SWEPT SOURCE OPTICAL COHERENCE TOMOGRAPHY FINDINGS OF PACHYCHOROID SPECTRUM OF DISEASES IN PATIENTS WITH UNILATERAL ACUTE CENTRAL SEROUS CHORIORETINOPATHY

Dissertation submitted to The Tamil Nadu Dr.M.G.R. Medical University in partial fulfilment of the requirements for the degree of MS Ophthalmology

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OPHTHALMOLOGY

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CERTIFICATE

This is to certify that this dissertation entitled "**Swept source optical coherence tomography findings of pachychoroid spectrum of diseases in patients with unilateral acute central serous chorioretinopathy**" is a bonafide work done by **Dr.T.Vandhana, M.B.B.S,** under our guidance and supervision in the vitreo-retinal services department of Aravind Eye Hospital and Post Graduate Institute of Ophthalmology in Madurai during her residency period June 2017 to May 2020.

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DECLARATION

I, Dr.T.Vandhana solemnly declare that the dissertation entitled "**SWEPT SOURCE OPTICAL COHERENCE TOMOGRAPHY FINDINGS OF PACHYCHOROID SPECTRUM OF DISEASES IN PATIENTS WITH UNILATERAL ACUTE CENTRAL SEROUS CHORIORETINOPATHY**" has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or by anyone else for award, degree, diploma to any other university board either in India or abroad.

This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical university, Chennai in partial fulfilment of the rules and regulation for the award of M. S. Ophthalmology (BRANCH-III) to be held in May 2020.

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PART I

INTRODUCTION

Central serous chorioretinopathy (CSCR) is the fourth most common retinopathy after age-related macular degeneration, diabetic retinopathy and branch retinal vein occlusion[1]. CSCR occurs in males in the age group of 20 to 50 years. There is no underlying proven pathophysiologic mechanism for CSCR. It is thought to occur due to hyper permeable choroidal capillaries in association with retinal pigment epithelium causing serous detachment of neurosensory retina. Recurrence rate of CSCR is about 33% and some texts show the recurrence rate of about 50%[3].

In 1866,the disease was first recognized by Albrecht Von Grafe and named as central recurrent retinitis. It has been reported under a variety of names such as idiopathic flat detachment of the macula by Walsh et al, central angiospastic retinopathy by Gifford et al, and central serous retinopathy by Straatsma et al. Gas et al, named the condition as Idiopathic central serous chorioretinopathy in 1967.

EPIDEMOLOGY

Most of the patients with CSCR are between the age group of 28 to 68 years with an average age of 43years. Patients above 50 years has more chance of having bilateral disease(50%) with RPE loss and choroidal neovascularisation[4]. Males(9.9/100,000) are 6 times more commonly affected than females 1.7/100,000).Similar prevalence of the disease were noted in African, American, Asian and Caucasian[5,6].

PATHOPHYSIOLOGY

There is no exact proven pathologic mechanism in CSCR. But epidemiology, hormonal studies, fluorescein angiography (FA) and optical coherence tomography(OCT) helps in elucidating some of the pathological manifestations.

THEORIES OF PATHOGENESIS

Role of the choroid

Currently, pathogenesis of CSCR is mainly based on the role of choroid. The hyperpermeability of choroid is thought to be major cause of CSCR due to stasis, ischemia or inflammation[7]. There is increased tissue hydrostatic pressure due to hyperpermeability of choroidal vessels which leads to the formation of retinal pigment epithelial detachments (PEDs),disrupts the barrier function of RPE leading to fluid accumulation between RPE and retina.

Role of retinal pigment epithelium

The pathogenesis of retinal pigment epithelium theories predominated in CSCR before wide spread acceptance of underlying choroidal pathology. RPE dysfunction plays a significant role in the pathogenesis of CSCR. Before the acceptance of an underlying choroidal pathology, RPE theories of pathogenesis predominated. The characteristic

of classic CSCR are focal areas of leakage through retinal pigment epithelium were one of the first clues to the pathogenesis of the disease. These pinpoint leaks were seen as focal defects in the RPE that were thought to be primarily responsible for the accumulation of subretinal fluid. However, Negi and Marmor showed that focal RPE defects promoted flow of fluid out of the subretinal space toward the choroid rather than vice versa[8]. An alternate theory is that focal loss of polarity of RPE cells leads to active fluid pumping into the subretinal space[9].

The most complete theory states that increased tissue hydrostatic pressure in the choroid causes disruption of barrier function of the RPE and leads to areas of fluid accumulation between the retina and the RPE. Some refer to the pinpoint areas of leakage seen in acute CSCR as micro-rips or blowouts. OCT showed that RPE abnormalities are present in nearly all asymptomatic, fluid-free contralateral eyes of CSCR patients[10]. This indicates that RPE damage is not necessarily a late sequela of CSCR, but occurs even in forme fruste.

Role of hormonal factors

Central serous chorioretinopathy and glucocorticoid use has strong association in pathogenesis. Both serum catecholamines[12,13] and serum glucocorticoids[11] are elevated in active CSCR. Glucocorticoids has impact on the course of the disease by affecting the choroid, Bruch's

membrane, or the RPE[14]. Proposed mechanisms in the choroid include effects on vascular autoregulation via increased transcription of adrenergic receptors[15,16] or potentiation of vascular reactivity[7],effects from steroid-induced systemic hypertension[17], or a prothrombotic effect[18]. Corticosteroids affect Bruch membrane by inhibition of collagen synthesis[19]. The epithelial water and ion transports are altered by corticosteroids[20], which impairs the barrier function of the RPE.

Local ocular glucocorticoid use is usually not associated with CSC[21]. Every form of exogenous glucocorticoid has been associated with exacerbation, and ocular and systemic glucocorticoids should be avoided in CSC. The association of elevated catecholamine levels is related to simultaneous increased levels of corticosteroids in a physiologic stress response.

Helicobacter pylori infection

Several studies showed that there is association between *H. pylori* infection and CSCR[22], and some have noted a beneficial effect in CSCR in patients treated for *H. pylori*[24]. *H. pylori* is a Gram-negative bacterium that causes gastritis and also associated with extra gastric conditions, including thrombotic

disease[23]. Thrombotic disease is a pathway through which infection can cause CSCR. Another proposed mechanism is immune-mediated damage to choroidal endothelial cells resulting from molecular mimicry[24].

Genetics

A review of literature revealed numerous reports of familial CSCR[25].Weenink and colleagues[24] who found CSC-like pathology in 52% families of chronic CSCR patients. Just one population-based prevalence study has been conducted to date, and this was in a predominantly white, American population. CSCR thought to have a higher prevalence in whites, Hispanics, and Asians than in African Americans[26].

RISK FACTORS

Central serous chorioretinopathy is associated with increased sympathetic activity and obstructive sleep apnoea. Common risk factors include pregnancy, antibiotic use, alcohol use, untreated hypertension, and obstructive sleep apnea[27].

Increased levels of the catecholamine, adrenaline and noradrenaline are seen in patients with CSCR. Increased plasma concentrations of adrenaline are highly correlated with central macular thickness. Cushing's syndrome from endogenous causes, such as pituitary adenoma, adreno cortical adenoma and carcinoma, as well as from exogenous corticosteroid use, are associated with CSCR. Among organ transplant patients receiving long-term corticosteroids, renal transplant patients were at particular risk, possibly due to underlying renal disease, hypertension, microangiopathy and previous exposure to haemodialysis, all of which may modify choroidal haemodynamics. Exogenous corticosteroid administration including oral, topical, intravenous, intranasal, intraarticular and intravitreal appear to be associated with increased risk of CSCR.

Fok found that patients having psychiatric disorders such as depression are more likely to have a recurrence[28]. On average studies showed that the acute stressor preceeds the onset of CSCR by a week .It

remains unclear that whether removal of stressor prevents recurrences or progression to chronic CSCR. Type A personality indivduals are at more risk. The association between CSCR and type A personality is proposed to be mediated through higher level of circulatory catecholamines and corticosteroids in type A personality compared to type B.

Pregnancy is associated with increased risk of developing CSCR. This is due to the increased endogenous corticosteroids that occur during pregnancy. Incidence of CSCR in pregnancy occurs commonly during the third trimester and resolves within 1–2 months after delivery. For reasons that remain unclear, white subretinal deposits is the common finding in CSCR in pregnancy.

CLINICAL FEATURES

The features of central serous chorioretinopathy can be divided into acute and chronic form. The presenting symptoms include central scotoma metamorphopsia, micropsia, or blurred vision. The refraction may show a hypermetropic shift due to subretinal fluid. In acute stage, serous macular detachment is seen. Occasionally, yellowish subretinal material may be seen which is due to subretinal fibrin. In long standing cases, Subretinal precipitate-like deposits are seen. RPE defects may be seen clinically. The acute episode of CSCR generally resolves within 3–4 months. If fluid persists beyond this period, it is called as "non resolving or persistent CSCR."

The chronic stage, termed as diffuse retinal pigment epitheliopathy, shows RPE degeneration along with shallow SRF and cystoid or schitic retinal edema. RPE degenerative changes may be seen all over macula affecting the vision. The RPE tracks due to the gravitational tracking of the fluid can be seen in tear drop configuration. After a documented resolution of fluid and betterment of symptoms, if the SRF reappears, it is called as "recurrent central serous chorioretinopathy."

Central serous chorioretinopathy may be associated with inferior exudative retinal detachment with shifting of fluid. In such cases there can be multifocal PED at the posterior pole and multifocal leaks on FFA.

This is generally seen where the patient is undergoing systemic corticosteroid therapy for conditions such as an organ transplant or autoimmune disease. Discontinuation of steroids showed resolution in 87.5% cases[29].

CSCR is generally self-limiting disease but chronic cases with RPE degeneration may progress to foveal atrophy or CNVM (2%–9% cases) causing vision loss. Furthermore, cases of PCV are shown to be associated with CSCR[30].

INVESTIGATIONS

Optical coherence tomography

Optical coherence tomography is the first line imaging modality used for the diagnosis of CSCR. Recently, emergence of OCT with deep imaging techniques such as enhanced depth imaging (EDI), en face swept source-OCT (SS-OCT) helps in imaging of RPE-Bruch membrane complex, and choroidal vasculature at variable depths. These newer imaging techniques help in improved morphological analysis and hence better elucidation of the pathophysiology and management of CSCR.

The hallmark of CSCR is the presence of a serous detachment of the neurosensory retina and sometimes associated with a serous RPE detachment and these findings are easily detected and quantified with optical coherence tomography (OCT). In long-standing CSCR, there will be intraretinal fluid and cystoid oedema. There can be precipitates which are hyperreflective on OCT and can be found both within the retina and in the subretinal space. OCT finding of disruption of the IS/OS junction, correlate with poor visual outcomes[36].

CSCR is currently considered as disease of the pachychoroid spectrum of disorders. A number of EDI-OCT-based studies have shown increased choroidal thickness in eyes with CSCR as well as in the fellow eyes, compared to normal healthysubjects[31,32,33,34]. Studies have shown

thicker choroid in the involved eye of CSCR compared to that in uninvolved fellow eye[35].Increased choroidal thickness is a common association with CSCR, but it is not a mandatory criterion for making a diagnosis of CSCR.

Swept source OCT

SS OCT and enhanced depth imaging mode of SD-OCT are used in visualising the choroid and to know about the disease process in pachychoroid spectrum of diseases. SS-OCT uses short cavity swept laser with a tunable wavelength instead of diode laser used in spectral-domain OCT[70].SS-OCT has more depth penetration compared to SD OCT using a wavelength of 1050 nm and has an axial resolution of5.3μm and axial scan rate of 100.000 scans per second. The 12 × 9 mm scan helps in simultaneous imaging of the macula, peripapillary area and the optic nerve head and the choroidal thickness. The 12 × 9 mm scan comprises256 B-scans each comprising 512 A-scans with a total acquisition time of 1.3 s [71].

The choroid is described in layers. Outer Hallers layer containing larger vessels, sattler layer containing medium sized vessels and inner choriocapillaris[72,73]. The choriocapillaris has fenestrations which is maintained by consecutive secretion of vascular endothelial growth factor (VEGF) by the RPE.

