Navigating the Retinal Periphery

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Abstract

The peripheral retina is crucial in diagnosing a variety of eye conditions, yet it often receives less attention than the central retina during routine examination. Peripheral retinal degeneration (PRD) is a broad term which includes various lesions such as the lattice degeneration, snail-track degeneration, snowflake degeneration, atrophic or operculated retinal holes, peripheral retinal tears, senile retinoschisis, white and dark without pressure areas, paving stone degeneration and peripheral cystoid degeneration.[1] While most of them are clinically insignificant, a few of these degenerations such as lattice degeneration, degenerative retinoschisis, peripheral retinal tears, cystic retinal tufts and, rarely, zonular traction tufts. can result in rhegmatogenous retinal detachment (RD).[2]

Key-words: Peripheral retinal degeneration (PRD), egenerative retinoschisis, rhegmatogenous retinal detachment (RD)

INTRODUCTION

Myopia is on the rise globally, with projections indicating that half of the world's population will be affected by 2050. The prevalence of pathologic myopia–related visual impairment has been reported as 0.1%-0.5% in European studies and 0.2%-1.4% in Asian studies. A thorough macular examination and peripheral depressed examination are key to detecting complications related to pathologic myopia.

CASE REPORT

A 24-year-old male came for routine ophthalmic evaluation. Patient is a glass wearer since 7 years of age. Upon examination his uncorrected visual acuity in both the eyes were counting fingers at 3 meters. His best corrected visual acuity was 6/6 in both the eyes. Anterior segment examination was found to be unremarkable (Table 1). A comprehensive fundus examination was done after instilling cycloplegics. Fundus examination of left eye showed hard exudates, telengiectatic vessesls and streak haemorrhage along the infero temporal vascular arcade (Figure 1). Notwithstanding initial expectations, a comprehensive ophthalmological assessment disclosed the unexpected presence of Coats disease, a singularly fascinating yet debilitating affliction of the retinal vasculature." A bespoke regimen of sectoral laser photocoagulation was judiciously administered to the patient, thereby facilitating judicious ablation of aberrant vasculature and optimizing visual prognosis (Figure 2).

Table 1: Table showing Anterior segment parameters

	Right Eye	Left Eye
UCVA	3/60	3/60
BCVA	6/6	6/6
STEEP K	43.50 *180'	42.30 * 178'
FLAT K	43.00* 80'	43.10 * 90'
AXIAL LENGTH	25.08 mm	24.78 mm
AC DEPTH	4.05 mm	4.00 mm

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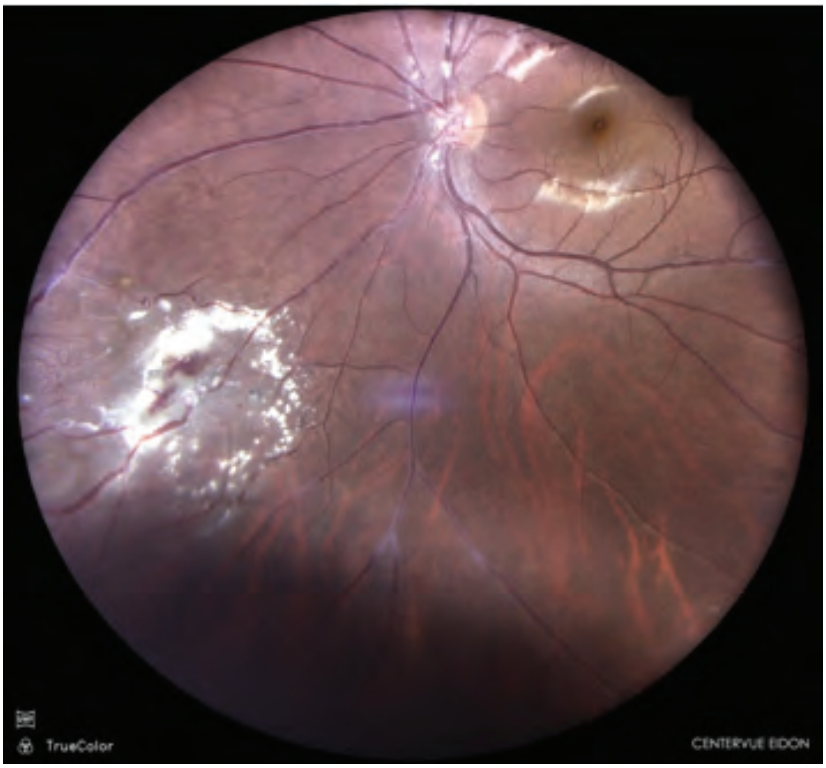

