was observed among the groups (F=2.46, p=0.06).

The  $\div^2$  test revealed similar (p>0.05) sex proportion among all the four groups (Male/Female: 6/14 vs. 13/7 vs. 14/6 vs. 15/5,  $\div^2$ =7.2 p=0.080). Mean duration of diabetes mellitus in years was 7.14  $\pm$  5.22 in No DR, 10.38  $\pm$  5.91 in NPDR and 12.18  $\pm$  4.66 in PDR groups. Significant association of severity of diabetic retinopathy with increase in the duration of the disease was documented (F=17.62, p<0.0001).

Mean glycated hemoglobin (%) was  $6.08 \pm 1.22$ ,  $6.36 \pm 0.61$ ,  $7.28 \pm 1.48$  and  $7.71 \pm 1.91$  in controls, NODR, NPDR and PDR respectively. No significant difference was found between glycated hemoglobin among the groups on analysis of variance (ANOVA).

Mean logMAR BCVA was  $0.04 \pm 0.09$  in control,  $0.3 \pm 0.36$  in No DR,  $0.5 \pm 0.39$  in NPDR and  $1.4 \pm 0.40$  in PDR groups. On ANOVA, significant difference in visual acuity was found among the group (F=42.68, p<0.0001).

Table 1 summarizes the central subfield thickness (CST) and cube average thickness (CAT) in study group. Decrease in BCVA was significantly associated with increase in CST (r=0.28, p=0.04) and CAT (r=0.262, p=0.018).

Mean values of the serum levels of CHO, HDL, LDL and VLDL has been shown in Table 2. While analyzing the lipid profile using ANOVA, difference in serum CHO (F=6.617, p<0.001), serum HDL (F=4.436, p<0.001), serum LDL (F=6.274, p<0.001), serum VLDL (F=6.17, p<0.001) was found between the study groups.

Table 3 shows correlations between various biochemical parameters with CAT, C9T and BCVA. On pearsons correlation analysis, CST was not significantly correlated with serum CHO (r=0.172, p=0.135), HDL (r=-0.120, p=0.297), LDL (p=0.192, p=0.095) and VLDL (r=-0.63, p=0.585). CAT was found to be correlated with CHO (r=0.403, p=0.00), HDL (r=0.42, p=0.714), LDL (p=0.343, p=0.02) and VLDL (r=0.159, p=0.167) on applying pearsons correlation. Increased logMAR BCVA was significantly associated with increased serum cholesterol (p=0.01) and decreased HDL (p=0.01). There was no significant association between BCVA with LDL (r=0.312, p=0.06) and VLDL (r=0.041, p=0.723).

Table 1: Summary (Mean ± SD) of central subfield thickness and cube average thickness in study group

Variable		Grou	ups	
Vallable	Controls	No DR	NPDR	PDR
Mean of central subfield thickness(µm)	249.90 ±11.52	234.73 ±31.63	313.35±120.05	367.1±119.9
Mean of cube average thickness(µm)	244.31±12.41	264.52±16.10	303.58±52.42	319.6±73.56

Table 2: showing various biochemical parameters amongst different groups

	Groups					
	Controls	No DR	NPDR		PDR	
S. cholesterol (mg/dl)	141.36±23.64	171.18±41.59	176.15±36.11		205.51±61.67	
S. triglyceride (mg/dl)	90.90±10.99	127.1±46.78	1 128.19±51.19	1	157.24±38.11	
S. high density lipoprotein (mg/dl)	45.23±7.89	44.44±14.93	43.41±15.69		39.07±10.88	
S. low density lipoprotein (mg/dl)	72.21±15.39	93.71±42.90	102.29±33.81		127.17±55.17	
S. very low density lipoprotein (mg/dl)	24.57±7.71	27.46±15.99	26.25±9.32		31.09±9.17	

Table 3: Correlation of various bio-chemical parameters with CST, CAT and visual acuity

	CST( µm)		CAT ( µm)		logMAR visual acuity	
	Correlation(r)	P value	Correlation (r)	P value	Correlation(r)	P value
S. cholesterol (mg/dl)	0.172	0.135	0.403	0.00	0.292	0.010
S. high density lipoprotein (mg/dl)	-0.120	0.297	-0.42	0.714	-0.148	0.010
S. low density lipoprotein (mg/dl)	0.192	0.095	0.343	0.02	0.312	0.06
S. very low density	-0.63	0.585	0.159	0.167	0.041	0.723
Visual acuity	0.28	0.04	0.262	0.018	1.00	1.00

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# Discussion

Our present study was aimed at establishing a correlation of deranged lipid profile and BCVA with CAT and found that deranged lipid profile is significantly correlated with decreased BCVA and with increased CAT. It also shows that increased CST and increased CAT are positively correlated with decreased BCVA.

Severity of diabetic retinopathy was found to be significantly associated with duration of disease in accordance with a study by Correa et al [12].

Various studies have found decreased visual acuity to be significantly associated with increase in grade of ELM and inner segment ellipsoid band (ISel) disruption in DM [13, 14]. The grades of disruption increases with increase in severity of diabetic retinopathy. Our previous studies involving nitric oxide, oxidative stress, advanced glycation end products, VEGF and ICAM in DR have been associated with in vivo structural changes in inner segment ellipsoid and retinal pigment epithelium [15, 16, 17]. Another recent study of ours has found a significant association between increase in central subfield thickness and grade of inner segment ellipsoid band (ISel) disruption on SD-OCT with progression of diabetic retinopathy [18].

Our current study has correlated significantly the decrease in visual acuity with increase in the severity of retinopathy, similar to studies concluded by Falkenstein et al [19]. We found in our study that decrease in BCVA was significantly correlated with increased CST and CAT and was in accordance with the study conducted by Sasaki et al [10]. But in a study by Otani et al, CST was found to be weakly and negatively correlated with BCVA [20]. Significant correlation has been found between OCT patterns of clinically significant diabetic macular edema and severity of retinopathy, central macula thickness (CMT) and BCVA [21].

The study by Wu et al demonstrated that heavily oxidized-glycated LDL induced the activation of caspase, mitochondrial dysfunction and apoptosis in human retinal capillary pericytes suggesting potentially important role of extravasated, modified LDL in promoting DR by promoting apoptotic pericyte loss [22]. Recently it has also been shown that levels of circulating oxidized LDL immune complexes (ox-LDL-ICs) predict the development of DR [23]. In retinal sections from people with type 2 diabetes mellitus, ox-LDL and IgG was present proportionate to DR severity. Ox-LDL-IC exhibited greater cytotoxicity than ox-LDL toward retinal pericytes. Another study elaborated the role of lipids in diabetic retinopathy by studying the effect of cholesterol lowering agents ie., statins on BRB in DR. Statins normalize the expression of pro-inflammatory factors which are drastically up-regulated in diabetic retina [24]. This further supports the role of lipids in pathogenesis of DME.

The study by Sasaki et al associated high LDL with increased CSMT and CSMV in diabetic patients without DME [10]. High serum cholesterol, LDL and non HDL levels were also found to be associated with retinal hard exudate formation, CSME, decreased BCVA and with DME in patients of type 2 DM [25, 26, 27, 28].

Our recent study highlighted, significant correlation of deranged lipid profile with ELM and ISel disruption [29]. Deranged lipid profile was found to have a significant correlation with progression of diabetic retinopathy in our present study which is in harmony of previous studies where high TGs and low HDL were found to be associated with increased severity of DR [30, 31, 32, 33, 34]. This present study significantly positively correlated increased serum levels of CHO and LDL levels with increased CAT but not with increased CST.

In our study increased serum CHO and decreased HDL was found to be significantly correlated with decrease in BCVA.

# Conclusion

Deranged lipid profile is significantly correlated with decreased BCVA and with increased CAT. Increased CST and increased CAT were positively correlated with decreased BCVA.

# References

- Cheung N, Wong TY: Diabetic retinopathy and systemic complications. In Diabetic Retinopathy. Duh EJ, Ed: Totowa, NJ, Humana Press. 2009: 465–482.
- Raman R, Rani PK, Reddi RS, Gnanamoorthy P, Uthra S, Kumaramanickavel G, Sharma T. Prevalence of diabetic retinopathy in India: Sankara Nethralaya Diabetic Retinopathy Epidemiology and Molecular Genetics Study report 2. Ophthalmology. 2009; 116: 311-318.
- West KM, Erdreich LJ, Stober JA. A detailed study of risk factors for retinopathy and nephropathy in diabetes. Diabetes. 1980; 29: 501-508.

- 4. Haddad OAWE, Saad MK. Prevalence and risk factors for diabetic retinopathy among Omani diabetics. Br J Ophthalmol. 1998; 82: 901-906.
- 5. Ashakiran S, Krishnamurthy N, Navin S, Patil S. Behaviour of serum uric acid and lipid profile in relation to glycemic status in proliferative and non-proliferative diabetic retinopathy. Current Neurobiology. 2010; 2: 57-61.
- 6. Bloomgarden ZT. Screening for and managing diabetic retinopathy: current approaches. American Journal of health-system pharmacy.2007; 64: S8-S14.
- CY Emily, KL Michael, L Frederick. Association of Elevated Serum Lipid Levels With Retinal Hard Exudate in Diabetic RetinopathyEarly Treatment Diabetic Retinopathy Study (ETDRS) Report 22. Arch Ophthalmol. 1996; 114: 1079-1084.
- 8. Yu JY, Lyons TJ: Modified Lipoproteins in Diabetic Retinopathy: A Local Action in the Retina. J Clin Exp Ophthalmol. 2013; 18: 314.
- Omri S, Omri B, Savoldelli M, Jonet L, Thillaye-Goldenberg B, Thuret G, Gain P, Jeanny JC, Behar-Cohen F. The outer limiting membrane (OLM) revisited: clinical implications. Clin Ophthalmol. 2010; 4: 183–95.
- Saxena S, Srivastava K, CM Chui, Cheung G, L Timothy. Photoreceptor inner segment ellipsoid band integrity on spectral domain optical coherence tomography. Clin Ophthalmol. 2014;8:2507–2522.
- Sasaki M, Kawashima M, Kawasaki R, Uchida A, Koto T, Shinoda H, Kazuo T, Wang JJ, Ozawa Y. Association of Serum Lipids With Macular Thickness and Volume in Type 2 Diabetes Without Diabetic Macular Edema. Investigative ophthalmology and visual science. 2014.
- 12. Corrêa ZMS, Freitas AM, Marcon IM. Risk factors related to the severity of diabetic retinopathy. Arg Bras Oftalmol. 2003; 66: 739-43.
- Maheshwary AS, Oster SF, Yuson RMS, Cheng L, Mojana F, Freeman WR. The association between percent disruption of the photoreceptor inner segment-outer segment junction and visual acuity in diabetic macular edema. AmJophthalmol. 2010; 150: 63–67.
- Browning DJ, Glassman AR, Aiello LP, Beck RW, Brown DM, Fong DS, Bressler NM, Danis RP, Kinyoun JL, Nguyen QD, Bhavsar AR, Gottlieb J, Pieramici DJ, Rauser ME, Apte RS, Lim JI, Miskala PH. Relationship between Optical Coherence Tomography–Measured

Central Retinal Thickness and Visual Acuity in Diabetic Macular Edema. Ophthalmology. 2007; 114: 525-536.