OCT enabling better visualisation of choroid are EDI of the SD-OCT and SS-OCT. In SD-OCT, depth information is encoded as different frequencies of the interference spectrum, with increasing depth into tissue, echoes beyond the point of detection are known as the "zero delay line." Enhanced depth Imaging was first reported by Spaide and associates. The SS-OCT has a longer wavelength of penetration of tissue greater than SD OCT, thus both vitreous and choroid can be imaged simultaneously. The first study on measuring choroidal thickness (CT) was done by Margolis and Spaide in which they investigated 54 normal, non-myopic eyes with the EDI of the SD-OCT and found a sub-foveal thickness of 287 μm[74].Evaluation of choroidal thickness is an important diagnostic hallmark of several retinal diseases. The main feature of CSCR is Choroidal hyperpermeability[75].Choroidal thickness is increased in thosepatients[76,77].Tan and colleagues compared choroidal thickness measurements in SD-OCT and SS-OCT where differences by more than 50μm (SD-OCT thicker) werefound[78].

The commercially available SS-OCT Triton (Topcon, Tokyo, Japan) measures the choroidal thickness with automated segmentation using a viewer software where each sector can be measured separately. This feature has a promising role on the sectoral analysis and follow-up of retinal pathologies involving the choroid[79].

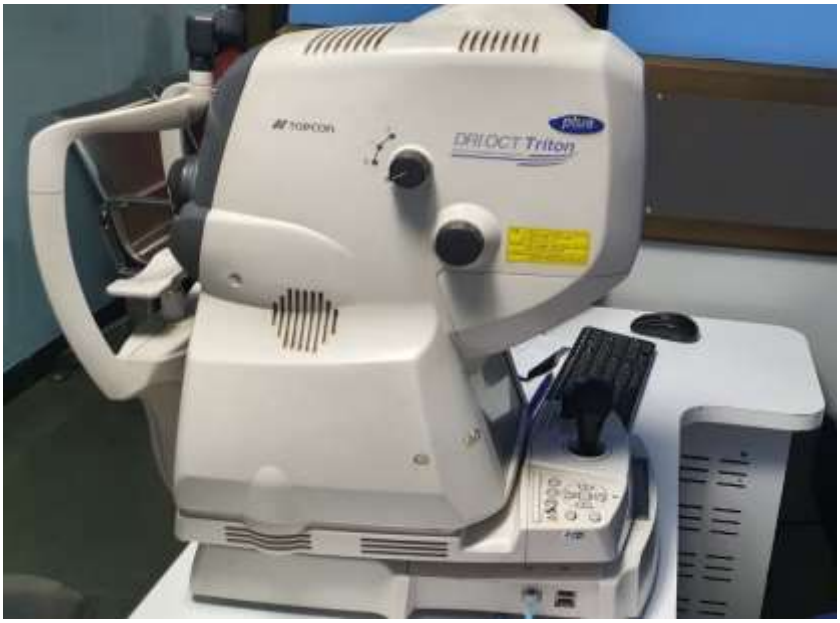


Figure1.1 Swept source OCT(Triton)

SS-OCT helps in the visualisation of choroidal vasculature in CSCR eyes. There will be thinning of inner choroidal layer in the involved area either due to primary atrophy of choriocapillaris or due to compression by dilated outer choroidal vessels[31]. Outer choroidal vessel dilatation is seen in eyes with CSCR as well as other entities of pachychoroid spectrum of disorders[32].

Fundus fluorescein angiography(FFA)

The characteristics FFA findings of central serous chorioretinopathy includes leakage from retinal pigment epithelium and irregular relative window defects. Leakage from retinal pigment

epithelium typically occurs in any one of the following 3 patterns 1.expansile dot pattern 2.smokestack pattern 3.diffuse pattern.

The most common pattern is expansile dot pattern which appears hyperfluorescent. It appears in early phase and it increases in size and intensity as the study progresses. Another pattern is smoke stack pattern occurs in 10% of cases which appears as hyperfluorescent in early phase spreading vertically and finally laterally as the study progresses to later phase resembling plume of smoke. This pattern is thought to be secondary to convection current and to a pressure gradient between the protein concentration of the subretinal fluid and fluorescein dye entering the detachment. In rare cases an extensive, often gravity dependent serous detachment of retina can develop from one or more leakage points outside the central area or may be associated with diffuse pattern of fluorescein leakage.

Indocyanine green angiography

ICG angiography can reveal choroidal vascular abnormalities including filling delays in the choroidal arteries and choriocapillaris, venous dilatation, hyperpermeability of choroidal vessels and characteristic multifocal choroidal hyperfluorescent patches that appears in the early phase of the study. These areas slowly enlarge but less prominent in late phase of the study. ICG angiography is helpful in

distinguishing atypical diffuse CSCR from both occult choroidal neovascularisation and idiopathic polypoidal choroidal vasculopathy.

Fundus autofluorescence imaging

There can be relative hypo autofluorescence corresponding to the site of focal RPE leak depicted on fluorescein angiography and in the area of subretinal fluid. In chronic cases subretinal fluid persisting can show mild to dramatic hypo autofluorescence. Increased autofluorescence often observed in areas of serous detachment may be related to unmasking of retinal pigment epithelium fluorescence by photoreceptor pigmentary atrophy.

TREATMENT

Acute CSCR often resolves spontaneously within a few months without significant visual impairment. In general, observation and finding out the risk factor and adjusting modifiable risk factors, particularly cessation of exogenous steroid use and control of systemic hypertension, is a useful initial management option without any intervention.

Recurrent CSCR is characterised by multiple spontaneously resolving episodes, whereas chronic CSCR appear to have persistent subretinal fluid for at least 3–6 months. Due to persistent serous retinal detachment chronic CSCR has progressive visual dysfunction. As chronic CSCR has progressive visual dysfunction, further treatment options are indicated.

Laser photocoagulation

Focal laser photocoagulation is considered as treatment option for acute CSCR by enabling sealing of any focal RPE defect that causes leakage seen on fluorescein angiography[37]. Studies showed that there will be decrease in neurosensory detachment seen in 8 to 10 weeks after laser photocoagulation in acute central serous chorioretinopathy[38]. Laser photocoagulation, particularly green laser, showed good response in the management of extrafoveal leaks in acute CSCR[39,40].

Photodynamic therapy with verteporfin

Treatment of chronic CSCR with photodynamic therapy (PDT) and verteporfin was first demonstrated in case series by Chan *et al.* using indocyanine angiography-targeted treatment[41]. It is thought that PDT alters choroidal vasculature structure and perfusion, thereby reducing choroidal permeability[42]. This reduces the subretinal fluid associated with the macular neurosensory detachment associated with CSCR. Diffuse application of PDT is associated with alteration in choroidal pigmentation, RPE atrophy, choriocapillaris nonperfusion and possible choroidal neovascularisation. As a result, various studies of modified forms of PDT have been evaluated with modification of PDT fluence, treatment times, dose level and treatment interval. In PDT, hypoxic areas with reduced choriocapillaris flow leads to excess treatment of normal healthy choroidal structure with the possibility of damage to adjacent healthy tissues. Reduction of PDT fluence results in more targeted treatment of affected choroid, limiting RPE disturbance and the possibility of visual dysfunction.

In patients treated with standard PDT, increased areas of choriocapillaris nonperfusion were observed. This suggests modification of PDT fluence which can still deliver good treatment outcomes with the possibility of reduced side effects. In summary, recent study of modified

PDT has suggested that both reduced (most notably half) dose and reduced-fluence PDT can deliver good treatment outcomes in CSCR with possible reduced adverse ocular side effects.

Subthreshold retinal laser treatment

Subthreshold retinal laser therapy has been evaluated in the treatment of various macular disorders. Unlike conventional continuous laser, subthreshold laser minimises associated thermal injury, making it more appropriate for application near to the fovea. It is thought that RPE is almost solely affected without significant treatment on the retina. This may, therefore, prevent the development of central scotoma, retinal scarring and choroidal neovascularisation, all of which have been associated as possible side effects of conventional argon retinal laser treatment. Stimulation of the RPE is thought to be important in repair of the inner blood retinal barrier and regulation of vascular endothelial growth factors (VEGFs) altering RPE permeability. Conventional laser delivers treatment in 0.1–0.5 s, whereas micro pulse laser delivers a train of repetitive short laser pulses within the same 0.1–0.5 s.

The 577 nm laser is a yellow laser that is minimally absorbed by the yellow pigment xanthophyll, thus sparing its action to the inner and outer plexiform layer near the fovea[43]. Treatment outcomes with

subthreshold retinal laser therapy are particularly encouraging, but long-term outcomes, including side effects, require evaluation.

Newer laser technology including NAVILAS® laser treatment has been suggested to deliver more targeted laser treatment using eye-tracking technology based on a previously planned fluorescein guided treatment. Many patients were noted to have no vision loss due to this treatment. This newer technology needs further evaluation in larger studies to determine the efficacy of NAVILAS® laser treatment in CSCR.

Mineralocorticoid antagonism

Exogenous steroid use has been consistently demonstrated as a risk factor for development of CSCR, while increased expression of ocular mineralocorticoid receptors were found in eyes with CSCR. Activation of steroid receptors can lead to alteration into electrolyte balance affecting generation of subretinal fluid[44].

Eplerenone is a mineralocorticoid receptor antagonist used in the treatment of hypertension and congestive heart failure. Eplerenone has increased ocular selectivity with reduced systemic side effects associated with other oral mineralocorticoids.

Small studies treating patients with chronic CSCR with oral eplerenone have shown improvement in visual acuity and reduction of central retinal thickness[45]. The possible side effect of these potassium-sparing diuretics is hyperkalemia, especially in those with renal disease and electrolyte levels should be monitored during treatment. Schwartz *et al.* evaluated the use of 50 mg dose of eplerenone for 3 months, whereas Rahimy *et al.* evaluated the use of 25 mg/day for 1 week then 25 mg for 8 weeks; both studies showed improvement in anatomical and visual outcomes[46].

DIFFERENTIAL DIAGNOSIS

Age-related macular degeneration

ARMD is the most important differential diagnosis in CSCR patients aged 50 years or more. Secondary Choroidal neovascular membrane, most commonly type2, can develop in patients with chronic CSCR during follow-up or after laser photocoagulation.

Polypoidal choroidal vasculopathy

Because of subretinal detachment, RPE alteration and choroidal hyperpermeability in ICG, sometimes it becomes difficult to distinguish Polypoidal choroidal vasculopathy (PCV) from chronic CSCR. Presence of subretinal haemorrhage, branching vascular network and leaking polyps in ICGA favours the diagnosis of PCV. OCT typically shows

serosanguineous, notched or tall peaked PED and higher optical density of subretinal fluid[47].

Optic disc pit

Optic disc pits are focal excavations present in the temporal aspect of optic nerve head, creates a communication between the vitreous cavity, the subretinal space and to some extent the subarachnoid space. They produce chronic or recurrent subretinal detachment(SRD) following schitic changes of inner retina with variable intraretinal cystoid oedema. Careful peripapillary examination and absence of leakage in FFA remain diagnostic of optic disc pits.

Inflammatory diseases

Vogt–Koyanagi–Harada (VKH), a bilateral granulomatous panuveitic condition, often presents with multiple SRD which mimics CSCR. Apart from its systemic, neurological and dermatological signs, the presence of vitritis, increased choroidal thickening in ultrasound and pinpoint multifocal leaks on FFA readily distinguish it from CSCR. Differentiation for this condition is most importance as unlike CSCR systemic steroids are the mainstay of treatment in VKH.

Autoimmune and vascular disorders

Autoimmune diseases, such as systemic lupus erythematous, polyarteritis nodosa and scleroderma can have neurosensory detachment during systemic steroid therapy. Non-autoimmune conditions such as malignant hypertension, toxaemia of pregnancy and disseminated intravascular coagulopathy can also present with a secondary NSD due to choroidal arterial occlusion.

Intraocular tumours

Various types of choroidal tumors including choroidal hemangioma, melanoma, osteoma and choroidal metastasis can cause exudative SRD mimicking CSCR. It is important to differentiate a malignant and potentially lethal condition from CSCR. Ultrasonography helps in detecting and differentiating the nature of the tumor. ICGA shows classical 'wash-out' phenomenon in late phases in choroidal hemangioma and EDI-OCT shows increased caliber of large choroidal vessels in the tumor along with normal choriocapillaris[48].

Recently central serous chorioretinopathy has been considered as one of the diseases in pachychoroid spectrum.

PACHYCHOROID SPECTRUM

The pachychoroid spectrum of diseases was first described by DavidWarrow, MD, and colleagues in 2013, who specified a group of conditions characterized by choroidal thickening and retinal pigment epithelial changes, with or without corresponding retinal abnormalities. In increasing visual significance and severity, the four disease groups in the pachychoroid disease spectrum are described below.

pachychoroid pigment epitheliopathy (PPE);

central serous chorioretinopathy (CSCR);

pachychoroid neovasculopathy (PNV)

polypoidal choroidal vasculopathy (PCV).