Figure 1: Explore the landscape Peripheral telangiectasia with exudation

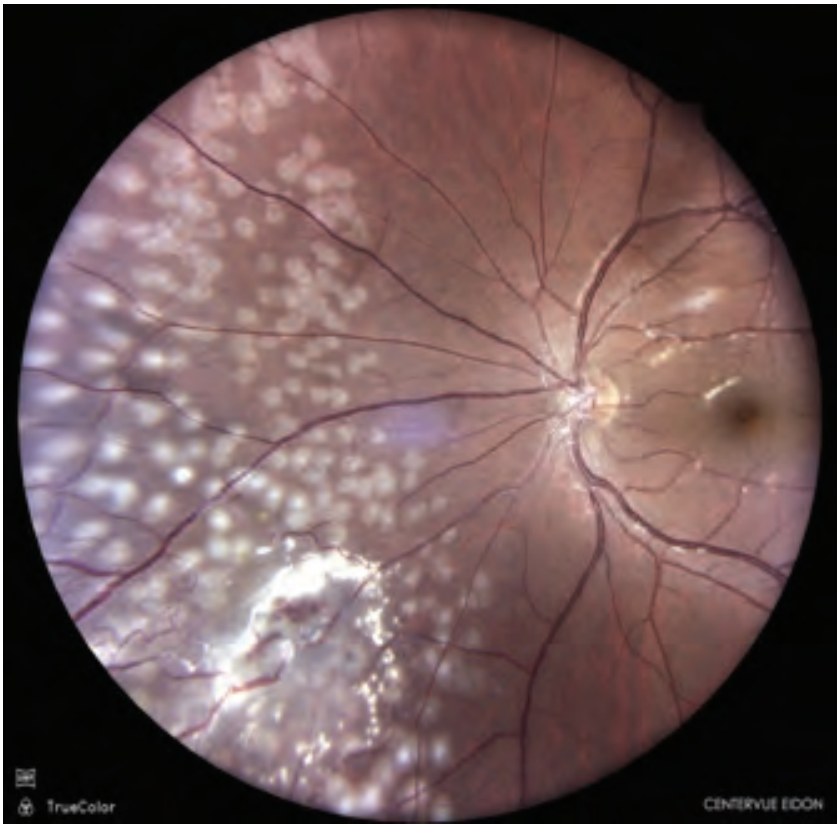

Figure 2: Resolution of telangiectasia and minimal residual peripheral exudation After treatment with diode laser photocoagulation

DISCUSSION

George Coats first described this condition in 1908 as unilateral retinal vascular abnormality with exudation occurring in young males.[3] Coats disease or Coats "response" has been inappropriately used as a blanket term for fundus changes seen in a variety of exudative retinopathies. It is primarily caused by aneurysms and telangiectatic vessels within the temporal retina. These abnormal vessels are leaky and there is exudation in various degrees. These have been described as "light bulb telangiectasias" because of the bulbous terminal configuration and associated extensive yellow exudation.[2] The anterior segment is usually not involved in early cases, but neovascularization of the iris or angle of anterior chamber, or neovascular glaucoma may be seen in later cases. Vascular abnormalities are usually prominent near the peripheral capillary dropout. Vascular leakage causes hard exudates which may be peripheral (near the vascular abnormalities) or mid peripheral and central (at the macula). In 2001, Shields et al classified Coats disease into five stages based on clinical features in 150 consecutive cases, ranging from asymptomatic retinal telangiectasia (stage 1), to telangiectasia with exudation (stage 2: 2A without macular involvement and 2B with macular involvement), exudative RD (stage 3: 3A1 with subtotal exudative RD sparing the fovea, 3A2 extending to the fovea, and 3B with total exudative RD), total RD with secondary glaucoma (stage 4), and end-stage disease with phthisis bulbi (stage 5).[4] The treatment options varied, with observation or laser photocoagulation for stage 1, laser photocoagulation or cryotherapy for stages 2-3A, cryotherapy for stage 3B with shallow RD, and surgical repair for stage 3B with bullous RD. Anti-VEGF are the newest additions to the armamentarium for management of Coats. In one analysis of 351 case, visual acuity following treatment was 20/20--20/40 (18%), 20/50--20/200 (22%), and 20/400 or worse (59%) and a trend toward better vision in recent years was documented, believed to be related to earlier recognition of the condition and more effective therapies.[5]

CONCLUSION

Peripheral retinal degenerative conditions are common, and often seen concomitantly. Predisposing factors include age and myopic refractive error; However, these may also occur as coincidental findings. Since vision-threatening sequelae serve as potential side effects, dilated fundus examination and ancillary imaging are crucial to determine the need for prevention, treatment and proper follow-up. Improvements in retinal imaging, diagnostic accuracy, surgical techniques for retinal detachment, lasers and intraocular injections have contributed to successful management. Greater awareness about the typical features, diagnosing the disease at an early stage and initiating appropriate treatment can greatly improve the visual outcomes and reduce the rate of enucleation in Coats disease. In the recent years, the visual outcome in Coats disease has improved which can be attributed to earlier detection and aggressive treatment.[4]

FINANCIAL SUPPORT AND SPONSORSHIP

Nil.

CONFLICTS OF INTEREST

There are no conflicts of interest.

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How to cite this article: T Karishma, Navigating the Retinal Periphery. Ocul Res J 2025;2(1): 17-20.