- 15. Sharma S, Saxena S, Srivastav K, Shukla R, Mishra N, Meyer CH, Kruzliak P, Khanna VK. Nitric oxide and oxidative stress is associated with severity of diabetic retinopathy and retinal structural alterations. Clin Experiment Ophthalmol. 2015; 12.
- Jain A, Saxena S, Khanna VK, Shukla R, Meyer CH. Status of serum VEGF and ICAM-1 and its association with external limiting membrane and inner segment-outer segment junction disruption in type 2 diabetes mellitus. Mol Vis. 2013; 19: 1760–1768.
- Saxena S, Mishra N, Khanna V, Jain A, Shukla R, Meyer CH. Increased Serum N-CML, VEGF and ICAM-1 is Associated with Photoreceptor Inner Segment Ellipsoid Disruption in Diabetic Retinopathy. JSM Biotechnol Bioeng 2: 1039
- Sharma SR, Saxena S, Mishra N, Akduman L, Meyer CH. The Association of Grades of Photoreceptor Inner Segment-Ellipsoid Band Disruption with Severity of Retinopathy in Type 2 Diabetes Mellitus. Journal of Case Reports and Studies. 2014: 10.
- 19. Falkenstein I, Cochran D, Azen S. Comparison 'of visual acuity in macular degeneration patients measured with Snellen and early treatment diabetic retinopathy study charts. Ophthal mology. 2008; 115: 319-323.
- 20. Otani T, Yamaguchi Y, Kishi S. Correlation between visual acuity and foveal microstructural changes in diabetic macular edema. Retina.2010; 30: 774-780.
- 21. A Hisham, K Dustan, AM Ahmed. The correlation between optical coherence tomographic features and severity of retinopathy, macular thickness and visual acuity in diabetic macular edema. International Ophthalmology. 2005; 26: 93-99.
- 22. Wu M, Chen Y, Wilson K, Chirindel A, Michael AI, Yu Y, Boulton ME, Szweda LI, Ma JX, Lyons TJ. Intraretinal Leakage and Oxidation of LDL in Diabetic Retinopathy. Invest. Ophthalmol. Vis. Sci. 2008; 49 (6): 2679-2685.
- Fu D1, Yu JY, Wu M, Du M, Chen Y, Abdelsamine SA, Li Y, Chen J, Boulton ME, Ma JX, Lopes FM, Virella G, Lyons JT. Immune complex formation in human diabetic retina enhances toxicity of oxidized LDL towards retinal capillary pericytes. J Lipid Res. 2014; 55: 860-9.

- 24. J Li, JJ Wang, D Chen. Systemic administration of HMG-CoA reductase inhibitor protects the blood-retinal barrier and ameliorates retinal inflammation in type 2 diabetes. Experimental eye research.2009; 89: 71-78.
- J Idiculla, S Nithyanandam, VA Mohan, Vasu U, Sadiq M. Serum lipids and diabetic retinopathy: A cross-sectional study. Indian J Endocrinol Metab. 2012; 16: S492–S494.
- Franks SM, Michel HP, Fioretto P, Valensi P, Davis T, Horton, Wanner C. Association Between Plasma Triglycerides and High-Density Lipoprotein Cholesterol and Microvascular Kidney Disease and Retinopathy in Type 2 Diabetes Mellitus A Global Case–Control Study in 13 Countries. Circulation. 2014; 129: 999-1008.
- 27. T A Chowdhury, D Hopkins, P M Dodson, Vafidis GC. The role of serum lipids in exudative diabetic maculopathy: is there a place for lipid lowering therapy?. Eye. 2002; 16: 689-693.
- Rema M, Srivastava BK Anitha B, Deepa R, Mohan V. Association of serum lipids with diabetic retinopathy in urban South Indians: the Chennai Urban Rural Epidemiology Study (CURES) Eye Study–2. Diabet Med. 2006; 23:1029–1036.

- 29. Jain A, Saxena S, Ruia S, Srivastav K, Natu MS. Altered lipid profile is associated with external limiting membrane and inner segment ellipsoid band disruption in type 2 diabetes mellitus: A preliminary study. Open Science Journal of Clinical Medicine. 2015; 3: 37-41
- 30. Rahman MR, Arslan MI, Hoque MM. Serum Lipids and Diabetic Retinopathy in Newly Diagnosed Type 2 Diabetic Subjects. J Enam Med Col. 2011;1: 63-66.
- Lyons JT, Jenkins AJ, Zheng D, Lackland DT, McGee D, Garvey WT, Klein RL. The DCCT/ EDIC Research Group. Diabetic retinopathy and serum lipoprotein subclasses in the DCCT/EDIC cohort. Invest Ophthalmol Vis Sci. 2004; 45: 910– 918.
- 32. Kissebah AH, Kohner EM, Lewis B, Siddiq YK, Lowy C, Fraser TR: Plasma lipids and glucose/ insulin relationship in non-insulin-requiring diabetics with and without retinopathy. Lancet 1975; 305: 1104-1108.
- 33. Yu Y., Lyons T.J. A lethal tetrad in diabetes: hyperglycemia, dyslipidemia, oxidative stress, and endothelial dysfunction. The American journal of the medical sciences. 2005; 330: 227-232.
- 34. D Guyer, L Yannuzzi, S Chang S. Diabetic Retinopathy. Retina. 2007; 1: 316-344.

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# Anterior Segment–OCT Guided, Endo-illuminator Assisted Management of Descemet's Membrane Detachment

# Kamaljeet Singh<sup>1</sup>, S. P. Singh<sup>1</sup>, Kshama Dwivedi<sup>1</sup>, Jagriti Rana<sup>1</sup>, Sushank A. Bhalerao<sup>1</sup>, Harsh Mathur<sup>1</sup>

Author Affiliation <sup>1</sup>Regional Institute of Ophthalmology at Govt. M. D. Eye Hospital, Allahabad, Uttar Pradesh, India.

# Abstract

Objective: To study the anatomic and visual outcomes of descemetopexy in Descemet's membrane detachment (DMD) after cataract surgery with the help of Anterior Segment -OCT (AS-OCT) and endo-illuminator. Design: A prospective study. Participants: Nineteen eyes of 19 patients. Methods: This study was carried out at Regional Institute of Ophthalmology (M.D. Eye Hospital, Dr. Katju Road, Nakhas Kona, Allahabad, India) after taking permission from ethical committee of M.L.N. Medical College, Allahabad from April 2013 to March 2014. We used Anterior segment OCT (AS-OCT) and endoilluminator in the management of Descemet's membrane detachment. Descemetopexy was performed with air or 14% isoexpansile perfluoropropane (C3F8) or 20% SF6 gas. Main Outcome Measures: Anatomical (reattachment rates) and functional results (best-corrected visual acuity) were studied. Secondary outcome measures were assessment of surgical complications and association of various factors with final visual outcome. Results: With the help of endo-illuminator

# Introduction

Descemet's membrane detachment (DMD) is an Duncommon but serious complication of intraocular surgery [1]. It occurs when fluid enters the corneal stroma through a break in Descemet's membrane (DM) or an area of separation between the DM and the corneal stroma. Acute loss of vision from severe corneal edema can be the first sign and may also be the cause of a delayed diagnosis [2]. In and OCT, we treated total 19 patients of Descemet's membrane detachment following cataract surgery with intra-cameral injection of air (3 patients), perfluoropropane (7 patients) and sulphur hexafluoride (9 patients), figure 2. Out of the 19 patients 13 (68%) patients had final visual acuity between 6/6-6/12 and 6 (32%) patients had final visual acuity between 6/12-6/24, figure 3. Reattachment occurred within 48 hours in 15 patients and after 48 hours in 4 patients. Residual DMD was found in one case in which we re-injected SF6. Conclusion: Anterior Segment Optical Coherence Tomography(OCT) guided, endoilluminator assisted intra-cameral injection of sulphur hexafluoride (SF6) gas is the best way of management of Descemet's membrane detachment as compared to intra-cameral injection of air and perfluoropropane (C3F8) gas. Financial Disclosure(s): The author(s) have no proprietary or commercial interest in any materials discussed in this article.

**Keywords:** Anterior Segment Optical Coherence Tomogram; Descemet's Membrane Detachment; Descemetopexy; Endo-Illuminator.

1928, soon after the advent of slit-lamp biomicroscopy, the first systematic description of DMD in the American literature was made by Bernard Samuels [3]. Samuels reported three patients with

> Reprint Request: Prof. Kamaljeet Singh, 4A / 7, Panna Lal Road, Allahabad, UP (INDIA) 211003. E-mail: kamaljs2@rediffmail.com

DMD after iridectomy, but he failed to realize its significance. Indeed, the subsequent literature reflected little interest in this entity until 1964, when Scheie [4] realized the potentially serious nature of this surgical complication in his report of three patients who did poorly with DMD after cataract extraction.

A review of literature revealed that only one report has determined the incidence of DMD. It was found to be 2.6% for extra-capsular cataract extraction (ECCE) and 0.5% for phacoemulsification [5]. The presence of DM tags or scrolls along the interior lip of the sclera-corneal incision have been noted, with an incidence determined by gonioscopy to be 11% to 42% [6, 7]. There is no clarity in the existing literature regarding the need for surgical reattachment [8-11] and the efficacy of various substances used as tamponade, such as 100% air, viscoelastic material, 14% isoexpansile perfluo ropropane (C3F8) and 20% sulphur-hexafluoride [12]. Potter and Zalatimo (13) have reported air to be the least efficacious tamponade for descemetopexy.

Early treatment of this condition is important as it can prevent permanent corneal opacification due to endothelial pump failure. Descemet's membrane detachment (DMD) has been classified as planar Descemet's membrane detachment if separation is <1mm and non Planar if separation is > 1mm from stroma. Each one is further classified in to central and combined (central and peripheral). Diagnosis can be made on slit-lamp examination, but in cases of corneal edema, anterior segment optical coherence tomogram (AS-OCT) would be helpful in determining the position of detached flap, which will allow us to prepare the surgical plan more appropriately. Descemet's membrane detachment has been reported with cataract extraction, iridectomy, trabeculectomy, penetrating keratoplasty, pars plana vitrectomy, deep sclerectomy, and viscocanalostomy etc.