The common characteristics of this spectrum of disorders are

1.Increase in choroidal thickness

2.dilated vessels in hallers layer

3.Thinning of choriocapillaris

Increase in choroidal thickness

Using enhanced depth imaging optical coherence tomography(EDI-OCT) or swept source OCT(SS-OCT), the choroid–scleral interface can be delineated thus helps in quantitative analysis of

choroidal thickness. In previous studies, Sub foveal choroidal thickness in normal subjects was reported to be between 191–350 µm [49], but choroidal thickness can be influenced by a variety of factors, including age, axial length, refractive error, blood pressure, as well as time of the day.

In view of the wide range of choroidal thickness and the multiple factors which may influence this parameter, there is no definitive quantitative threshold for defining an eye as having abnormally thick choroid. However, many investigators may consider sub foveal choroidal thickness >300 µm as pathological [32,50].Studies showed that there is regional variation of choroid such that the thickest region beneath the fovea and thinnest areas nasally[51].

Pachyvessels

Increased choroidal thickness is mainly due to dilatation of choroidal vessels in Haller's layer. Increased diameter of choroidal vessels appears as larger hypo reflective lumen in OCT in eyes with CSCR, PCV, focal choroidal excavation (FCE) [52]. In histological sections of PCV,there can be dilated choroidal vessels with diameter of up to 300 µm. Pathologically dilated vessels in eyes with pachychoroid disease can be detected within the deep choroid [53]. In addition to increase in calibre, pachyvessels do not taper toward the posterior pole, but retain their large caliber and terminate abruptly and this is considered as differentiating

feature from normal choroidal vessels. This feature is best appreciated using en face OCT or ICGA.

Pachyvessels may be present diffusely or focally involving 1 or 2 quadrants. When localized, they correlate spatially with the areas of maximal choroidal thickening, as well as disease focus within the RPE or retina [32]. In areas overlying pachyvessels, choriocapillaris thinning may be noted as evidenced by inward displacement of large dilated choroidal vessels which become visible in a more superficial en face plane.

Thinning of choriocapillaris

Healthy eyes with abnormally thick choroids may be considered to have "pachychoroid" or "uncomplicated pachychoroid". Regardless of choroidal thickness, presence of morphological features of pathologic sequelae resulting from abnormally dilated choroid may be a more significant finding for diagnosing "pachychoroid disease" [54,55]. A key feature is inner choroidal attenuation, characterized by focal or diffuse attenuation of the choriocapillaries and intermediate caliber vessels within Sattler's layer in areas overlying abnormally dilated Haller's layer vessel [32].

In addition to thinning of the inner choroid, thinning of outer nuclear layer (ONL) was observed in eyes with pachychoroid disease. Interestingly, the ONL was thinner in eyes with pachychoroid pigment

epitheliopathy (PPE) than in eyes with uncomplicated pachychoroid in one study, suggesting that degeneration of photoreceptors and/ or RPE may also occur, even in the absence of Subretinal fluid[50].

Pachychoroid pigment epitheliopathy

The term PPE was first introduced by Warrow and colleagues to refer to a condition characterized by retinal pigment epithelial changes occurring in the posterior pole in regions of choroidal thickening [58]. PPE is noted in uninvolved fellow eyes of unilateral CSCR patients with no history of neurosensory detachment. These patients were often misdiagnosed with pigmentary age-related macular degeneration (AMD), and sometimes with pattern dystrophy or "retinal pigment epitheliitis" [59]. However, PPE is usually asymptomatic. The clinical appearance of the pigment epitheliopathy included RPE mottling, irregular areas of RPE elevation termed "drusenoid RPE lesions".

FAF showed similarly mottled hypo autofluorescence but also revealed hyper autofluorescent features which correlated with foci of apparent RPE thickening or hyperplasia seen on cross-sectional OCT [32]. The choroidal findings of this patients show hyperpermeability with ICGA in the distribution of the pigment epitheliopathy and pathologically dilated vessels in Haller's layer [32]. It is differentiated from non neovascular age related macular degeneration by reduced fundus

tessellation together with the frequently extrafoveal location of the pigment epitheliopathy and the relatively young age of the patients. Studies showed that since no eyes had manifested neurosensory detachment, PPE was considered a forme fruste of CSCR. Moreover, it was subsequently observed that patients with PPE can develop type 1 neovascularization, with or without polypoidal lesions, without necessarily developing CSCR[32,56,57].

Pachychoroid neovasculopathy

Although development of secondary CNVM has been described in CSCR[4,64], the incidence has not been well established. Chronic CSCR from AMD is difficult to differentiate as the two conditions may have very similar features on FA and ICGA, characterized by RPE atrophy and diffuse leakage. With advances in choroidal imaging, differences in choroidal features have been noted among patients presenting with type 1 neovascularization.

Fung described a group of patients showing type1 neovascularization with clinical and imaging findings more consistent with long-standing CSCR than with age related macular degeneration[65]. The features which differentiated PNV patients from neovascular AMD are increased choroidal thickness, absent or minimal soft drusen, and younger age. Some of these eyes also had polypoidal structures within

their type 1 neovascular network. Importantly, Fung's study established a clear temporal sequence of CSCR which predated the development of type 1 neovascularization (mean interval of 139 months), and thus support a pathogenic sequence.

The authors also emphasized that typical neovascular AMD should be differentiated from this type of neovascularisation.[65]. Subsequently, in eyes with other pachychoroid disease entities the occurrence of type 1 neovascularization was described. Pang proposed that PNV which is a disease of pachychoroid spectrum occurs due to a pachychoroid-driven process involving choroidal congestion and hyperpermeability. The characteristic features of PNV on OCT include presence of type 1 neovascularization as a shallow irregular separation of the RPE from Bruch's membrane which appears as "double layer sign" overlying pachyvessels[66]. The presence of sub RPE neovascularisation appears as heterogeneously hyperreflective material in the sub-RPE space. Small peaked PEDs may develop the margin of these lesions within which aneurysmal (polypoidal) lesions may be identified with ICGA or OCTA.

The background features common to the pachychoroid disease spectrum, including an absence of soft drusen and reduced fundus tessellation indicative of a thickened choroid in the area of the type 1 neovascular lesion is seen in eyes with PNV. Importantly the areas of

type 1 neovascularization are correlated to areas displaying pachychoroid features [32].In FFA presence of neovascularization can be confirmed by detection of leakage in late phase in undetermined origin, and a corresponding late staining "plaque" on ICGA. Eyes with pachychoroid features such as shallow irregular PED on SD-OCT should be evaluated with OCTA, as these eyes frequently has neovascular tissue which is a feature of polypoidal neovasculopathy.

Polypoidal choroidal vasculopathy

In 1990 Yanuzzi et al described Idiopathic PCV in which haemorrhagic and exudative neurosensory detachments seen in the peripapillary region and macula[48]. In 1995, Spaide and colleagues described the characteristic findings of PCV as branching vascular network (BVN) with terminal aneurysmal (polypoidal) dilatations in ICGA[67]. Recent multimodal imaging demonstrated that PCV in a variant of type 1 (sub-RPE) neovascularization, as both the vascular dilations and their feeding vascular network are consistently found in a potential space bounded anteriorly by the RPE and its basal lamina and posteriorly by the inner collagenous layer of Bruch's membrane[68].

Recent studies using EDI-OCT and SS-OCT have demonstrated that there will be thick choroids in PCV patients and in contrast choroidal thinning is seen in eyes with typical neovascular AMD [69]. In patients with

PCV the presence of choroidal thickening and choroidal hyperpermeability suggests a link between this entity and the pachychoroid disease spectrum, in particular PNV.

Management considerations of pachychoroid disorders

In general, most pachychoroid disease in patients without any symptoms can be observed and monitored without treatment. Observed cases might include eyes with pachychoroid pigment epitheliopathy, central serous chorioretinopathy and extrafoveal subretinal fluid and/or Pigment epithelial detachment, and polypoidal choroidal vasculopathy with inactive non-leaking aneurysms. However, in patients experiencing vision loss due to pachychoroid disease, treatment should be considered to improve or stabilize visual function. These cases might include central serous chorioretinopathy with persistent central subretinal and/or large pigment epithelial detachment, pachychoroid neovasculopathy associated with macular exudation, and Polypoidal choroidal vasculopathy macular exudation originating from either the aneurysm or the associated branched vascular network. Intravitreal anti-VEGF therapy with or without adjunctive vPDT is now commonly used as the standard treatment of symptomatic macular PCV/AT1 related to pachychoroid disease.

REVIEW OF LITERATURE

1. Kunal K. Dansingani et al in 2016 conducted study in en face imaging of pachychoroid spectrum of diseases including pachychoroid pigment epitheliopathy, central serous chorioretinopathy, polypoidal choroidal vasculopathy and pachychoroid neovasculopathy.They correlated clinical manifestations with choroidal morphology in pachychoroid disorders[61].

Patients with pachychoroid spectrum diagnoses were identified through multimodal imaging. Each eye with uncomplicated pachychoroid, pachychoroid pigment epitheliopathy, central serous chorioretinopathy, pachychoroid neovasculopathy and polypoidal choroidal vasculopathy were identified and underwent bilateral swept-source OCT.

Eyes with all 4 pachychoroid spectrum disorder was found to have increased choroidal thickness and dilated outer choroidal vessels. Some cases with chronic CSCR has focal atrophy of choriocapillaris .They concluded that diseases of pachychoroid spectrum share common morphologic findings such as increased choroidal thickness and outer dilated vessels. En face SS OCT localizes these changes to disease foci and shows additional findings that may unify our understanding of disease pathogenesis.

2. Gupta P et al in 2010 conducted study to find out retinal pigment epithelial changes in affected and unaffected eyes of idiopathic CSCR using SD OCT[10].It is a prospective study in which 3 dimensional single-layer RPE map was done in both eyes for morphological alterations, and findings were correlated with clinical presentation, fluorescein angiogram, and 5 Line raster scan.

The results were in patients with CSCR, 3D single-layer RPE analysis of asymptomatic eyes showed presence of RPE bumps in 94% of eyes and pigment epithelium detachment 11.8% of eyes. The 5 Line raster scan was normal in all eyes. They found that SD OCT showed RPE changes in both eyes of idiopathic CSCR.

3. Lee WJ et al in 2017 conducted study to evaluate the tomographic features of choroidal vasculature in acute and chronic CSCR using swept source optical coherence tomography en face imaging[33]. It was a retrospective study in which 29 patients with acute and chronic CSCR underwent 6x6 macular scans with SS OCT, FFA and ICG.

They observed that choroidal vessel dilatation was seen in 91% of acute and 88% of chronic CSCR. In acute CSCR, choroidal vessel dilatation was divided into focal in 81.8% and diffuse 18.2% patients. The chronic CSC cases demonstrated focal vessel dilatation in 33.3% and

diffuse 66.6%. 80% of the acute CSCR and chronic CSC eyes were found to have obscured choriocapillaris and Sattler's layers on en face imaging.

They concluded that En face imaging of SS-OCT is useful when combined with angiography in CSCR for evaluating choroidal vessel dilatation at Haller's layer and to identify obscured upper layers. They have identified different choroidal vessel dilatation patterns between acute and chronic CSC. These findings might be useful for pathophysiological understanding of CSC.

4.Margolis R et al in 2009 conducted pilot study on measurement of choroidal thickness at different points in normal eyes using EDI OCT and compared with age[51].They found that choroid is thickest sub foveally of 287 µm (SD ±78µm).Choroidal thickness decreased nasally to fovea and averaged 145 µm (+/- 57 µm) at 3 mm nasal to the fovea.

Study showed that Choroidal thickness seems to vary topographically within the posterior pole. The thickness of the choroid showed a negative correlation with age.

5.Yutaka Imamura et al in 2009 conducted to evaluate choroidal thickness in central serous chorioretinopathy[76].Patients with CSCR underwent EDI SD-OCT which was obtained by positioning OCT device close enough to the eye to acquire an inverted image. The choroidal thickness was measured from outer border of retinal pigment epithelium

to inner scleral border. The mean age of the patients were 59.3 years.The choroidal thickness measured was 505 μm (standard deviation, 124μm), which was significantly greater than the choroidal thickness in normal eyes. EDI SD-OCT demonstrated a very thick choroid in patients with CSCR. This finding provides additional evidence that central serous chorioretinopathy may be caused by increased hydrostatic pressure in the choroid.

6. Yang L et al in 2013 conducted study to measure the choroidal vessel diameter in central serous chorioretinopathy[52].They included patients with unilateral CSCR and a control group of normal subjects. Sub foveal choroidal thickness (SFCT) and the largest diameter of choroidal hypo reflective lumen as surrogates for the choroidal vessels were measured by EDI OCT. They found that mean sub foveal choroidal thickness was larger in the affected eyes(455±73μm) than in contralateral unaffected eye(387±94μm) and larger than in control group(289±71μm). In a parallel manner, the mean diameter of the largest hypo reflective lumen was larger, in the affected eyes (305 ± 101 μm) than in the in the contralateral unaffected eyes (251 ± 98 μm), and larger than in the control group (140 ± 40 μm).

7.Kim YT et al in 2011 measured choroidal thickness in both eyes of patients with unilateral acute CSCR and compared with unaffected fellow eye and normal eyes using EDI OCT[80]. The mean choroidal

thickness of the affected eyes, unaffected fellow eyes, and normal individuals were 445.58±100.25, 378.35±117.44, and 266.80±55.45 μm, respectively. In this study they observed that compared to normal eye, choroidal thickness is significantly increased in active CSCR compared to inactive fellow eyes.

8.Ferrera D et al in 2014 conducted study to evaluate features of chronic CSCR using SS OCT[53].They found that SS OCT enables the visualization of pathologic features of the RPE and choroid in eyes with chronic CSCR which is not usually appreciated with standard spectral domain (SD) OCT. En face SS-OCT imaging seems to be a useful tool in the identification of CNV without the use of angiography. This better visualisation of RPE and choroidal vasculature at variable depths may help elucidate the pathophysiology of disease and can contribute to the diagnosis and management of chronic CSCR.

PART – II

AIM AND OBJECTIVE

To describe retinal and choroidal findings in involved and uninvolved eye of patients with unilateral acute central serous chorioretinopathy with a focus on pachychoroid spectrum of disorders using SS OCT.

MATERIALS AND METHODS

This study was conducted at vitreo retinal clinic in Aravind eye hospital, Madurai.

STUDY DESIGN

This is a prospective observational study to assess retinal and choroidal findings in patients with unilateral acute central serous chorioretinopathy presenting to our vitreo-retinal clinic in Aravind eye hospital, Madurai.

DURATION OF STUDY

Twelve months from December 2017 to November 2018.

SAMPLE SIZE

Sample size of 127 patients were included in this study to assess the retinal and choroiSdal findings in unilateral acute CSCR with focus on pachychoroidal spectrum using SS OCT with 5% precision error and 95% confidence interval.

INCLUSION CRITERIA

- Patients with unilateral acute central serous chorioretinopathy
- Age group of 20 to 60 years
- Defective vision <6 weeks

EXCLUSION CRITERIA

- Patients will be excluded if they are having age related macular degeneration, diabetic retinopathy, hereditary macular degenerations, choroidal neovascular membrane of any other etiology and inflammatory conditions of either eye.
- Refractive error of > -6D
- Patients with media opacities
- Patients, who underwent any previous vitreoretinal surgery, lasers and intravitreal injections.

METHODOLOGY

All patients were asked for history and risk factors such as diabetes, hypertension, steroid usage, smoking, type A personality and pregnancy.

All the patients underwent a thorough ophthalmic examination which included:

Snellen best corrected visual acuity

Non-contact tonometry

Fundus examination with +90D lens with slit lamp biomicroscopy

Indirect ophthalmoscopy

Swept source OCT using Triton-3D macular scan and 5 line Raster scan was taken for both affected and fellow eyes.

In SS OCT we assessed the following retinal and choroidal findings.

1. Subretinal fluid

2. Pigment epithelial detachment

3. RPE irregularity

4. Subretinal fibrin

5. Double layer sign

6. IS OS disruption

7. Thinning of choriocapillaris

8. Pachyvessels

9. Measurement of Choroidal thickness, pigment epithelial detachment height and neurosensory detachment height was done.

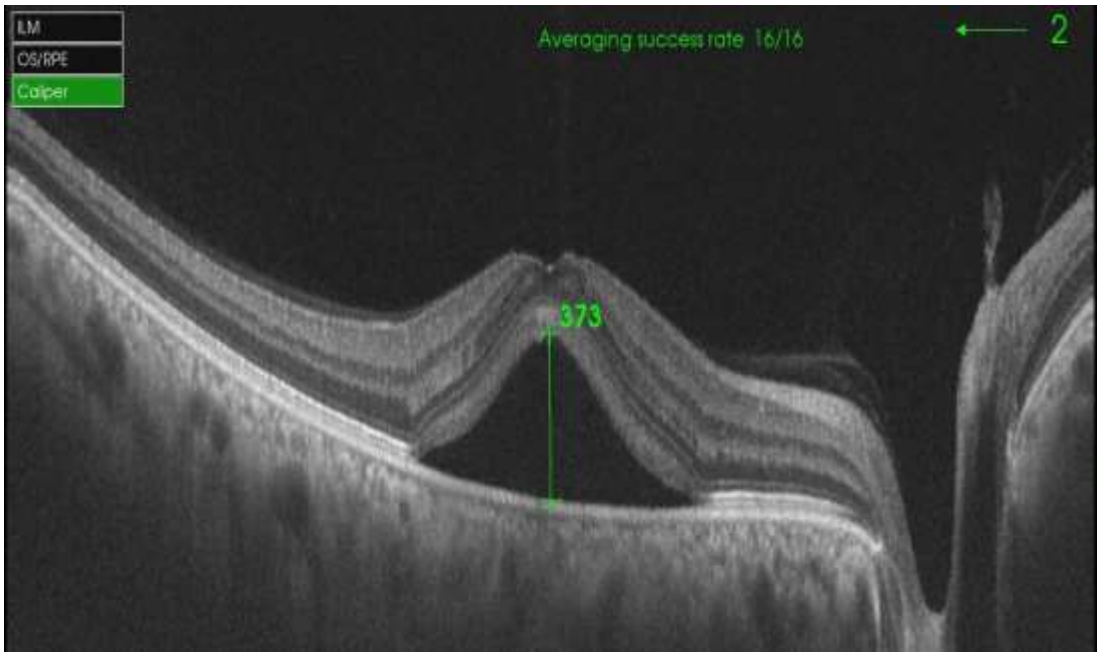


Figure 1 Subretinal fluid in CSCR eye

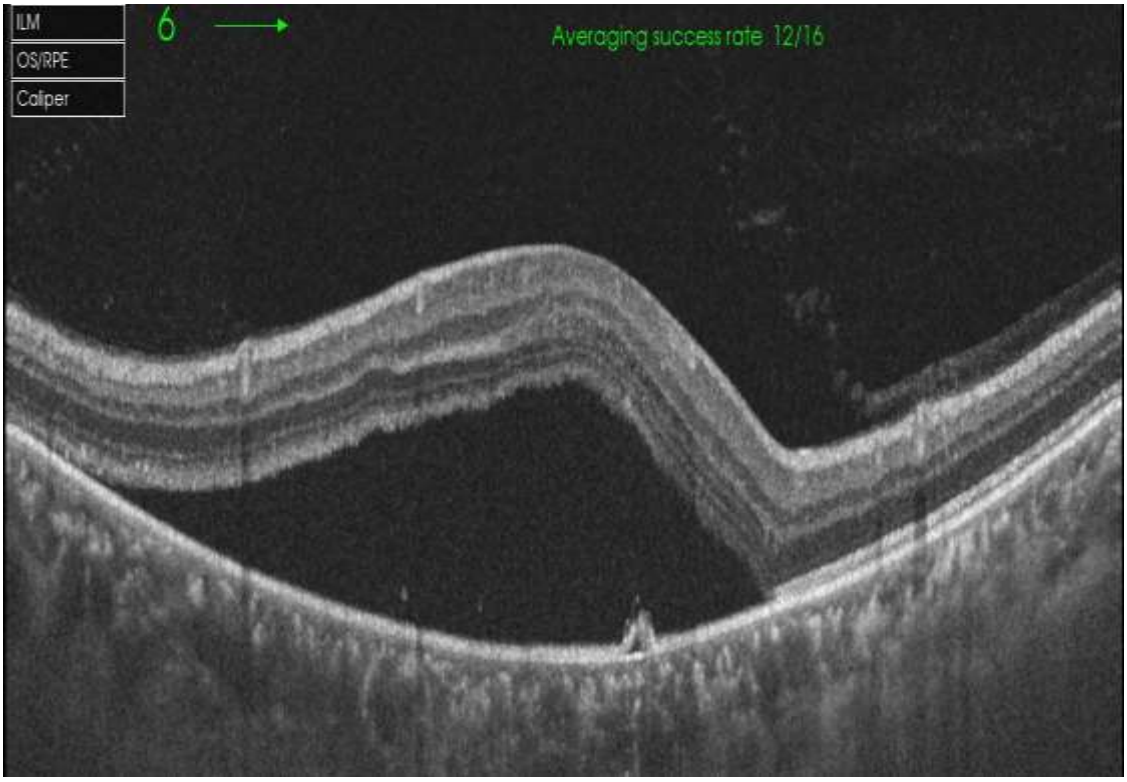


Figure 2 Subretinal fluid with pigment epithelial detachment in affected eye.