Considering the significance of DMD discussed earlier and taking into account the lack of consistency and clarity in the existing literature, we aim to compare the outcomes of descemetopexy after cataract surgery with respect to the use of air, C3F8 and SF6 gas. To the best of our knowledge, this is the first comparative study published so far to report the use of Anterior Segment –OCT (AS-OCT) and endoilluminator in the management of Descemet's membrane detachment (DMD) after cataract surgery.

# Material and Methods

# Study Design and Subjects

This prospective study was carried out at Regional Institute of Ophthalmology (M.D. Eye Hospital, Dr.

Katju Road, Nakhas Kona, Allahabad, India) after taking permission from ethical committee of M.L.N. Medical College, Allahabad from April 2013 to March 2015. As per hospital protocol, written informed consent was obtained from all patients before all the surgical procedures and the investigations that they underwent.

This study included patients who underwent anterior chamber gas injection (descemetopexy) for the treatment of DMD after cataract surgery during April 2013 to March 2015. Descemet's membrane detachment was identified on slit-lamp examination as separation of the DM from the posterior stroma with an area of corneal edema overlying it (Figure 1). Inclusion criteria were: the patients who underwent cataract surgery, non-planar type of Descemet's membrane detachment, patients diagnosed as Descemet's membrane detachment within one month of cataract surgery. Therefore, 19 patients who fulfilled the inclusion criteria were included in the study.

Patients Diagnosed as Descemet's Membrane Detachment after One Month of Cataract Surgery were Excluded From The Study.

# Data Collection

The data collected from the medical records included age, sex, the eye operated, presence of any corneal opacity (which obscured the view of the anterior segment during the cataract surgery), type of initial cataract surgery, preoperative visual acuity, pre-operative and post-operative intraocular pressure(IOP), location and degree of DMD and duration between descemetopexy and previous cataract surgery. Other details noted were the type of gas used for anterior chamber injection and any intra-operative or postoperative complications. Postoperative details at 1 month included status of DM attachment and bestcorrected visual acuity.

# Outcome Measures

The primary outcome measures were studied after 1 day, 1 week and 1 month after descemetopexy with the help of anterior segment optical coherence tomogram (AS-OCT). Assessment was done for the final status of the detached portion of the DM (anatomic) and the improvement in the visual acuity (functional). The size of the gas bubble was also assessed. The secondary outcome measures included complications of the procedure and the association of various factors with the final visual outcome.

# Preoperative Examination

All patients underwent visual acuity testing, slitlamp examination, intraocular pressure measurement by non-contact tonometer (Topcon CT-80), direct and indirect ophthalmoscopy and anterior segment optical coherence tomography (AS-OCT and Carl Zeiss Meditech, Dublin, CA, USA). AS-OCT helps in the detailed evaluation of Descemet's membrane detachment in all the quadrants of the cornea.

# Surgical Technique

Descemetopexy was performed by the same surgeon (KJS) in the operating room with all aseptic precautions using topical or local anesthesia. In this study, we used a powerful light source in the form of endoilluminator, which is more commonly used in vitreo-retinal surgeries. The endoilluminator, when placed at the limbus, illuminates the anterior chamber and thus provides a better view of the detached Descemet's membrane. Under an operating microscope, a 30G cannula was taken and mounted on a one-ml disposable syringe. The syringe was filled with 100% air, isoexpansile mixture of 14% C3F8 or 20% SF6 gas, the choice of which was predecided.

The gas was aspirated into the syringe through a micropore filter. The site of entry was in an area 180 degree opposite to the area of the DMD. The syringe filled with gas or air was inserted into the anterior chamber under the guidance of the endoilluminator. A continuous, single bubble of the gas was aimed into the anterior chamber. The eye was patched in most cases.

# Postoperative Management

On the first postoperative day, the patient was examined specifically to assess the attachment of the DM with the help of anterior segment optical coherence tomogram (AS-OCT). A standard postoperative treatment of topical antibiotics (ofloxacin 0.3% or moxifloxacin 0.5% eye drops) given for 1 week and tapering doses of topical steroids (betamethasone 1% or prednisolone acetate 1%) given for 5 weeks was followed. Postoperatively, any increase in intraocular pressure measured with noncontact tonometer (NCT) was managed with topical or oral antiglaucoma medication. Further follow-ups were done at 1 week and 1 month. The anatomic attachment of the DM, vision, and intraocular pressure were noted at every visit. Anterior segment optical coherence tomogram (AS-OCT) was performed in all patients postoperatively to confirm the attachment of the DM. The surgical procedure was repeated if the DMD persisted on the first postoperative day, as evaluated on a slit lamp or AS-OCT.

# Results

We treated 19 patients (19 eyes) with DMD after cataract surgery. 17 patients were operated in our institution and 2 were patients who referred to our institution. The mean age at the time of presentation was 71 years (range, 61–79 years); 12 subjects were women, and 9 were men.

The endoilluminator when placed at the limbus illuminates the anterior chamber and thus provided a better view of the detached Descemet's membrane during descemetopexy. With the help of endo-illuminator and OCT, we treated total 19 patients of Descemet's membrane detachment following cataract surgery with intra-cameral injection of air (3 patients), perfluoropropane 14% (7 patients) and sulphur hexafluoride (9 patients), figure 2. Out of the 19 patients 13 (68%) patients had final visual acuity between 6/6-6/12 whereas 6 (32%) patients had final visual acuity between 6/12-6/24, figure 3. Reattachment occurred within 48 hours in 15 patients while reattachment occurred after 48 hours in 4 patients, figure 4. It is better to use air and SF6 gas as compared to C3F8 because C3F8 gas lasts much longer than SF<sub>6</sub> gas and may consequently be more toxic to the endothelium.

We found that reattachment rates were higher when descemetopexy was performed within one week of cataract surgery as compared to when performed later. No post-operative complications were noted except raised intraocular pressure found in one patient with intra-cameral C3F8 gas injection which was treated with acetazolamide tablet BD. Residual DMD was found in one case in which we re-injected SF6 gas which was previously treated with Air Descemetopexy.

Results with intra-cameral injection of air, perfluoropropane 14% and sulphur hexafluoride are summarised in following table:

C Pre-op		1	Degree of	Gas	Re-attachment of	Pre-Op IOP	Post-Op IOP		Post-Op BCV	/A
Case	VA	Location of DMD	DMD in mm	injected	DMD	(mm Hg)	(mm Hg)	Day 1	Day2	1 month
								Day 1	Day2	1 month
1	FC 1m	Combined, Central and peripheral	>1mm	AIR	24 hrs	10	14	6/36	6/12	6/6P
2	FC 2m	Combined, Central and peripheral	>1mm	AIR	72 hrs	12	16	6/60	6/18	6/12
3	FC 1m	Combined, Central and peripheral	>1mm	SF6	24 hrs	14	18	6/36P	6/9	6/6
4	FC 3m	Combined, Central and peripheral	>1mm	C3F8	24 hrs	12	16	6/24	6/12	6/6P
5	FC 2m	Combined, Central and peripheral	>1mm	C3F8	36 hrs	12	14	6/60	6/18	6/12
6	FC 3m	Combined, Central and peripheral	>1mm	SF6	18 hrs	10	16	6/36	6/9	6/6
7	FC 3m	Combined, Central and peripheral	>1mm	SF6	24 hrs	12	16	6/36P	6/12	6/6P
8	FC 1m	Combined, Central and peripheral	>1mm	C3F8	24 hrs	10	14	6/36	6/12	6/6P
9	FC 2m	Combined, Central and peripheral	>1mm	C3F8	36 hrs	12	16	6/60	6/18	6/18
10	FC 1m	Combined, Central and peripheral	>1mm	SF6	24 hrs	14	18	6/36P	6/9	6/6
11	FC 3m	Combined, Central and peripheral	>1mm	AIR	24 hrs	12	16	6/24	6/12	6/6P
12	FC 1m	Combined, Central and peripheral	>1mm	SF6	72 hrs	10	14	6/36	6/12	6/12
13	FC 2m	Combined, Central and peripheral	>1mm	C3F8	96 hrs	12	16	6/60	6/18	6/12
14	FC 1m	Combined, Central and peripheral	>1mm	C3F8	24 hrs	14	18	6/36P	6/9	6/6
15	FC 3m	Combined, Central and peripheral	>1mm	SF6	24 hrs	12	16	6/24	6/12	6/6P
16	FC 1m	Combined, Central and peripheral	>1mm	SF6	24 hrs	10	14	6/36	6/12	6/6P
17	FC 2m	Combined, Central and peripheral	>1mm	SF6	72 hrs	12	16	6/60	6/18	6/12
18	FC 1m	Combined, Central and peripheral	>1mm	SF6	24 hrs	14	18	6/36P	6/9	6/6
19	FC 3m	Combined, Central and peripheral	>1mm	C3F8	24 hrs	12	16	6/24	6/12	6/12

Table 1: Pre- operati	/e and post-operative	e assessment of	patients of d	lescemetopexy \	with air,	C3F8 and SF6
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Fig. 1: Slit-lamp photographs of a case of Descemet's membrane detachment showing corneal edema with Descemet's membrane (DM) break.



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Fig. 2: Distribution of patients as per the gases injected



Fig. 3: The pie chart representing the visual outcomes in the 19 patients



Visual Outcome

Fig. 4: The bar diagram showing reattachment of DMD within 48 hours and after 48 hours



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# Discussion

Descemet's membrane detachment is a rare but vision threatening complication of cataract surgery [1, 2, 5]. There is a relative paucity in the literature regarding the guidelines for the management of DMD. Some of the questions that still remain unanswered are whether an intervention is warranted in all cases of DMD and, if required, what duration is ideal, which is the best agent to treat it with, and whether any other factors influence its final outcome.

Since Samuels's article [3] in 1928, several mechanisms of surgically induced DMD have been proposed. Samuels alluded to a shallow chamber as a risk factor and stated that complicated or repeated operations were at particular risk. The inadvertent insertion of instruments between the corneal stroma and Descemet's membrane was first suggested by Scheie [4].

Observation in cases of smaller or inferior DMD might avoid the inherent risks of intervention [8, 9], but a delay in repair may be complicated by fibrosis, shrinkage, and wrinkling of DM, which may subsequently prevent reattachment [14]. In a series of 5 patients, Assia et al [15] stated that observing a non-scrolled DMD conservatively might lead to its resolution, but the duration of recovery was prolonged, ranging from 1 to 3 months. Modern cataract surgery is considered to be a refractive surgery, and patients expect excellent vision almost immediately. Thus, despite several reports of spontaneous DMD reattachment, [8, 9] an early repair is advocated [13, 16, 17].