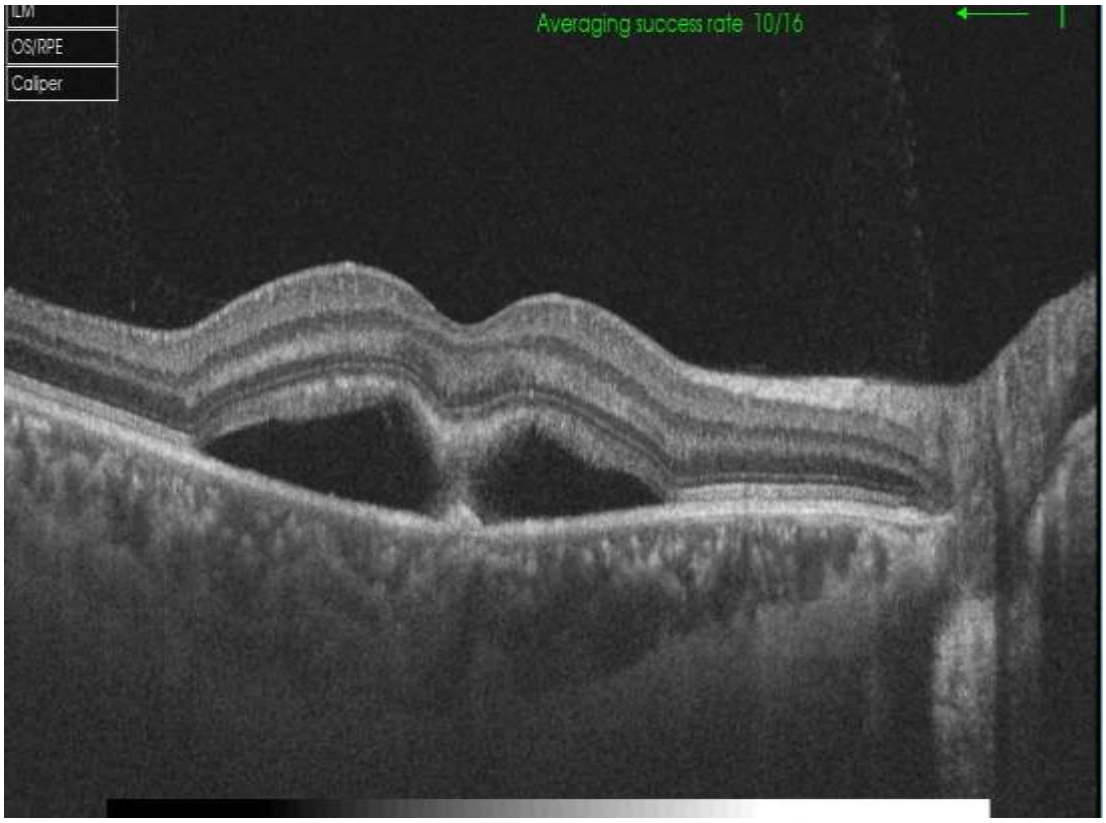


Figure 3 Subretinal fibrin in CSCR

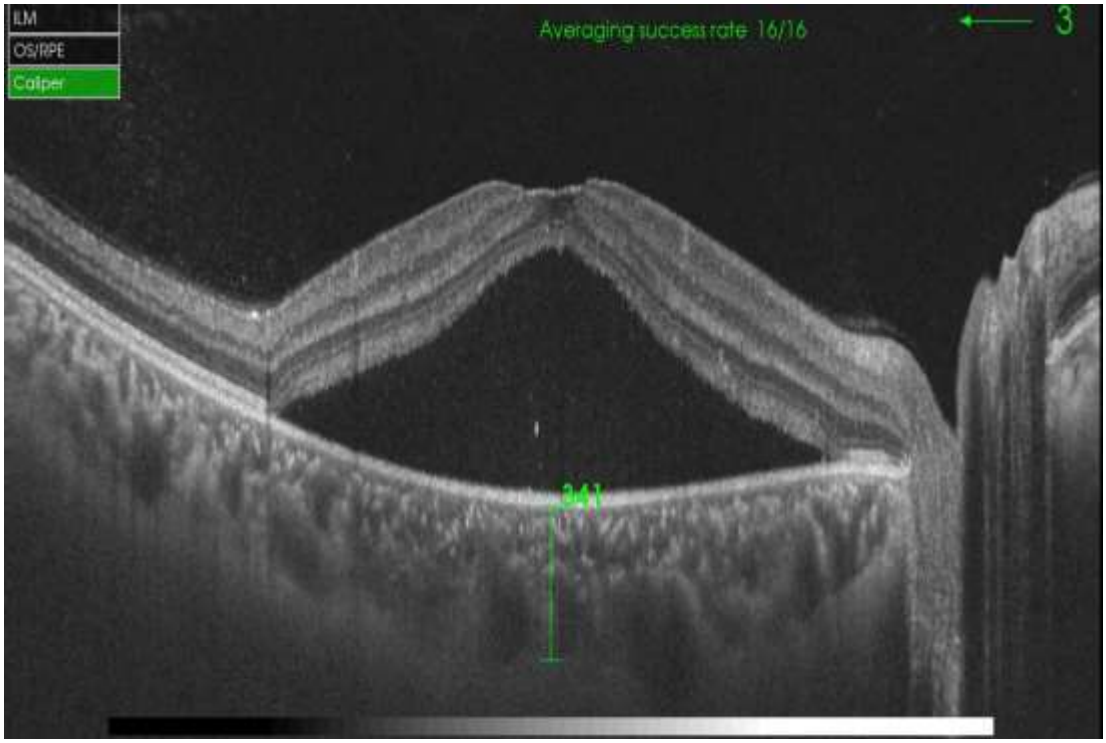


Figure 4 showing choroidal thickness measurement in CSCR eye. Choroidal thickness is measured from hyperreflective band representing RPE and bruchs to sclerochoroid interface.

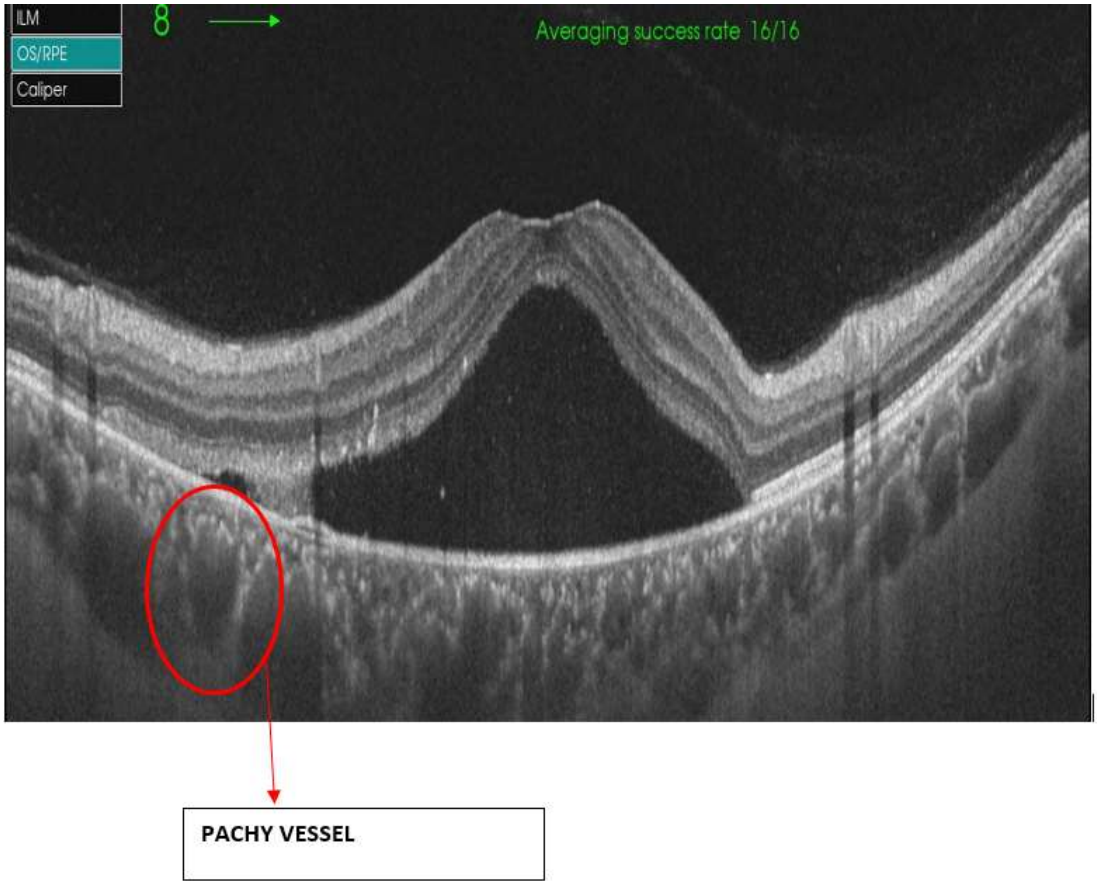


Figure 4 showing CSCR eyes with pachy vessel and thinning of choriocapillaris.

STATISTICAL METHODS

Descriptive variables will be given with Frequency (Percentage) or Mean (Standard deviation). Chi square test or Fisher's exact will be used for finding the association between categorical variables. P value < 0.05 will be considered as statistically significant. All the statistical analysis will be done using statistical software STATA 14.1 (Texas, USA).

RESULTS

Table 2.1 Age distribution in CSCR patients

	N	Mean (SD)	Min-Max
Age in years	127	40(7.11)	23 – 60

The participants ranged from 23 to 60 years with mean age of 40 (7.11) years.

Figure 2.1

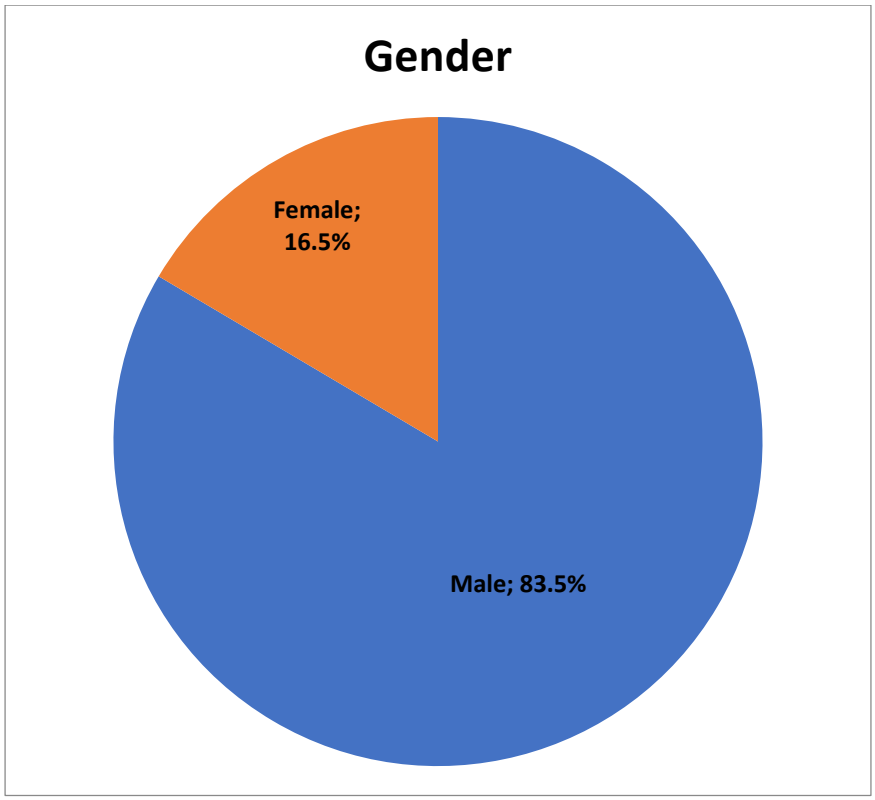


Table 2.2 Sex distribution

GENDER	N(%)
Male	106(83.5)
Female	21(16.5)
Total	127

Among 127 patients, 83.5% (n=106) were males and 16.5% (n=21) were females.

Table 2.3 Laterality of eye

AFFECTED EYE	N(%)
RE	71(56.0)
LE	56(44.0)
Total	127(100)

Out of 127patients, right eye was affected in 56%(n=71) and left eye in 44%(n=56).

Figure 2.2 Laterality

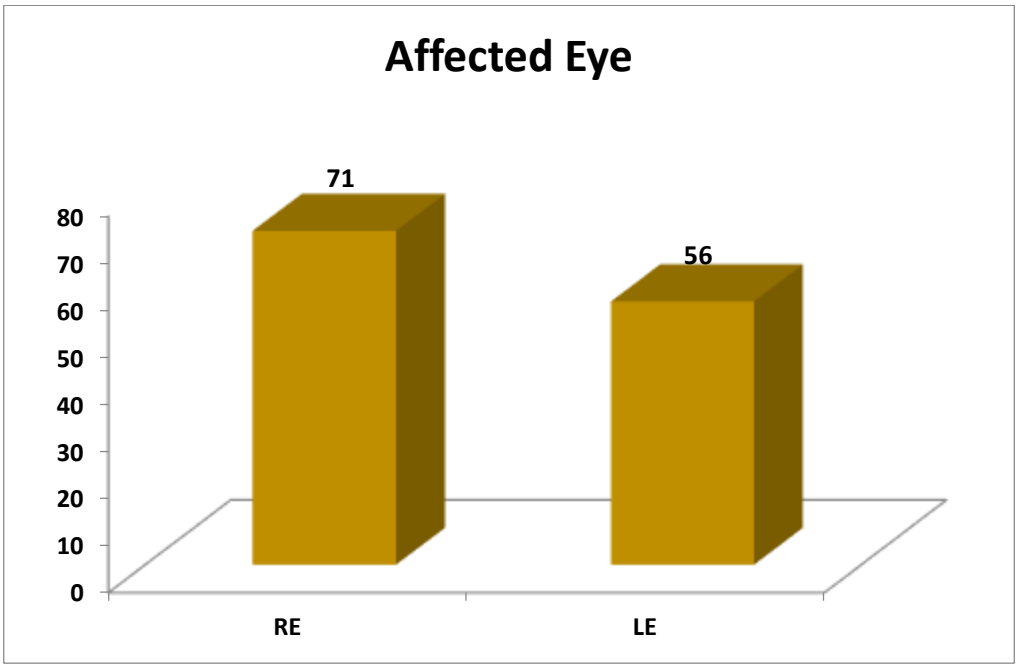
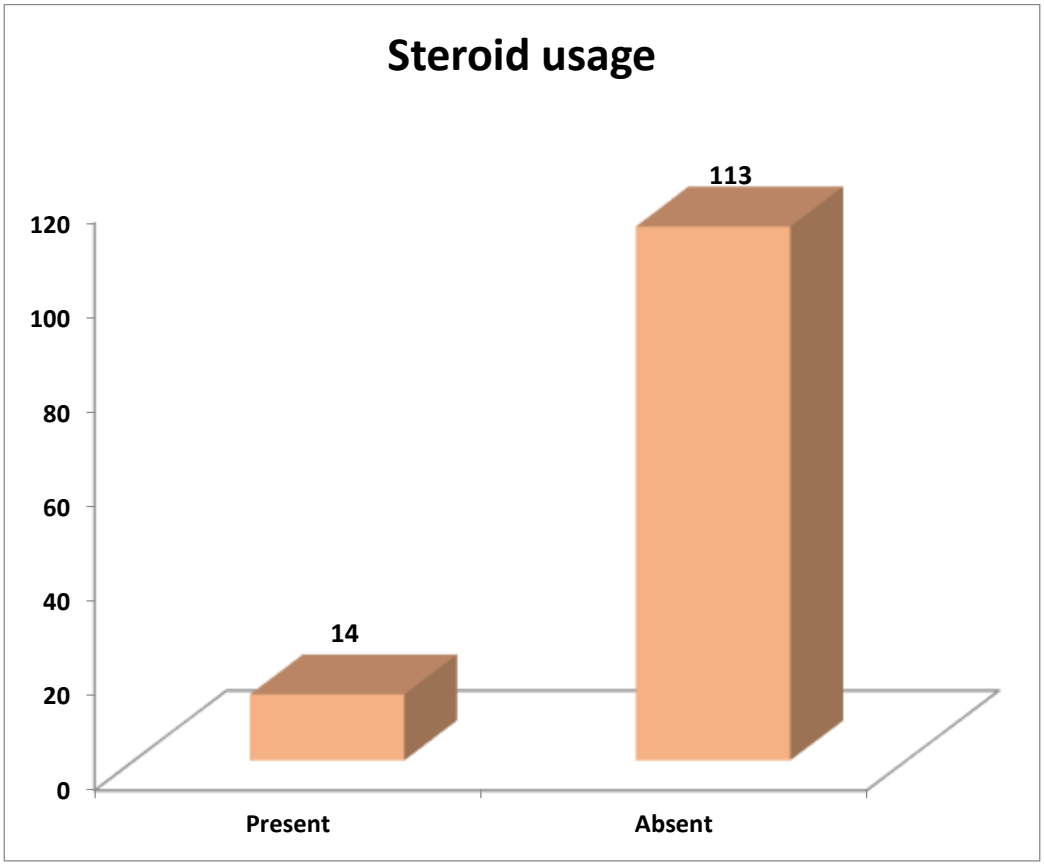


Table 2.4 Systemic illness associated with CSCR

Systemic Illness	Yes n(%)	No n(%)	Total n(%)
Hypertension	12(9.5)	115(90.5)	127(100)
Diabetes	8(6.3)	119(93.7)	127(100)
Asthma	5(3.9)	122(96.1)	127(100)
Steroid Usage	14(11.0)	113(89.0)	127(100)
Smoking	22(17.3)	105(82.7)	127(100)
Personality Type	19(15.0)	108(85.0)	127(100)

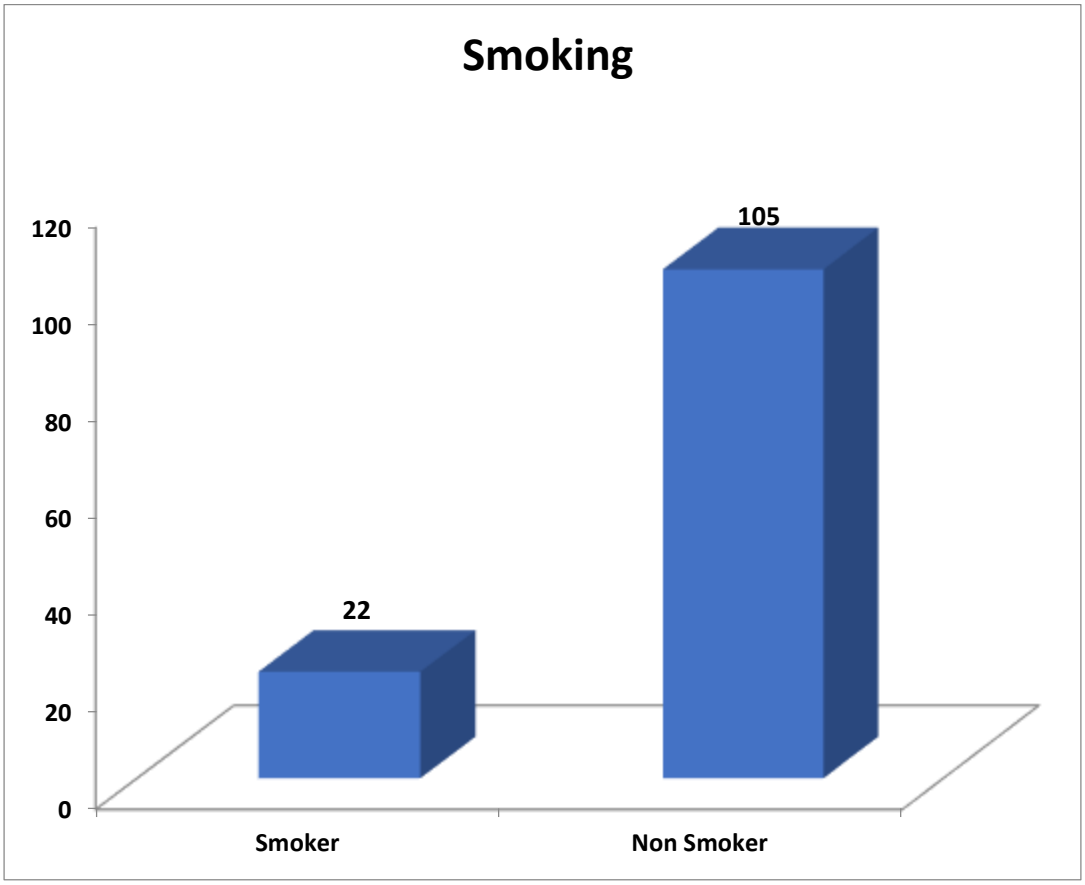
Among 127 patients, 17.3%(n=22) were smokers followed by type A personality 15%(n=19) and steroid usage 11% (n=14).

Figure 2.3 Steroid usage and CSCR



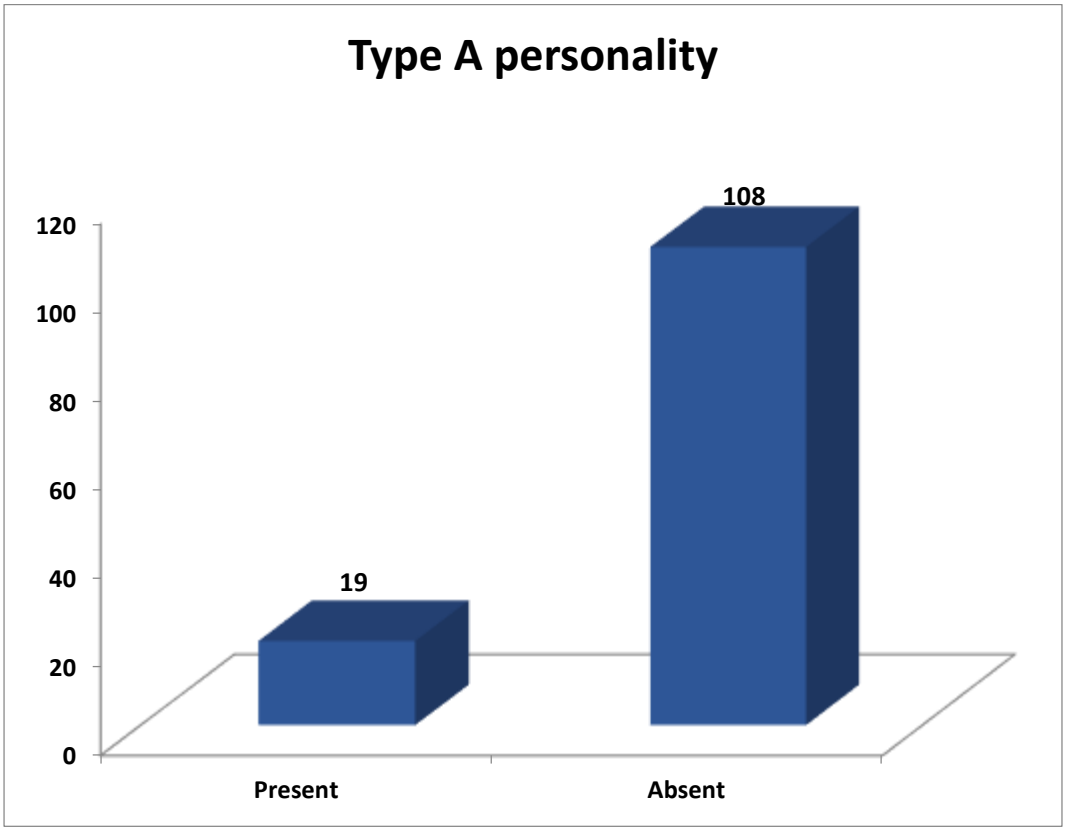
Among 127 patients, steroids usage was seen in 11%(n=14) of patients

Figure 2.4 Smoking and CSCR



Among 127 patients, 17.3% of patients were smoker and 82.7% were non smoker.

Figure 2.5 Type A personality and CSCR



Among 127 patients, Type A personality was found in 15% (n=19) of patients.

Table 2.5 Visual acquity

Visual Acuity	CSCR		P-value@
	Affected eye	Normal eye	
UCVA n Median(IQR)	 127 6/12(6/9 – 6/24)	 127 6/6(6/6 – 6/6)	 <0.001
BCVA n Median(IQR)	 127 6/12(6/6 – 6/18)	 127 6/6(6/6 – 6/6)	 <0.001

@Mann-Whitney U test

The mean visual acquity in CSCR affected eyes was 6/12 and normal eyes was 6/6.

Table 2.6 RPE irregularity in affected and normal eyes

RPE Irregularity	AFFECTED EYE n(%)	NORMAL EYE n(%)	Total n(%)	p-value*
Present	26(20.5)	11(8.7)	37(14.6)	0.008
Absent	101(79.5)	116(91.3)	217(85.4)	
Total	127	127	254	

*Chi-square test

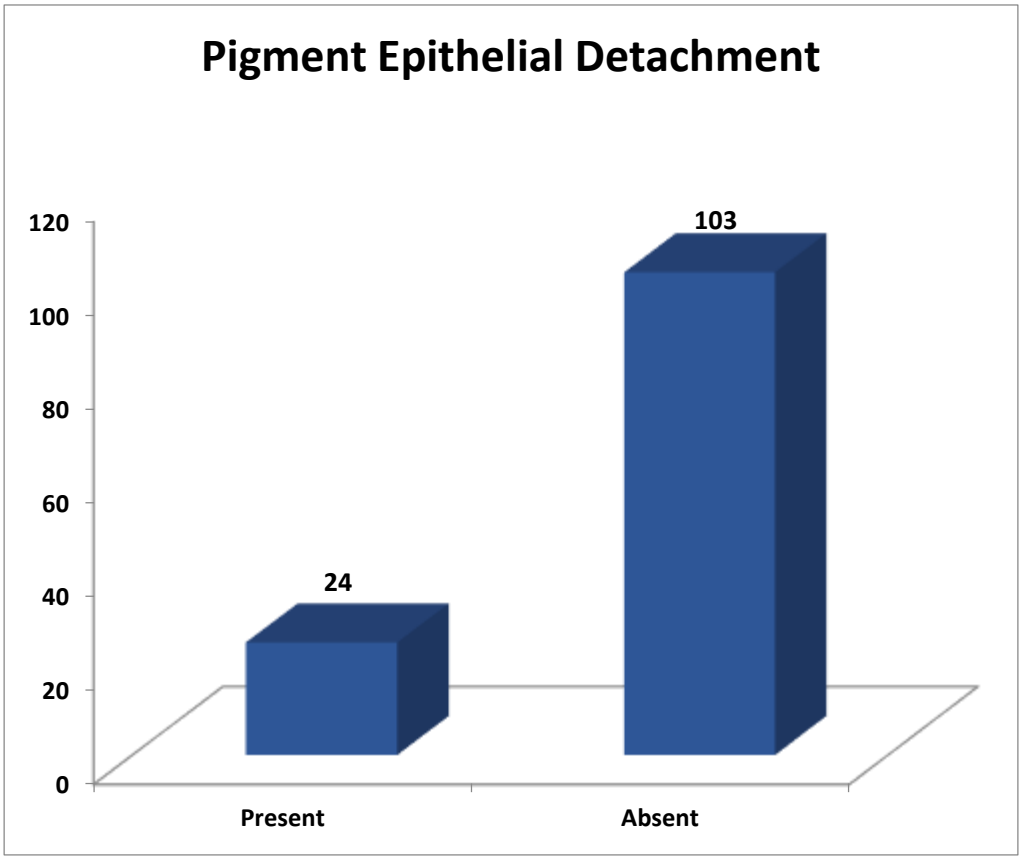
Among 127 patients, RPE irregularities were seen in 20.5% of affected eyes and 8.7% of normal eyes. The p-value 0.008(<0.05) shows that there is an association between the RPE irregularity and CSCR eye.