Anterior chamber injection of gas as the primary management strategy has been well described [2]. It can hasten the absorption of corneal edema and, thus, visual recovery. We have attempted to analyze the various risk factors that affect the occurrence and resolution of a DMD. In this study, we found that descemetopexy with SF6 or C3F8 for DMD after cataract surgery had good anatomic and functional outcomes. However, the final visual acuity in our study was adversely affected if the Descemet's detachment occurred in patients who had a more advanced cataract, underwent an SICS, or had a prolonged duration of intervention after cataract surgery.

Similar to our findings, the existing literature states that the visual acuity of patients with DMD after the descemetopexy procedure is significantly better than their preoperative visual acuity, [18, 19] proving the necessity of treatment for DMD. A repeat descemetopexy also is not uncommon. One of 19 patients required repeat intervention in our study, with comparable visual and functional outcomes at 1 month. Mahmood et al [14] and Marcon et al [18] also had to resort to repeat injections in a few patients in their study because the primary injections failed to successfully attach the detached DM.

Contrary to our findings, Marcon et al [18] and Kim et al [20] reported that DMDs did not require urgent surgical repair, and the decision on when to intervene in DMDs must be made on a case-by-case basis. Mackool and Holtz [21] proposed a classification scheme to help determine the prognosis for DMDs. They separated DMDs into planar and non-planar, depending on whether Descemet's membrane was separated from the posterior corneal stroma by  $\leq 1$  mm or >1 mm, respectively. They believed that planar detachments had the best prognosis for spontaneous reattachment, whereas non-planar detachments were unlikely to spontaneously reattach and should be repaired early. We performed descemetopexy only in all non-planar type of descemet's membrane detachment (DMD).

The use of 100% air as a tamponade has been believed to be least effective, and success has been shown with longstanding intra-cameral gases such as sulphur-hexafluoride or C3F8(13). In this study, we compared the use of 20% SF6, 14% isoexpansile C3F8 and 100% air for descemetopexy and found that the reattachment rates were the same with the 14% C3F8 gas and 20% SF6 gas. But, it was better to use air and SF6 gas as compared to C3F8 because C3F8 gas lasts much longer than SF6 gas and may consequently be more toxic to the endothelium. Early pupillary block was also seen in an eye in which C3F8was used. SF6 was found to be better than air as it lasted for 2 weeks as compared to air which got absorbed within 3 to 4 days. Residual DMD was found in one case in which we re-injected SF6 which was previously treated with air Descemetopexy.

We found significantly worse final visual acuity in eyes with a longer duration between the cataract surgery and the descemetopexy, in eyes that had undergone SICS procedures, and in eyes in which C3F8 gas was used.

# Conclusion

We concluded that Anterior Segment Optical Coherence Tomography(OCT) guided, endo-

illuminator assisted intra-cameral injection of sulphur hexafluoride (SF6) gas is the best way of management of Descemet's membrane detachment as compared to intra-cameral injection of air and perfluoropropane(C3F8) gas.

# References

- Jaffe NS. Cataract surgery and its complications. In: Jaffe NS, editor. 4th ed. St. Louis: Mosby, 1984. p. 627-36.
- Makley TA Jr, Keates RH. Detachment of Descemet's membrane (an early complication of cataract surgery). Ophthalmic Surg 1980; 11: 189 –91.
- 3. Samuels B. Detachment of Descemet's membrane. Trans Am Ophthalmol Soc 1928; 26: 427–37.
- 4. Scheie HG. Stripping of Descemet's membrane in cataract extraction. Trans Am Ophthalmol Soc 1964; 62: 140–52.
- Mulhern M, Barry P, Condon P. A case of Descemet's membrane detachment during phacoemulsification surgery [letter]. Br J Ophthalmol 1996; 80: 185–6.
- 6. Anderson CJ. Gonioscopy in no-stitch cataract incisions. J Cataract Refract Surg 1993; 19: 620–1.
- 7. Monroe WM. Gonioscopy after cataract extraction. South Med J 1971; 64: 1122– 4.
- Minkovitz JB, Schrenk LC, Pepose JS. Spontaneous resolution of an extensive detachment of Descemet's membrane following phacoemulsification. Arch Ophthalmol 1994;112: 551–2.
- 9. Morrison LK, Talley TW, Waltman SR. Spontaneous detachment of Descemet's membrane. Case report and literature review. Cornea 1989; 8: 303–5.

- 10. Bergsma DR Jr, McCaa CS. Extensive detachment of Descemet membrane after holmium laser sclerostomy. Ophthalmology 1996; 103: 678–80.
- 11. Macsai MS. Total detachment of Descemet's membrane after small-incision cataract extraction [letter]. Am J Ophthalmol 1992; 114: 365–6.
- 12. AI-Mezaine HS. Descemet's membrane detachment after cataract extraction surgery. Int Ophthalmol.2010; 30: 391– 6.
- 13. Potter J, Zalatimo N. Descemet's membrane detachment after cataract extraction. Optometry.2005; 76: 720–4.
- 14. Mahmood MA, Teichmann KD, Tomey KF, al-Rashed D. Detachment of Descemet's membrane. J Cataract Refract Surg.1998; 24: 827–33.
- Assia EI, Levkovich-Verbin H, Blumenthal M. Management of Descemet's membrane detachment. J Cataract Refract Surg. 1995; 21:714 –7.
- 16. Bergsma DR Jr, McCaa CS. Extensive detachment of Descemet membrane after holmium laser sclerostomy. Ophthalmology 1996; 103: 678–80.
- 17. Macsai MS. Total detachment of Descemet's membrane after small-incision cataract extraction [letter]. Am J Ophthalmol.1992; 114: 365–6.
- 18. Marcon AS, Rapuano CJ, Jones MR, et al. Descemet's membrane detachment after cataract surgery: management and outcome. Ophthalmology.2002; 109: 2325–30.
- 19. Chaurasia S, Ramappa M, Garg P. Outcomes of air descemetopexy for Descemet membrane detachment after cataract surgery. J Cataract Refract Surg.2012; 38:1134 –9.
- 20. Kim IS, Shin JC, Im CY, Kim EK. Three cases of Descemet's membrane detachment after cataract Surgery. Yonsei Med J.2005; 46: 719–23.
- 21. Mackool RJ, Holtz SJ. Descemet membrane detachment. Arch Ophthalmol.1977; 95: 459–63.

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# Targeted Screening for Awareness Creation and Glaucoma Case Finding in South Indian Community

Praveen Subudhi<sup>1</sup>, Dipankar Datta<sup>2</sup>, P. Namperumalsamy<sup>3</sup>

Author Affiliation: <sup>1</sup>Consultant,<sup>2</sup>Chief Consultant, <sup>3</sup>Chairman Emeritus, Ruby Eye Hospital, Sushruta Nagar, Govinda Vihar, Berhampur, Odisha 760009

# Abstract

Purpose: To determine the effectiveness of targeted screening for glaucoma case finding. Study setting: Aravind Eye Hospital, Theni. Materials and Methods: From hospital database 1493 case sheets of glaucoma patients were retrieved who were informed by postcards (1007), telephone (243) and door to door verbal communication by field workers (253) to come to the base hospital along with their 1<sup>st</sup> degree relatives for glaucoma evaluation on a particular day. Visual acuity, tension applanation, detailed anterior segment evaluation by slit lamp examination, pachymetry, gonioscopy for relevant patients and optic nerve head evaluation by + 90D slit lamp bio-microscopy were done. New cases of glaucoma were established by IOP, optic nerve head damage and Humphrey visual field (HFA 24-2) analysis. Results: 520 patients (New cases/established glaucoma cases - 171/349) attended screening in the hospital in a single day. 150 were first degree relatives. 27 (18% of 1st degree relatives) new cases were detected among 1st degree relatives. Overall 48 cases (new/review 27/21) were called at later date for HFA 24-2, 45 cases (18/27) were advised cataract surgery, 10 cases (0/10) were advised combined surgery and 10 patients (6/4) were advised 1064 Nd: YAG laser peripheral iridotomy. Conclusion: Glaucoma screening and awareness creation program in rural setting is very essential as the major Indian population resides here. This methodology of screening is unique as it provides tertiary care expertise at the doorsteps of rural population and provides awareness of generic nature of disease.

**Keywords**: Intraocular Pressure; Humphrey's Visual Field Analysis; Field Workers; Vision Centers; Nd: YAG lasers.

# Introduction

Iaucoma, the second most common cause of Jblindness after cataract, causes irreversible loss of vision [1,2,3]. According to World Health Organization, in 2002, 12.3% (4.6 million) of the 37 million blind people worldwide could be attributed to glaucoma. It is predicted that prevalence of glaucoma will exponentially increase to 79.6 million with an estimated 11 million blind in both eyes from glaucoma by 2020 [2]. India has the largest population of persons with blindness and vision impairment with an estimated 18.7 million people blind in India which may increase to 31.6 million by 2020 [4]. Aravind comprehensive eye study done in rural population of southern India reported to have a prevalence of 2.6% for all types of glaucoma with primary open angle glaucoma as the leading cause followed by angle closure glaucoma and pseudoexfoliation glaucoma [5].

Glaucoma is an eye condition which is treatable and where early interventions can alter or halt the progression of the disease to blindness. However, screening for glaucoma is not as well established as for cataract or refractive errors primarily because of the large differential in prevalence and the relative difficulty to establish a diagnosis (more tests and more equipment in the screening setting) compared with cataracts and refractive errors. This has led to recommendations to use case finding as the first line

> Reprint Request: Praveen Subudhi, Consultant, Ruby Eye Hospital, Sushruta Nagar, Govinda Vihar, Berhampur, Odisha 760009

> > E-mail: subudhipraveen@gmail.com

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for identification of persons with glaucoma in India [6].

Aravind Eye Hospital, Theni, established in 1985 provides eye care services to a 1.1 million rural population base in Theni, a remote district of Tamil Nadu. We designed a high risk targeted screening approach focused on the first degree relatives of persons with glaucoma (index cases of glaucoma were identified and confirmed at the outpatient department of the hospital) considering that several studies have reported a heritable basis for glaucoma [7, 8, 9, 10]. This manuscript reports on the results of this high risk targeted screening strategy to identify persons with glaucoma. Here we have tried to provide tertiary health care benefits to rural people by utilizing primary health care workers.

# Methods

This retrospective analysis had approval of the ethics committee of Aravind eye hospital and post graduate institute and followed all tenets of the Declaration of Helsinki.

With this background a one day glaucoma screening camp was conducted at the base hospital of Aravind Eye Hospital Theni to monitor known glaucoma patients and to screen their 1<sup>st</sup> degree relatives. 1493 case records of indexed glaucoma patients were retrieved from medical records department using ICD code. Of the indexed cases 1160 (77.7%) case records were from hospital database and 333(22.3%) case records were from vision center database. These vision centers are primary health care centers providing eye care to 50,000 rural populations. There are 9 vision centers situated covering all directions of the district which are well connected with satellite and internet communication with base hospital. A unique methodology of publicity was carried out to inform and educate patients to attend glaucoma screening along with their family members.