Table 2.7 Pigment epithelial detachment in affected eyes and normal eyes

PIGMENT EPITHELIAL DETACHMENT	AFFECTED EYE n(%)	NORMAL EYE n(%)
Present	73(57.5)	24(18.9)
Absent	54(42.5)	103(81.1)
Total	127	127

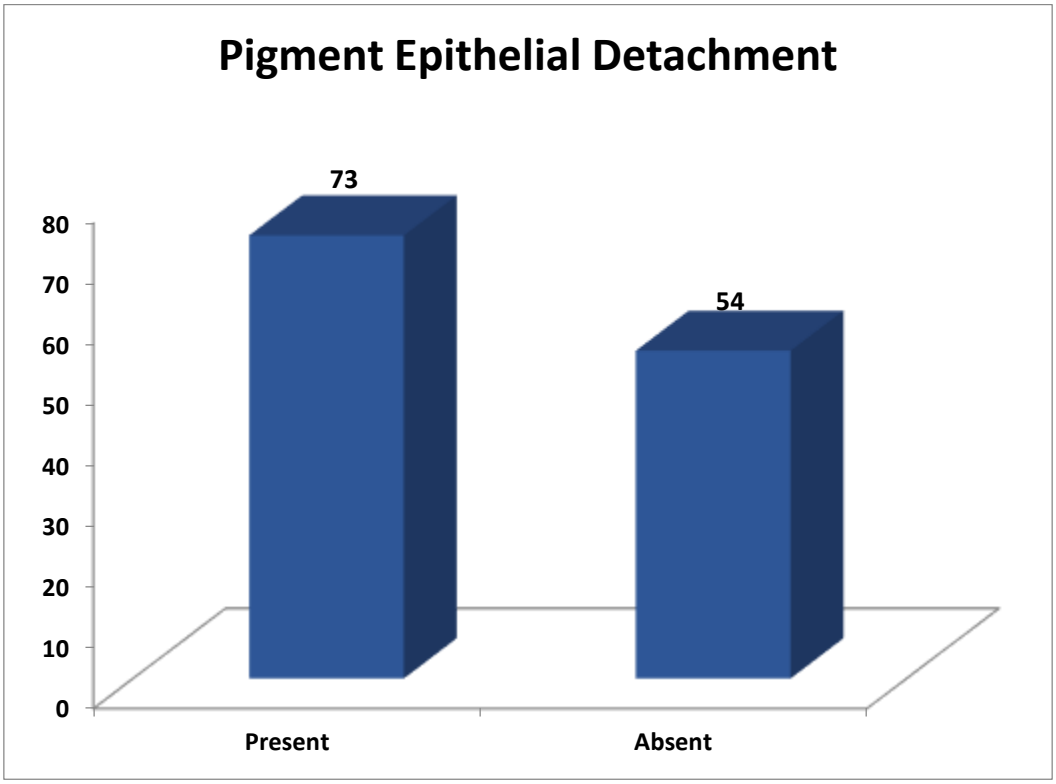
Among 127 participants,57.5% (n=73) patients had PED in affected eyes and 18.9%(n=24) had PED in normal eyes.

Figure 2.6 PED in unaffected eyes



Among 127 unaffected eyes,24 eyes (18.9%) had PED.

Figure 2.7 PED in affected eyes



Among 127 CSCR eyes, 73 (57.5%) eyes had PED.

Figure 2.8 Subretinal fibrin in CSCR eyes

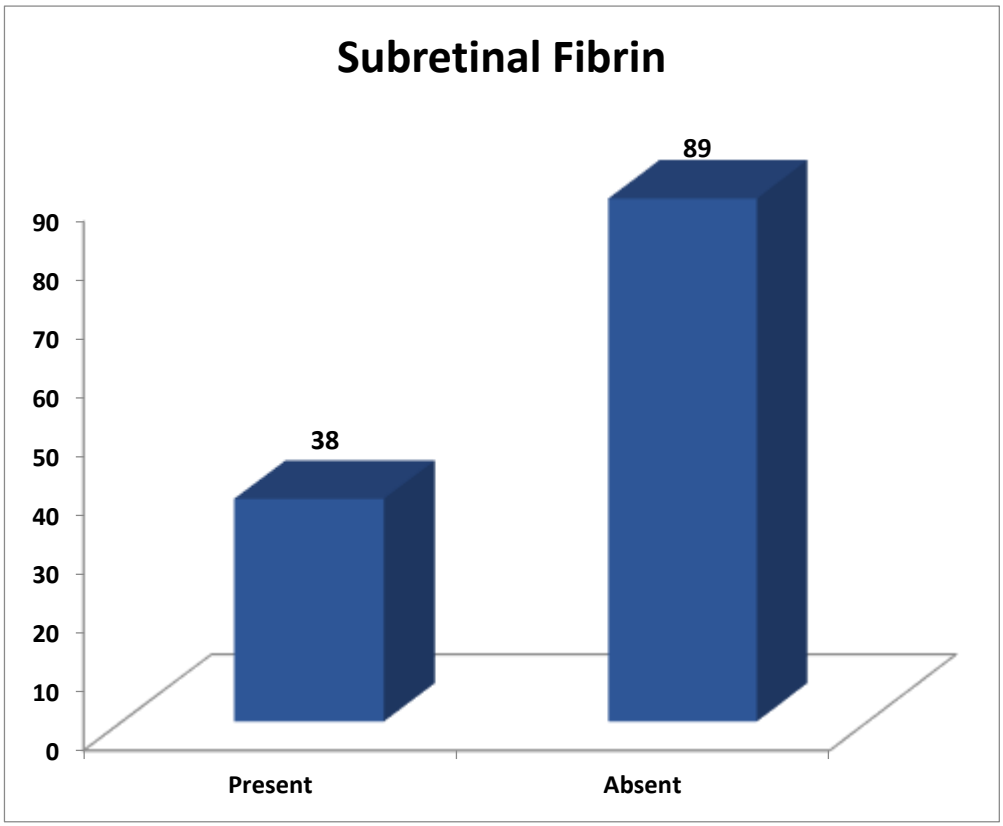


Table 2.8 Subretinal fibrin

SUBRETINAL FIBRIN	AFFECTED EYE n(%)
Present	38(29.9)
Absent	89(70.1)
Total	127

Out of 127 CSCR eyes, 29.9%(n=38) eyes had subretinal fibrin.

Table 2.9 IS OS disruption in CSCR eyes

IS OS DISRUPTION	AFFECTED EYE n(%)
Present	-
Absent	127(100)
Total	127

Among 127 patients with acute CSCR, IS OS disruption is not seen in affected eye of any patients.

Table 2.10 Double layer sign in CSCR eyes

DOUBLE LAYER SIGN	AFFECTED EYE n(%)
Present	2(1.6)
Absent	125(98.4)
Total	127

Among 127 participants, double layer sign was present in 1.6% (n=2) patients in affected eyes.

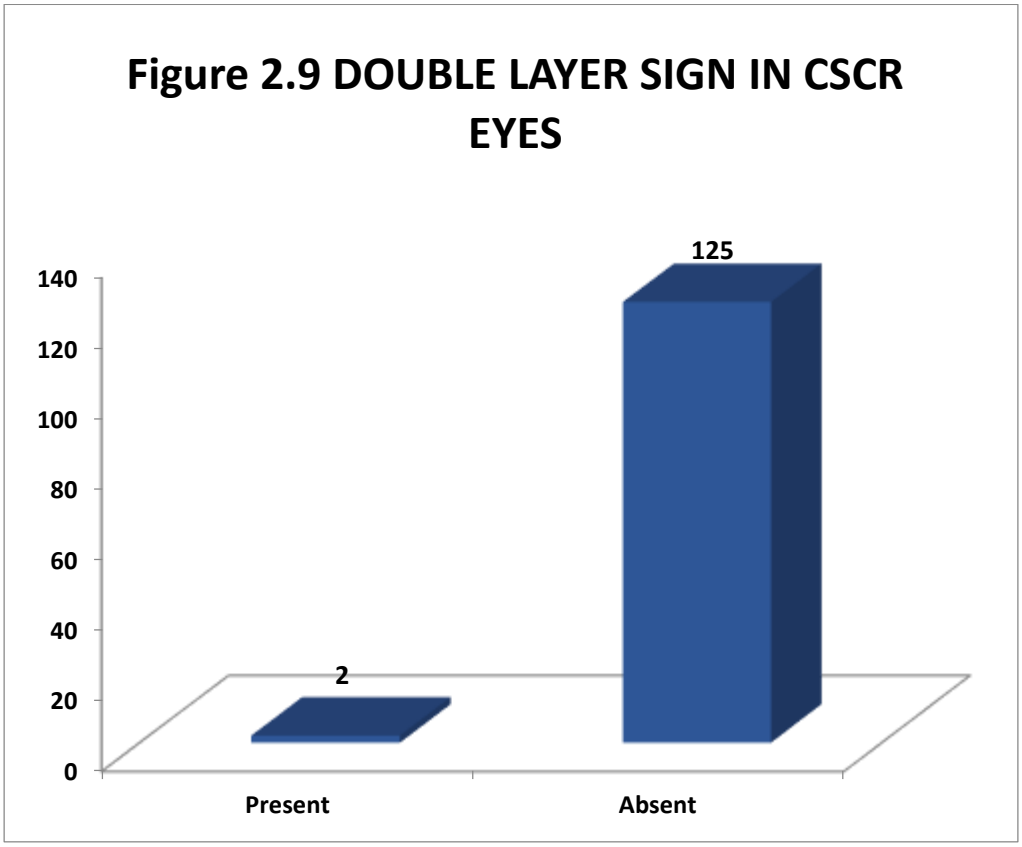


Table 2.11 Mean subfoveal choroidal thickness

CSCR	Choroidal Thickness(µm)			P-value#
	N	Mean (SD)	Min-Max	
Affected eye	127	431.6(73.62)	238 – 620	0.001
Normal eye	127	387.51(65.4)	267 – 553	

#Independent t – test

Among 127 patients, Mean SFCT in CSCR eyes was 431.6 µm (SD±73.62) and 387.51µm(SD±65.4) in normal eyes. The p-value 0.001 (<0.05) shows that there is a significant difference in choroidal thickness between the affected and normal eye.

Table 2.12 Choroidal thickness in CSCR eyes and normal eyes

CSCR	CHOROIDAL THICKNESS n(%)		Total	P-value@
	<300µm	≥300µm		
Present (Affected eye)	2(1.6)	125(98.4)	127(100)	0.010
Absent (Normal eye)	11(8.7)	116(91.3)	127(100)	
Total	13(5.1)	241(94.9)	254(100)	

@chi –Square test

Among 127 patients, choroid thickness of ≥300µm was seen in 98.4%(n=125) eyes with CSCR and 91.3%(n=116) in normal eyes. The p-value 0.010 (<0.05) shows that there is an association between the choroidal thickness and CSCR eyes.

Table 2.13 Pachyvessel in CSCR and normal eyes

CSCR	PACHYVESSEL n(%)		Total	P-value@
	Present	Absent		
Present (Affected eye)	32(25.2)	95(74.8)	127(100)	0.001
Absent (Normal eye)	8(6.3)	119(93.7)	127(100)	
Total	40(15.8)	214(84.2)	254(100)	

@chi –Square test

Among 127 patients, pachyvessel were found in 25.2%(n=32) eyes with CSCR and 6.3% (n=8) of normal eyes. The p-value 0.001 (<0.05) shows that there is an association between the pachy vessel and CSCR eyes.

Table 2.14 Thinning of choriocapillaries in CSCR and normal eyes

CSCR	THINNING OF CHORIOCAPILLARIES n(%)		Total	P-value@
	Present	Absent		
Present (Affected eye)	31(24.4)	96(75.6)	127(100)	0.001
Absent (Normal eye)	9(7.1)	118(92.9)	127(100)	
Total	40(15.8)	214(84.2)	254(100)	

@chi –Square test

Among 127 patients, thinning of choroidal capillaries were seen in 24.4% (n=31) eyes with CSCR and 7.1% (n=9) in normal eyes. The p-value 0.001 (<0.05) shows that there is an association between the thinning of choriocapillaris and CSCR eyes.

Table 2.15 Gender and pachyvessel

GENDER	PACHYVESSEL n(%)		Total	P-value*
	Present	Absent		
Male	35(16.5)	177(83.5)	212(100)	0.454
Female	5(11.9)	37(88.1)	42(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

The p-value 0.454 (>0.05) shows that there is no association between the pachy vessel and gender.

Table 2.16 Age and pachyvessel

AGE (in years) {Mean=40.0}	**PACHYVESSEL n(%)**		**Total**	**P-value***
	Present	Absent		
≥ Mean age	25(19.5)	103(80.5)	128(100)	0.095
< Mean age	15(11.9)	111(88.1)	126(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

The p-value 0.095 (>0.05) shows that there is no association between the pachy vessel and age in years.