Of the 1493 patients, 1007 patients were informed by hand written post cards by our trained paramedics in local language which were sent by post to their respective addresses, 243 patients were informed by phone calls and 254 cases were informed by verbal communication at their doorstep by our field workers. Field workers are ground health staff who are employed in vision centers and take care of outreach activities. Each vision center employs one field worker. Besides mass publicity was done using local cable channels and mike announcements 2 days prior to camp. Glaucoma awareness posters were displayed in various hospitals (Government and Private), vision centers and primary and secondary health care centers.

On the day of camp all patients underwent registration process initially followed by uncorrected visual acuity examination using snellen charts by trained paramedical staff. Intraocular pressures of all the patients were measured by noncontact applanation tonometry followed by preliminary examination of anterior segment by slit lamp. Gonioscopy and pachymetry was done in necessary patients. 3 trained glaucoma experts were invited from our tertiary care center for assessing and diagnosing glaucoma cases. Detailed optic disc evaluation was done with +90D slit lamp biomicroscopy through mydriatic pupils by trained glaucoma experts. Optical coherence tomography 9Stratus OCT, Zeiss meditech) was done in both new and established diagnosed glaucoma cases/ suspects and optic disc evaluation was done using moor-fields regression analysis. Automated perimetry was not part of the protocol. However established glaucoma cases had their Humphreys visual analysis (24-2 and/or 10-2 as indicated) done, field examination for newly diagnosed or glaucoma suspects was performed at a later date. Appropriate management was advised by glaucoma experts followed by counseling by trained counselors. At the end all patients attended a video show depicting and educating the public about glaucoma. Patient flow algorithm is depicted in figure 3.

# Results

520 patients attended the glaucoma screening camp. 349 patients or 67.11% of the total registrants were known glaucoma patients and 171 individuals or 32.88% of the registrants were newly screened.150 individuals / 87.7% of new registrants were 1<sup>st</sup> degree relatives and 21 individuals / 12.3% of new registrants were 2<sup>nd</sup> degree relatives. Mean age group of all the registrants was 61.63 years (range 46-92 years). 69.37% were males and 30.63% were females.

29 cases / 16.9% of new registrants were diagnosed of glaucoma in all forms. 27 cases/ 18% of 1st degree relatives were diagnosed of glaucoma. Mean age group of the 1<sup>st</sup> degree relatives was 51.25 years (range 42-62) Of the 29 cases; 15 cases (51.72%) were primary open angle glaucoma, 5 cases (17.24%) were primary open angle suspects, 6 cases (20.68%) were primary angle closure glaucoma and 3 cases (10.34%) were pseudo-exfoliation glaucoma. 27 newly diagnosed cases were called another day for

full threshold Humphreys visual field analysis 24-2 for confirmation of diagnosis.2 new cases were not advised visual field analysis in view of their advanced cataract. New cases were not advised combined surgery because final intervention is done after analysis of visual field reports at later date.

Overall 10 cases were advised Nd: YAG laser iridotomy of which 6 cases were newly identified cases and the rest 4 were from established glaucoma cases. 10 from the established glaucoma group cases showed remarkable progression in optic nerve head damage (established by OCT) and hence they were advised combined trabeculectomy with cataract surgery and intraocular lens implantation. 18 cases from the new registrants were advised cataract surgery and 27 cases from the established group were advised cataract surgery with intraocular lens implantation through temporal approach as their IOP were well controlled by medical therapy (to salvage superior conjunctiva for trabeculectomy in future). 21 cases from the established group were advised repeat Humphreys visual filed analysis in view of their poor compliance and non-progression of optic nerve head damage in OCT. (Figure 1 shows flow chart of new cases identified and Figure 2 shows flow chart of follow up and management of established glaucoma cases).

Field workers had a pivotal role in the success of this camp; table 1 shows the performance of each of the field worker in their respective fields.

Fig. 1: Flow chart showing total number of new registrants, newly identified cases and overall management plan.



Fig. 2: Flow chart Showing Distribution Of Types Of Glaucoma In Established Glaucoma Patients And Advice Advocated By Trained Glaucoma Experts



POAG:- Primary Open Angle Glaucoma, POAG Suspect:- Primary Open Angle Glaucoma Suspect, PACG:- Primary Angle Closure Glaucoma, PXG:- Pseudoexfoliation Glaucoma, IOL:- Cataract surgery with intraocular lens insertion, Combined surgery:- Combined Trabeculectomy with intraocular lens insertion, YAG PI:- Peripheral iridectomy with Nd:YAG laser, HFA:- Humphreys visual field analysis.

	Door to door visited	No of New Glaucoma
Vision Center Name	by field workers	cases identified
Cumbum	60	3
Ambasamudram	20	0
Andipatty	37	1
Bodi	35	1
Chinnamanur	32	2
Periyakulam	25	1
Kadamalaikundu	10	0
Thevaram	15	0
Batlagundu	20	2
Total	254	10

Table 1: Showing the role of individual field workers of each of the vision centers in detecting new glaucoma cases.

# Discussion

Glaucoma as a cause of blindness is of public health significance which needs to be addressed proactively and seriously. In comparison to cataract there is very limited data available for glaucoma. Due to its silent progression most of the people come in late stages where extensive damage to optic nerve head has already been done. Previous studies done in south India estimated 11.2 million people to have glaucoma aged 40 years and above, of which 6.48 million have primary open angle glaucoma and 2.54 million to have primary angle closure glaucoma [5]. Robin et al [10] report from Aravind Comprehensive Eye study that three fourths of rural population aged 40 years and above required eye care services. Lee et al [11] found that knowledge and perceptional barriers were the major obstacles in follow up of the

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patients under study amounting to 40% in south India. If this is the pattern of follow ups in known glaucoma patients then situation will be still worse in cases without established diseases [12]. Rural urban disparities in eye care services have further compounded the problem [12, 13, 14]. Only 25% of ophthalmologists practice in rural India. In India ophthalmologist to population ratio is 1:100,000 which is still worse in rural India.

Moreover women have poor access to eye care especially in rural India. Our study also showed poor turnout of females in comparison to males. Thapa et al [15] of Tilganga eye care had conducted similar glaucoma screening camps and awareness creation week for consecutive four years and there was drastic increase in patient attendance but only lacunae was major beneficiaries were from urban region nearly 90% whereas only 10% were from rural region.

Besides glaucoma being a major public health problem it also runs in families. First degree relatives usually have a very high risk of developing glaucoma. Francielli vegini et al [17] of Sao Paolo University did a study among first degree relatives of known glaucoma cases attending glaucoma clinic. They examined 101 first degree relatives and found 10.9% had prior diagnosis of glaucoma and 9.8% were newly diagnosed as glaucoma or glaucoma suspects. In our study we examined 150 first degree relatives, 27 cases /18% were newly diagnosed of glaucoma. The glaucoma inheritance study in Tasmania and other states of Australia examined 442 individuals with strong family history of glaucoma (not only first degree relatives of glaucoma),13% had prior diagnosis of glaucoma or glaucoma suspect status and 16% were newly diagnosed [18]. Even Baltimore eye survey and Barbados eye study showed that people with positive family history of primary open angle glaucoma are more vulnerable to develop glaucoma [19, 20]. Randall et al published a study in which 86 relatives of POAG were examined; showed prevalence of disease in relatives was 30% [21].

# Conclusion

The methodology adopted here utilizing paramedics and field workers to detect new cases among first and second degree relatives is very essential and helpful in preventing progression of glaucoma to blindness. As majority of Indian population resides in rural areas with inadequate infrastructure and poor awareness, there is increasing role of local health workers to survey and create awareness of generic nature of the disease.

# References

- Resnikoff S, Pascolini D, Etya'ale D, Kocur I, Pararajasegaram R, Pokharel GP, Mariotti SP, Global Data on visual impairment. Bull World Health Organ. 2004 Nov; 82(11): 844-51. [Pubmed].
- 2. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol.2006 Mar; 90(3): 253-4. [Pubmed].
- The World Bank Group Nepal Transport Sector. 2007. http://go.worldbank.org Accessed May 15, 2007.
- 4. Hatt S, Wormald R, Burr J. Screening for prevention of optic nerve damage due to chronic open angle glaucoma. *Cochrane Database Syst Rev*: CD006129. 2006 Oct 18. [PubMed].
- Tielsch JM. The epidemiology of primary openangle glaucoma. *Ophthalmol Clin North Am.* 1991; 4: 649–657.
- Tielsch JM, Katz J, Singh K, Quigley HA, Gottsch JD, Javitt J, Sommer A. A population-based evaluation of glaucoma screening: the Baltimore Eye Survey. Am J Epidemiol. 1991; 134: 1102–1110. [PubMed].
- Wilson MR, Khanna S. The value of different screening techniques for glaucoma. *Curr Opin Ophthalmol.* 1994; 5: 69–75. doi: 10.1097/ 00055735-199404000-00011. [PubMed] [Cross Ref]
- Fraser S, Bunce C, Wormald R, Brunner E. Deprivation and late presentation of glaucoma: case-control study. *BMJ*. 2001; **322**: 639–643. doi: 10.1136/bmj.322.7287.639. [PMC free article] [PubMed] [Cross Ref]
- Saw SM, Gazzard G, Friedman D, Foster PJ, Devereux JG, Wong ML, Seah S. Awareness of glaucoma, and health beliefs of patients suffering primary acute angle closure. *Br J Ophthalmol.* 2003; 87: 446–449. doi: 10.1136/bjo.87.4.446. [PMC free article] [PubMed] [Cross Ref]
- Utilisation of eye care services in rural south India: the Aravind Comprehensive Eye Survey P K Nirmalan, [1, 2] J Katz, [3, 4] A L Robin, [3, 5] R Krishnadas, [2] R Ramakrishnan, [2] R D Thulasiraj, [1, 2] and J.Tielsch [3, 4] Br J Ophthalmol. 2004 October; 88(10): 1237–1241.

- Lee DJ, GÃ<sup>3</sup>mez-MarÃ-n O, Lam BL, Zheng DD, Caban A. Visual impairment and morbidity in community-residing adults: the national health interview survey 1986-1996. Ophthalmic Epidemiol. 2005 Feb; 12(1): 13-7.
- 12. Krishnaiah S, Kovai V, Srinivas M, Shamanna BR, Rao GN, Thomas R. Awareness of Glaucoma in the rural population of Southern India. *Ind J. Ophthalmol.*2005; 53: 205–8.
- 13. Urbanization, inequality and economic growth: Evidence from Indian states Preliminary findings of a paper prepared for the WDR 2009 Massimiliano Calì *Overseas Development Institute* November 2007.
- 14. Rural-Urban distribution *Census of India: Census Data 2001: India at a glance: Rural-Urban Distribution.* Office of the Registrar General and Census Commissioner, India.
- A novel approach to glaucoma screening and education in Nepal Suman S Thapa, Kurt H Kelley, Ger V Rens, Indira Paudyal, and Lan Chang BMC Ophthalmol. 2008; 8: 21. Published online 2008 October 26. doi: 10.1186/1471-2415-8-21.
- 16. Hatt S, Wormald R, Burr J. Screening for prevention of optic nerve damage due to chronic

open angle glaucoma. *Cochrane Database Syst Rev*.CD006129. 2006 Oct 18. [PubMed].

- 17. Prevalence of Open Angle Glaucoma in Accompanying First Degree Relatives of Patients with Glaucoma Franciele Vegini, Natanael Figueiroa Filho, Raphael Furlan Lenci, Diogo Garcia Neto, and Remo Susanna Junior, Clinics. 2008 June; 63(3): 329–332. PMCID: PMC2664248.
- McNaught AI, Allen JG, Healey DL, Coote MA. Accuracy and Implications of a reported family history of glaucoma: experience from the glaucoma inheritance study in Tasmania. *Archives of Ophthalmology*. 2000; 118: 900–4. [PubMed].
- 19. Tielsch JM, Katz J, Sommer A. Family history and the risk of primary open angle glaucoma. The Baltimore Eye Survey. *Arch Ophthalmol.* 1994; 112: 69–73. [PubMed].
- 20. Nemesure B, Leske MC, He Q, Mendell N. Analyses of reported family history of glaucoma: a preliminary investigation. The Barbados Eye Study Group. *Ophthalmic Epidemiol.* 1996; 3: 135– 41. [PubMed].
- 21. Nguyen RL, Raja SC, Traboulsi EI. Screening relatives of patients with familial chronic open angle glaucoma. *Ophthalmology*. 2000; 107: 1294–7. [PubMed].

# Disease and Drug Related Ocular Complications of Tuberculosis

# Sonya Puri<sup>1</sup>, Madan Deshpande<sup>2</sup>, Sagarika Patyal<sup>3</sup>, Poninder Kumar<sup>4</sup>, Vijay Mathur<sup>4</sup>

Author Affiliation: <sup>1</sup>Associate Professor,<sup>3</sup>Professor and Head,<sup>4</sup>Professor, Base Hospital and Army College of Medical Sciences, Delhi Cantt. <sup>2</sup>Chief Medical Director, HV Desai Eye Hospital, Pune.

# Abstract

Introduction: Tuberculosis continues to be a major health problem in India. Ocular tuberculosis can involve any part of the eye. It can occur with or without active systemic tuberculosis. Among the antitubercular drugs, ethambutol is most commonly associated with ocular toxicity. Aim: This study was done to find the incidence of ocular tuberculosis among patients with active systemic tuberculosis and to study the ocular toxicity of antitubercular drugs. Methods and Materials: 152 patients of newly diagnosed active systemic tuberculosis (pulmonary and extra-pulmonary) were screened for ocular tuberculosis. A detailed general and ocular examination including visual acuity, slit lamp bio-microscopy, indirect ophthalmoscopy, color vision was done. Specialized investigations including Fundus fluoresce in angiography, Visual fields and VEP were done where indicated. Montoux test, ESR, AFB, Quantiferon TB gold test were done. They were followed up at 2mths and 6mths to look for any ocular toxicity of antitubercular drugs. Results: 9 cases (5.9%) were detected to have ocular tuberculosis, of these 5 cases had pulmonary and four cases extra-pulmonary tuberculosis. Of the 52 cases on treatment with ethambutol 2 cases (3.8%) were detected to have ethambutol toxicity. They had constriction of visual fields which recovered 2 months after discontinuing the drug. Conclusion: The incidence of ocular tuberculosis in a study of 152 cases of active systemic tuberculosis was 3.28 % and the incidence of ocular toxicity was 3.8 % among patients receiving ethambutol. The ocular toxicity was reversible on discontinuing the drug.

**Keywords:** Tuberculosis; Choroiditis; Periviascultitis; Optic atrophy; Papilloedema; Ethambutol Toxicity.

# Introduction

Tuberculosis is a specific communicable disease caused by Mycobacterium tuberculosis. In India it is a major public Health problem, the incidence being 171 per 1,00,000 population [1]. The prevalence of TB infection (latent infection) in India is about 40% [2]. There is a higher incidence in HIV positive patients.

Ocular tuberculosis can involve all parts of the eye. It normally occurs as part of a post primary infection due to direct haematogenous spread or by hypersensitivity responses [3].

Ocular toxicity has been reported with ethambutol in the form of optic neuritis [4]. It is dose related and usually reversible on cessation of treatment [5]. Till date two types have been reported.

- (a) An axial type which manifests with loss of central vision associated with central scotoma and color vision defects.
- (b) A paraxial type which manifests peripheral visual field construction [6].

Ocular toxicity is usually dose-related [7].

INH rarely causes optic neurits and atrophy [8].

# Methods and Materials

152 patients with active systematic tuberculosis (pulmonary and extra-pulmonary) were examined

Reprint Request: Col. Poninder Kumar, Professor of Ophthalmology, Army College of Medical Sciences, Brar Square, Near Base Hospital Delhi Cantt., New Delhi – 110010 E-mail: poninder@hotmail.com

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tubercular meningitis.

in a tertiary hospital. The patients were predominantly males and their ages ranged from 11 ½–70 years.

They were divided into 2 groups: those with pulmonary and those with extra-pulmonary tuberculosis. Each group was further divided into those on ethambutol and those no on ethambutol.

A detailed general, systematic and ocular examination was done. The diagnosis of tuberculosis and confirmed by a physician by clinical examination and investigations. Investigations included blood ESR, Hb, TLC, DLC, Sputum-acid fast bacilli, Montoux test, X-Ray chest and Quantiferon T.B. gold test. Specialized investigations



evidence suggestive of ethambutol toxicity.

were done in relevant cases like CSF studies in

Ocular examination was carried out before starting

ATT, after 2mths and 6mths. They were asked to report

immediately in case of any visual disturbance. History

of any ocular complains was taken. Detailed ocular examination included visual acuity for distance and

near, color vision, Amsler, anterior segment-slit lamp

bio-microscopy, Fundus under full mydriasis with

binocular indirect opthalmoscope, Fields - Automated

perimetry with Humphery Field Analyzer (24-2 and

60-2). VEP was done in patients who showed



Fig. 3: Subhyaloidhaemorrhage with perivasculitis



Results

A total of 152 cases were studied of these 52 cases (34.2%) were on treatment with ethambutol and 100 cases (65.8%) were not. In the pulmonary tuberculosis group the patients were 115 (75.65%) males and 22 (14.47%) females. Table 1 shows the distribution of cases studied. 17 patients (11.2%) from the ethambutol group and 20 patients (13.1%) from

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Fig. 1: Choroidal tubercle in macula

the non- ethambutol group were sputum positive for AFB. Table 2 shows the distribution of sputum positive cases.

Table 3 shows the incidence of ocular tuberculosis. In sputum AFB+ it is 0.65% and in sputum AFB – patients and in extra-pulmonary tuberculosis patients it is 2.63%. The overall incidence in all groups is 5.9%. Choroiditis was the commonest, it occurred in 3 cases (1.95%) followed by perivasculitis (2 cases), optic atrophy (2 cases) and papilloedema (2 cases). Only one patient had anterior segment involvement in the form of irises. All patients with pulmonary tuberculosis had radiological evidence of tuberculosis, Montoux test more than 15 mm. All patients except one had high ESR. All patients with extra-pulmonary tuberculosis had raised ESR, positive Montoux (more than 15mm).

Out of 52 patients treating with ethambutol, 2 cases showed ocular toxicity (3.8%) Table 4. Both had constriction of peripheral fields. One case developed symptoms 6 days after starting ethambutol and one case after 6 months. Both cases recovered in 2 months after discontinuing the drug and had complete reversal of the field loss.

### Table 1: Treatment Groups

	Pulmonary		Extrapulmonary		Total
	♂Male	♀ Female	් Male	♀ Female	
Ethombutol	42	6	2	2	52
Ethanibutor	27.6%	3.9%	1.3%	1.3%	34.2%
Non Ethombutal	73	16	4	7	100
Non-Ethambutor	48%	10.5%	2.6%	4.6%	65.8%
Total	115	22	6	9	152
	75.65%	114.47%	3.9%	5.9%	

# Table 2: Sputum positivity for AFB

Treatment Group	Number of cases	AFB+	AFB
Ethambutol	48	17	13
Ethambutor	31.5%	11.2%	20.4%
Non-Ethambutol	89	20	69
	58.5%	13.1%	45.3%
Total	137	37	100
Total	90%	24.3%	65.2%

### Table 3: Incidence of Ocular Tuberculosis

	Pulmonary		Extrapulmonary	Total
	AFB+	AFB-	Extrapalitionary	
Number of Cases	1	4	4	9
	0.65%	2.63%	2.63%	5.9%

Table 4:	Incidence	of	Ocular	Toxicity to	ATT
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Treatment Group	Number of cases	Cases Showing Ocular Toxicity	Percentage
Ethambutol	52	2	3.8%
Non-Ethambutol	100	0	0%

## Discussion

In the present study, 152 cases of systemic tuberculosis (pulmonary and extra-pumImonary) were evaluated for ocular tuberculosis and ocular toxicity to anti tuberculosis drugs.

9 patients (5.9%) were found to have ocular findings known to be associated with tuberculosis,

of these 5 had pulmonary and 4 had extra-pulmonary tuberculosis.

Donahue (1940–1966) [9], Lal and Gupta (1985) [10], Biswas (85–86) [11], BouzaEMerino (1997) [12], and BeareNAKublin et al [13], studied 10254, 3064, 1005, 100 and 109 cases respectively and found an incidence of ocular tuberculosis of 1.4%, 5.74%, 1.4%, 18% and 2.8% respectively. The high incidence (18%)

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in the study by BouzaEMerino et al is because of coinfection with HIV in 60% of cases.

Of the 9 patients with ocular tuberculosis, 1 patient (0.72%) had Panuveitis, 2 patients (1.45%) had old healed choroiditis, 2 patients (1.45%) had optic perivasculitis, 2 patients (1.45%) had optic atrophy and 2 patients (1.45%) had papilloedema. 1 patient with choroiditis also had a choroidal tubercle. There was no case of orbital, lid, and lacrimal gland, conjunctival or corneal tuberculosis.

In other studies Golden Burg and Fabricant [14] (1930), Donahne (1940-66), Lal and Gupta (1985) and Biswas (1985-86) found an incidence of 0.6%, 0.27% 0.68% and 0 of irises respectively. 2.1%, 0.44%, 0.09% and 1.2% of choroiditis respectively.

Biswas reported a 0.1% incidence of chorodial tubercle. Illingrowth and Wright [15] (1948) have reported a 60% and 5.5% of chorodial tubercle with miliary tuberculosis and tuberculosis meningitis respectively.

All the 9 patients with ocular tuberculosis had a positive Montoux test and had a high ESR. Only 1 patient of the 9 patients of ocular tuberculosis was sputum positive for acid fast bacilli, ocular findings in this case were old healed choroditis.

Of the 52 patients on treatment with ehtambutol, only 2 patients (3.8%) were detected to have ethambutol toxicity. Both the patients had peripheral constriction of visual fields. 1 patient had a delayed VEP. One of the patients developed symptoms with 6 days of treatment and the other 6 months after starting antitubercular treatment. Both recovered 2 months after discontinuing ethambutol. No patient had color defect, central fields defect, papillary abnormality or papillitis. No toxicity was noted to other anti tubercular drugs.

The remaining 100 patients who were not on ethambutol were taken as a control. None of them showed constriction of fields or any other changes suggestive of ehtmabutol toxicity. Previous studies have shown ethambutol toxicity ranging from 0.5 – 6.6% as compared to our study showing 3.8%.

The limitations of this study are that the sample size is small compared to studies by Donahne (1940-66), Lal, Gupta (1985) and Biswas (1985-86). However it is larger than studies by BouzaEMerino (1997) and BeareNAKublin et al (2002). HIV status of the patients was not taken into account in this study.

## References

- 1. World Health Organization, Global Tuberculosis Report. Available from http://data.worldbank.org/ indicator/SH.TBS.INCD/countries
- Central TB Division, Directorate General of Health Services, New Delhi. Tuberculosis Burden. In: TB India 2011, Revised National TB Control Program, Annual Status Report. p. 5-10. Available from http://planningcommission.nic. in/reports/genrep/health/RNTCP\_2011.pdf
- 3. Rosen DH, Spalton DJ, Graham EM, Intraocular tuberculosis, Eye, 1990, 4, 486 492.
- 4. Citron KM, Thomas GO. Ocular toxicity from ethambutol. Thorax 1986; 41: 737-739.
- 5. Chatterjee VKK, Buchanan DR, Freidman A, Green M, Ocular toxicity following ethambutol in standard dosage. Br.J. Dis Chest, 1986: 80: 288-91.
- 6. Barron GJ, Tepper L, Iovine G, Ocular toxicity from ethambutol, Am J Ophthamol 1974, 74: 256 260.
- Harcombe A, Kinnear W, Britton J, Macfarlane J, Ocular toxicity of ethambutol, Resp. Med 1991, 85: 151-3.
- Goodman LS and Gilman A. Pharmacological Basis of Therapeutics, 7<sup>th</sup> edition, New York, MacMillan.1985: 1209.
- Donahne HC, Ophthalmic experience in a tuberculosis Sanitorium, Am J Ophthalmol 1967; 64: 742-8.
- Lal BB, Gupta RK, Ocular Involvement in tuberculosis A Clinical Study Afro-Asian J Ophthalmol IV, Dec 85-2.
- 11. Biswas J, Ocular Morbidity in patients with active pulmonary tuberculosis, Insight 88, Vol V, 3-5.
- 12. Bouza EMerino PMunoz PSanchez-Carrillo CYanez JCortes C Ocular tuberculosis: a prospective study in a general hospital. *Medicine* (*Baltimore*) 1997; 76: 53- 61.
- 13. Beare NAKublin JGLewis DK et al. Ocular disease in patients with tuberculosis and HIV presenting with fever in Africa. *Br J Ophthalmol* 2002; 86: 1076–1079.
- 14. Helm CJ, Holland GN, Ocular Tuberculosis, Survey Ophthalmol, 1993; 229 – 256.
- 15. Illingrowth RS, Wright T, Tubercules of the choroid. Br. Med J. 1948; 2: 364–368.

# **Orbital Cellulitis: A Rare Case Report**

# Roopa Naik<sup>1</sup>, Nikita shah<sup>2</sup>, Jaineel Gandhi<sup>2</sup>

Author Affiliation <sup>1</sup>Professor and Head of Department Dept of ophthalmology, <sup>2</sup>Resident, Padmashree Dr. Vithalrao Vikhe Patil Medical College & Hospital, Near Govt. Milk Dairy, Vilad Ghat, Ahmednagar, Maharashtra.

# Abstract

Orbital cellulitis is common due to the infective etiologies. It can also rarely occur due to benign and malignant lesions. The differential diagnoses of orbital cellulites include mucormycosis (fungal infection), sarcoidosis and dysthyroid exophthalmos, neoplasia with inflammation, lymphoma, and glioma of optic nerve, pseudotumour and so on.

We here present a case of orbital cellulitis in 70 year female who was referred to us. She also complained of gradual, painless, progressive swelling in the left parietal region since 1 year. The soft tissue was also seen to extend in the left orbit as well as in left infra temporal fossa. The soft tissue in the orbit was extraconal and was displacing the eyeball infero-medially. No obvious invasion of the optic nerve or sclera was seen.

**Keywords:** Orbital Cellulites; Osteogenic Sarcoma; Parietal Bone; Orbit; Infra-Temporal Fosse; Extraconal Lesion.

## **Case Report**

A 70 yr female came to the ophthalmology department with complaints of gradual, painless forward bulging of left eye along with restricted ocular movements in the same eye since 3 months. She also complained of painless swelling in the temporal region which was gradual and slowly progressing past 1 year. But now the swelling had progressed suddenly to approximately 5\*3cm.The swelling was firm in consistency. There was no local rise of temperature. The overlying skin was intact. The swelling was tender. The patient was put on the treatment for orbital cellulites past 2 months for which she did not respond and so she was referred to us. She had no history of trauma, diplopia, sinusitis, any ocular surgery in the past. No history of diabetes mellitus, hypertension, tuberculosis, allergy etc.

She was afebrile, conscious, oriented of time, place and person. Pulse was 70 beats per minute BP was 130/80mmhg. Respiratory rate was 20cycles. She had signs of pallor. She had no signs of clubbing, icterus, lymphadenopathy, cyanosis and oedema.

B-scan was done. It showed extraconal soft tissue on lateral compartment displacing the eyeball medially. The lateral rectus appears separate. No invasion of the eyeball was noted. Incidentally noted was soft tissue in the left temporal region. It showed increased vascularity. The patient was not able to move the eye ball laterally.

CT scan was ordered for further evaluation of swelling over the parietal bone. Contrast enhanced CT scan was done. It showed erosive lesion in the left parietal bone. The lesion had wide zone of transition. The margins were not well appreciated. The cranial part showed linear onion peel like periosteal reaction. There was associated well defined soft tissue component in the intracranial as well as extra cranial compartments. The intracranial component of the soft tissue was seen to cause mass effect over the

> Reprint Request: Dr. Roopa Naik Department Of Ophthalmology, Padmashree Dr. Vithalrao Vikhe Patil Medical College and Hospital, Vilad ghat, Ahmednagar, India. Pin- 414111. E-mail: roopa1704@gmail.com

On Local Examination		
	O D	0 8
Visual acuity	No PL	Fcat 1m
Head Posture	Ν	Ν
Facial Symmetry	Bilaterally Symmetry	-
Occular Movements	Phthisis Bulb	Restricted in all Directions
Eyebrow, Eyelid, Eyelash	Ν	Ν
Conunctiva	Atrophed	Chemosed
Cornea	Mated	Clear
A/C	-	N Content Depth
Iris	-	N Colour Pattern
Pupil	-	Sluggishly Reacting to light
Lens	-	Nuclear
Іор	Ν	Ν

### Investigations

HB	8gm%
WBC	12000ells
	per cubic millimeter
	(cmm)
Neutrophils	75%
LDH	375IU/L
Platelets	100000
	Lakhs/cmm
ESR	30mm/hr
LFT	Ν
RFT	Ν
Alkaline phosphatase	200IU/L

underlying cerebral hemisphere with associated oedema The soft tissue was also seen to extend in the left orbit as well as in left infra temporal fossa. The soft tissue in the orbit was extraconal and was displacing the eyeball infero-medially. No obvious invasion of the optic nerve or sclera was seen .The soft tissue showed moderate heterogeneous contrast enhancement. The CT findings indicated neoplastic aetiology. As patient was 60 yrs strong possibility of osteosarcoma was considered. It was proved on biopsy. Patient denied surgery and hence was referred to radiotherapy department.

Fig. 1: 70 yr old with swelling over left parietal and orbital region with proptosis of left globe.



Fig. 2: Soft tissue extraconal mass displacing the globe inferomedially.



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CECT was done. CT scan showed erosive lesion in the left parietal bone .The lesion had wide zone of transition. The margins were not well appreciated. The cranial part showed linear onion peel like periosteal reaction. There was associated well defined soft tissue component in the intracranial as well as extra cranial compartments. The intracranial component of the soft tissue was seen to cause mass

effect over the underlying cerebral hemisphere with associated oedema. The soft tissue was also seen to extend in the left orbit as well as in left infra temporal fossa. The soft tissue in the orbit was extra-conal and was displacing the eyeball infero-medially. No obvious invasion of the optic nerve or sclera was seen. The soft tissue showed moderate heterogeneous contrast enhancement.



**(B)** 

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Coronal CECT image shows mass in intracranial as well as extra cranial.(A)Mass is displacing left

globe medially and inferiorly.(B) Sagittal images shows mass in intra orbit and extending into infra temporal fossa.



CECT in bone window showing erosive lesion involving left parietal bone.

Chest x-ray and USG abdomen and pelvis were normal.

# Histopathology

USG guided biopsy from the edge of the swelling was taken. On histopathology report they confirmed with the diagnosis of osteosarcoma.

Histologically osteosarcoma shows malignant osteoblast which shows osteoid production and has osteoblastic, chondroblastic and fibroblastic subtypes.

# Treatment

She was treated for orbital cellulitis. The patient was referred to the higher centre for chemotherapy as the mass was unresectable .Osteosarcoma is often treated with a combination of therapies that can include surgery, chemotherapy and radiation therapy. Most patients with high grade tumours receive about three months of chemotherapy, known as neo-adjuvant therapy, before surgery. Now the follow up of the patient post therapy is awaited.

# Discussion

Orbital cellulites are common due to the infective aetiologies. It can also rarely occur due to benign and malignant lesions. The differential diagnoses of orbital cellulites include mucormycosis (fungal infection), sarcoidosis, dysthyroid exophthalmos, neoplasia with inflammation, lymphoma and glioma of optic nerve, pseudotumour and so on.

So based on histopathology report here is a rare case of osteosarcoma of parietal bone causing orbital cellulites. Osteosarcoma is more common in boys than girls [1]. It has predilection for the metaphyseal region of the long bones Any bone in the body can be affected, but the most common sites are the arms or legs, particularly around the knee joint [2]. The incidence of primary osteogenic sarcomas of the skull is about 1 to 2% of all skull tumors [3].

There are several different types of osteosarcoma, such as parosteal [4], periosteal telangiectatic and small cell osteosarcoma.

Primary osteosarcoma arises from the metaphysis of the long bones usually and approx. 10 % are located in the flat bone mainly pelvis and 1-2 % ribs, sternum and clavicle [5].

There are many corresponding studies that reported the rarity of osteosarcoma of the skull. For

instance, Nora et al, reported that 21 of 1,000 osteosarcoma cases had tumor in the skull, and only 14 out of 21 cases (1.4%) were de novo tumor. Huvos et al, reported that only 10 out of 1,200 osteosarcoma cases (0.8%) over a 60 year period were de novo osteosarcoma of the skull.

Biochemical studies are usually normal, except for elevations in ALP, LDH, and ESR. As the osteosarcoma of the skull is very rare it is difficult to arrive at definitive treatment plan.

Orbital cellulitis due to parietal bone osteosarcoma is an example of a rare, potentially fatal condition, and an early diagnosis is often a challenge. So we should consider orbital cellulitis due to parietal bone osteosarcoma in cases. It is a rare presentation with orbital cellulitis still on should keep in mind while investigating a case of orbital cellulitis.

Osteosarcoma treatment has progressed greatly over the past thirty years. The standard treatment of osteosarcoma consists of the combination of chemotherapy and surgery, and in some cases radiation [6].

If a cure is to be achieved, surgical removal of all the tumor tissue at any site should always be attempted. Complete surgery is the treatment of choice for osteosarcoma [7]. In selected cases, however, radiotherapy has proven helpful [8].

## Conclusion

This case describes a patient with osteosarcoma invading into the orbit, mimicking orbital cellulites. The case underscores the importance of considering alternative diagnoses when patients with orbital cellulites do not respond to antibiotic treatment. We here by conclude that osteosarcoma of skull is very rare entity and needed early diagnosis and treatment due to its aggressive nature and poor prognosis. Biopsy and histological confirmation is needed for suspected sites of metastatic disease. Chemotherapy can successfully eradicate primary deposits if initiated at a time when disease burden is low. The prognosis depends mainly on the degree of intracranial involvement at the time of the diagnosis is rather than the mode of therapy.

### References

- Huvos AG. Bone Tumours, Diagnosis, Treatment and Prognosis. Philadelphia: WB Saunders. 1991; pp 192-193.
- Skubitz KM, D'Adamo D. Sarcoma. Mayo Clin Proc.2007; pp1409-1432.
- L.Mascarenhas, A. Peteiro, C. A. Ribeiro, Z. Magalhs9, H. Romì; F. Magalhs9, A. M. Reis, J. Resende Pereira, M. Honavar, M. Resende, A.Rocha Vaz, Skull osteosarcoma: illustrated review, Acta Neurochirurgica, Volume 146, Issue 11, Nov 2004, pp 1235 – 1239.
- R Kumar, RP Moser Jr, JE Madewell and J Edeiken ,Parosteal osteogenic sarcoma arising in cranial bones: clinical and radiologic features in eight patients,American Journal of Roentgenology, 1990 Vol 155, pp113-117.
- Mirra JM, Gold RH, Picci P. Osseous tumours of intramedullary origin. In: Mirra JM, editor. Bone Tumors: Clinical, Radiological, and Pathological Correlations. Philadelphia: Lea and Febiger; 1989. pp.143–438.
- 6. Fukunaga, Low-grade central osteosarcomaof the skull. Pathol Res Pract.2005; 201(2): 131-5.
- 7. Primary osteogenic sarcoma of skull, Bikash Bose Surg Neurol; 58 (3-4): 234-9; discussion 239-40.
- Sunderasan N, Huvog AG, Rosen G. Combined modality treatment of osteogenic sarcoma of the skull. J Neurosurg. 1980; 63: 562-567.

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# Retinopathy Associated with Cone Dystrophy

Bijnya B. Panda<sup>1</sup>, Anusha Venkataraman<sup>1</sup>

Author Affiliation: Department of ophthalmology, All India Institute of Medical Sciences, Bhubaneswar, India.

# Abstract

Progressive cone dystrophies represent a heterogeneous group of diseases with onset in later teens and adult age diagnosed by an abnormal photopic electroretinographic response and a normal or near-normal rod isolated response. We report the case of a young male with typical characteristics of cone dystrophy and a brief description about the inheritance pattern and pathology of the disease.Progressive cone dystrophies represent a heterogeneous group of diseases with onset in later teens and adult age diagnosed by an abnormal photopic electroretinographic response and a normal or near-normal rod isolated response. We report the case of a young male with typical characteristics of cone dystrophy and a brief description about the inheritance pattern and pathology of the disease.

# Introduction

A cone dystrophy is a hereditary ocular disorder characterized by the loss of cone cells, the photoreceptors responsible for both central and colour vision [1, 2]. The most common symptoms of vision loss are: (age-range, from the late teens to the sixties), sensitivity to bright lights, and poor colour vision. Fluorescein angiography (FA) and Optical coherence tomography are useful adjuncts in the workup of someone suspected to have cone dystrophy, as it may detect early changes in the retina that are too subtle to be seen by ophthalmoscope. This pictorial shows the typical FA and OCT findings in a patient who was diagnosed to have cone dystrophy.

# Discussion

This 48 year old male presented with complaints of gradual decrease in vision of four years duration. His BCVA was 20/1200 OD and 20/600 OS with a near vision of N36 OU. Colour vision was abnormal. Colour fundus photo showed the presence of a central hyperpigmented area with surrounding concentric areas of hypo and hyper pigmentation. Fluorescein angiography showed a corresponding area of central hypo-fluorescence with surrounding concentric areas of hyper, hypo and hyper-fluorescence resembling a 'target' sign. Optical coherence tomography revealed

> Reprint Request: : Bijnya B. Panda, Department of ophthalmology, All India Institute of Medical Sciences, Bhubaneswar, India E-mail: bigyan\_panda@yahoo.co.in



Fig. 1: This composite picture shows the colour fundus photos (upper tier) showing bulls eye maculopathy, FFA findings (middle tier) showing target appearance, OCT findings (lower tier) showing foveal atrophy in both eyes.

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disruption of the IS-OS junction and foveal atrophy. ERG confirmed the diagnosis of cone dystrophy. The most common type of macular lesion seen during ophthalmoscopic examination has a bull's-eye appearance and consists of a doughnut-like zone of atrophic pigment epithelium surrounding a central darker area. In another, less frequent form of cone dystrophy there is rather diffuse atrophy of the posterior pole with spotty pigment clumping in the macular area. Rarely, atrophy of the choriocapillaris and larger choroidal vessels is seen in patients at an early stage. Visual field testing in cone dystrophy usually reveals a central scotoma. Photopic Electroretinography (ERG) shows reduced single-flash and flicker response and a normal scotopic ERG. At least one type of autosomal dominant cone-rod dystrophy is caused by mutations in the guanylate cyclase 2D gene (GUCY2D) on chromosome 17. Though there is no cure for Cone dystrophy, certain supplements delay the progression of the disease. The betacarotenoids, lutein and zeaxanthin, have been evidenced to reduce the risk of developing age related macular degeneration (AMD) [3], and may therefore provide similar benefits to Cone dystrophy sufferers. Consuming omega-3 fatty acids (docosahexaenoic

acid and eicosapentaenoic acid) has been correlated with a reduced progression of early AMD, and in conjunction with low glycemic index foods, with reduced progression of advanced AMD,[4] and may therefore delay the progression of cone dystrophy.

## References

- 1. Stephen J. Ryan et al., *Retina*, 3rd ed. (C.V. Mosby, 2001) ISBN 0-323-00804-6.
- 2. Stephen J. Ryan et al., *Retina*, 4th ed. (C.V. Mosby, 2005) ISBN 0-323-02598-6.
- Carpentier S, Knaus M, Suh M (2009). "Associations between lutein, zeaxanthin, and age-related macular degeneration: An overview". Critical reviews in Food Science and Nutrition.49 (4): 313–326.
- Chiu CJ, Klein R, Milton RC, Gensler G, Taylor A (June 2009). "Does eating particular diet alter the risk of age-related macular degeneration in users of the Age-Related Eye Disease Study supplements"? Br J Ophthalmol.93 (9): 1241–6.

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# Novel Association of Blepharophimosis Syndrome with Xanthoma Tuberosum

# Bijnya Panda<sup>1</sup>, Susanta Pujahari<sup>1</sup>, Anusha Venkataraman<sup>1</sup>

Authors Affiliation: <sup>1</sup>Department of ophthalmology, All India Institute of Medical Sciences, Bhubaneswar, Orissa India.

Six year old male referred to our Oculoplasty clinic for bilateral drooping of eyelids was diagnosed to have Blepharophimosis syndrome, (Blepharo- phimosis, ptosis, epicanthus inversus) (Fig. A) incidentally found to have multiple eruptions over flexure aspect of knees, ankles, buttock (Fig. B, C), arcus juvenilis suggestive of familial hyper -cholesterolemia. These co-existing autosomal dominant mutations for Blepharophimosis syndrome and Familial hypercholesterolemia in a single patient has never been reported in literature till date.



Fig. 1a: Blepharophimosis syndrome showing bilateral ptosis, Blepharophimosis, epicanthus inversus.





Fig. 2b and c: Xanthoma Tuberosum over flexure aspect of buttock, knees and ankles.

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[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003;61:347-55.

## Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997;195 Suppl 2:3-9.

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[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000;71:1792-801.

# Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. Dent Mater 2006.

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[6] Hosmer D, Lemeshow S. Applied logistic regression, 2<sup>nd</sup> edn. New York: Wiley-Interscience; 2000.

## Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

# No author given

[8] World Health Organization. Oral health surveys - basic methods, 4<sup>th</sup> edn. Geneva: World Health Organization; 1997.

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[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme\_health/ HSQ 20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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- Abbreviations spelt out in full for the first time. Numerals from 1 to 10 spelt out
- Numerals at the beginning of the sentence spelt
   out

# **Tables and figures**

- No repetition of data in tables and graphs and in text.
- Actual numbers from which graphs drawn, provided.
- Figures necessary and of good quality (color)
- Table and figure numbers in Arabic letters (not Roman).
- Labels pasted on back of the photographs (no names written)
- Figure legends provided (not more than 40 words)
- Patients' privacy maintained, (if not permission taken)
- Credit note for borrowed figures/tables provided
- Manuscript provided on a CDROM (with double spacing)

# Submitting the Manuscript

- Is the journal editor's contact information current?
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