Table 2.17 Steroid usage and pachyvessel

STEROID USAGE	PACHYVESSEL n(%)		Total	P-value#
	Present	Absent		
Present	7(25.0)	21(75.0)	28(100)	0.17
Absent	33(14.6)	193(85.4)	226(100)	
Total	40(15.8)	214(84.2)	254(100)	

#Fisher's exact test

Among 127 patients,25% of steroid users had pachyvessels in affected eyes and 14.6% of normal eyes showed pachyvessel. The p-value 0.17 (>0.05) shows that there is no association between the pachyvessel and steroid usage.

Table 2.18 Smoking and pachyvessel

SMOKING	PACHYVESSEL n(%)		Total	P-value*
	Present	Absent		
Yes	5(11.4)	39(88.6)	44(100)	0.38
No	35(16.7)	175(83.3)	210(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

Among 127 patients, pachyvessels were seen in 11.4% of patients who were smokers and 16.7% in non - smokers. The p-value 0.38 (>0.05) shows that there is no association between the pachyvessel and smoking.

Table 2.19 Type A personality and pachyvessel

TYPE A PERSONALITY	PACHYVESSEL n(%)		Total	P-value*
	Present	Absent		
Yes	8(21.0)	30(79.0)	38(100)	0.33
No	32(14.8)	184(85.2)	216(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

Among 127 patients, pachyvessels were seen in 21% of patients with type A personality and 14.8% of normal patients. The p-value 0.33 (>0.05) shows that there is no association between the pachyvessel and personality type.

Table 2.20 Gender and thinning of choriocapillaries

GENDER	THINNING OF CHORIOCAPILLARIES n(%)		Total	P-value*
	Present	Absent		
Male	33(15.6)	179(84.4)	212(100)	0.858
Female	7(16.7)	35(83.3)	42(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

The p-value 0.858 (>0.05) shows that there is no association between the thinning of choriocapillaris and gender.

Table 2.21 Age and thinning of choriocapillaries

AGE (in years) {Mean=40.0}	THINNING OF CHORIOCAPILLARIES n(%)		Total	P-value*
	Present	Absent		
≥ Mean age	22(17.2)	106(82.8)	128(100)	0.526
< Mean age	18(14.3)	108(85.7)	126(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

The p-value 0.526 (>0.05) shows that there is no association between the thinning of choriocapillaris and age in years.

Table 2.22 Steroid usage and thinning of choriocapillaries

STEROID USAGE	THINNING OF CHORIOCAPILLARIES n(%)		Total	P-value#
	Present	Absent		
Present	8(28.6)	20(71.4)	28(100)	0.057
Absent	32(14.2)	194(85.8)	226(100)	
Total	40(15.8)	214(84.2)	254(100)	

#Fisher's exact test

The p-value 0.057 (>0.05) shows that there is no association between the thinning of choriocapillaris and steroid usage.

Table 2.23 Smoking and thinning of choriocapillaries

SMOKING	THINNING OF CHORIOCAPILLARIES n(%)		Total	P-value*
	Present	Absent		
Yes	6(13.6)	38(86.4)	44(100)	0.672
No	34(16.2)	176(83.8)	210(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

The p-value 0.672 (>0.05) shows that there is no association between the thinning of choriocapillaries and smoking.

Table 2.24 Type A personality and thinning of choriocapillaries

TYPE A PERSONALITY	THINNING OF CHORIOCAPILLARIES n(%)		Total	P-value*
	Present	Absent		
Yes	8(21.0)	30(79.0)	38(100)	0.33
No	32(14.8)	184(85.2)	216(100)	
Total	40(15.8)	214(84.2)	254(100)	

*Chi-Square test

The p-value 0.33 (>0.05) shows that there is no association between the thinning of choriocapillaris and personality type.

Table 2.25 Gender and choroidal thickness

GENDER	CHOROIDAL THICKNESS n(%)		Total	P-value#
	<300µm	≥300µm		
Male	11(5.2)	201(94.8)	212(100)	>0.999
Female	2(4.8)	40(95.2)	42(100)	
Total	13(5.1)	241(94.9)	254(100)	

#Fisher's exact test

The p-value 0.999 (>0.05) shows that there is no association between the choroidal thickness and gender.

Table 2.26 Age and choroidal thickness

AGE (in years) {Mean=40.0}	CHOROIDAL THICKNESS n(%)		Total	P-value*
	<300µm	≥300µm		
≥ Mean age	8(6.3)	120(93.7)	128(100)	0.409
< Mean age	5(4.0)	121(96.0)	126(100)	
Total	13(5.1)	241(94.9)	254(100)	

*Chi-Square test

The p-value 0.409 (>0.05) shows that there is no association between the choroidal thickness and age in years.

Table 2.27 Steroid usage and choroidal thickness

STEROID USAGE	CHOROIDAL THICKNESS n(%)		Total	P-value#
	<300µm	≥300µm		
Present	2(7.1)	26(92.9)	28(100)	0.642
Absent	11(4.9)	215(95.1)	226(100)	
Total	13(5.1)	241(94.9)	254(100)	

#Fisher's exact test

The p-value 0.642 (>0.05) shows that there is no association between the choroidal thickness and steroid usage.

Table 2.28 Smoking and choroidal thickness

SMOKING	CHOROIDAL THICKNESS n(%)		Total	P-value#
	<300µm	≥300µm		
Yes	3(6.8)	41(93.2)	44(100)	0.477
No	10(4.8)	200(95.2)	210(100)	
Total	13(5.1)	241(94.9)	254(100)	

#Fisher's exact test

The p-value 0.477 (>0.05) shows that there is no association between the choroidal thickness and smoking.

Table 2.29 Type A personality and choroidal thickness

TYPE A PERSONALITY	CHOROIDAL THICKNESS n(%)		Total	P-value#
	<300µm	≥300µm		
Yes	3(7.9)	35(92.1)	38(100)	0.420
No	10(4.6)	206(95.4)	216(100)	
Total	13(5.1)	241(94.9)	254(100)	

#Fisher's exact test

The p-value 0.42 (>0.05) shows that there is no association between the choroidal thickness and personality type.

DISCUSSION

Central serous chorioretinopathy is a major cause of vision threat in middle aged indivduals. In 2013 David Warrow and colleagues specified a group of condition with choroidal thickening and retinal pigment epithelial changes with or without corresponding retinal abnormality which is termed as pachychoroid spectrum. CSCR is considered as one of the diseases among this spectrum. This spectrum of disorders has common characteristic features such as increased choroidal thickness, dilated outer choroidal vessel (pachy vessel) and thinning of choriocapillaris. This study mainly aimed to assess the retinal and choroidal findings in unilateral acute CSCR with focus on pachychoroid spectrum.

In a study Margolis et al found that choroid is thickest sub foveally of 287µm (SD±78µm) and thickness decreased rapidly in nasal direction averaged 145µm±57µm 03mm nasal to fovea[51]. Hamzah F Shinjima A et al measured choroidal thickness in CSCR eyes using EDI OCT and SS OCT. With SS OCT they observed that subfoveal choroidal thickness in acute CSCR was 332±96.7µm and that in chronic CSCR was 392.6±101.3µm[77].

Kim et al measured choroidal thickness in affected and unaffected eyes of unilaterally active CSCR[80]. The choroidal thickness of the

affected eyes, unaffected eyes, and normal eyes were analysed using EDI OCT. The mean choroidal thicknesses of the affected eyes, unaffected fellow eyes, and normal individuals were 445.58±100.25, 378.35±117.44, and 266.80±55.45 µm, respectively. Compared with normal eyes, subfoveal choroidal thickness was increased significantly in the eyes with active CSCR and also in the unaffected fellow eyes. The choroidal thickness was significantly greater in the eyes with active CSCR than in the unaffected fellow eyes.

In our study mean subfoveal choroidal thickness in CSCR eyes were 431.6µm (SD ±73.62) compared to normal eyes which was 387.51µm (SD ±65.4).The p-value 0.001 (<0.05) showed that there is a significant difference in choroidal thickness between the affected and normal eyes. In our study choroidal thickness was significantly greater in eyes with CSCR than unaffected fellow eyes. This study did not compare the choroidal thickness of CSCR patients with normal patients. Also in our study we used SS OCT which has better penetration than EDI OCT used in the previous quoted studies.

In a study Kunal k Dansingani et al included 66 eyes of 33 patients with anyone pachychoroid disorders in both eyes and underwent SS OCT. They found that out of 66 eyes, 50 eyes had increased choroidal thickness of >300µm[61].

In our study among 127 patients with unilateral acute CSCR, choroidal thickness of ≥300µm was seen in 98.4% of affected eyes and 91.3% of unaffected eyes. The p value of 0.010 shows that there is an association between choroidal thickness and CSCR eyes.

Lee WJ et al in his study evaluated tomographic features of choroidal vasculature in acute and chronic CSCR using SS OCT and found that 91% of eyes had choroidal vessel dilatation in acute CSCR[33]. In a study by Yang L et al measured choroidal vessel diameter in affected and unaffected eyes of patients with unilateral CSCR using EDI OCT[52]. They found that mean diameter of largest hyporeflective lumen was larger than (305±101µm) than in contralateral unaffected eye(251±98µm).

In our study among 127 patients, dilated vessels were seen in 25.2% of CSCR eyes and 6.3% in normal eyes. The p value <0.001 shows that there is an association between pachy vessels and CSCR eyes. But our study did not measure the exact choroidal vessel diameter.

In our study among 127 patients, thinning of choroidal capillaries were seen in 24.4% (n=31) eyes with CSCR compared to normal eyes which is 7.1%(n=9).The p-value of 0.001 (<0.05) shows that there is an association between the thinning of choriocapillaris and CSCR eyes.

In a study Gupta et al evaluated retinal pigment epithelial changes in affected and unaffected eyes of idiopathic CSCR using SD OCT and found that 94% had RPE bumps and 11.8% had Pigment epithelial detachment in asymptomatic eyes[10].In our study, among 127 patients 57.5% CSCR eyes has pigment epithelial detachment and 18.9% normal eyes has PED.

Kitzmann A S et al in his study found that incidence of CSCR per 100,000 were 9.9 for men and 1.7 for women[3].The incidence is approximately 6 times higher in men than in women. In our study among 127 patients 83.5%(n=106) patients were male and 16.5%(n=21) were female which showed the disease has higher incidence among male population.

Kitzmann et al in his study observed that there is a variability in the reported age of affected patients, reporting a higher mean age than generally assumed, ranging between 39 and 51 years[3].Our study data showed that the patients with CSCR ranged from 23 to 60 years with mean age of 40 years.

Hyewon chung et al reviewed many articles to find out the association of focal choroidal excavation and pachychoroid spectrum using multimodal imaging[62]. They observed that focal choroidal excavation appears to be a manifestation of pachychoroid spectrum

disease associated with choroidal thickening and pachyvessels on structural OCT and choroidal hyperpermeability on indocyanine green angiography. In our study among 127 patients we did not find focal choroidal excavation . Our study showed no IS OS disruption in affected eyes.

In our study, male preponderance, mean age of 40 years, pigment epithelial detachment, RPE irregularities, choroidal thickness of > 300µm, pachyvessel and choriocapillaris showed significant association in CSCR eyes.

We observed that risk factors such as steroid usage, smoking, type A personality showed no association between increased choroidal thickness, pachyvessel and choriocapillaris thinning.

LIMITATION OF THE STUDY

Large sample size needed.

No control group were included in this study.

Follow up studies are needed to assess the progression of disease.

CONCLUSION

In this study, we observed an increase in choroidal thickness of > 300µm, dilated vessels in choroid and thinning of choriocapillaris in both eyes of patients with unilateral acute CSCR. This suggests most often CSCR is found to be a part of pachychoroid spectrum of disorders. The risk factors such as steroid usage, smoking and type. A personality included in this study showed no association between the characteristic findings (increased choroidal thickness, pachyvessels and thinning of choriocapillaris) of pachychoroid spectrum.

Further studies with larger sample size and follow ups may be required to monitor further progression of the disease.

ANNEXURES

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ABBREVIATION

OCT	Optical Coherence Tomography
CSCR	Central Serous Choroidal Retinopathy
PED	Pigment Epithelial Detachment
RPE	Retinal Pigment Epithelium
FFA	Fundus Fluorescein Angiography
FAF	Fundus autofluorescence
ICGA	Indocyanine green angiography
PDT	Photodynamic Therapy
ARMD	Age -related macular degeneration
PCV	Polypoidal Choroidal Vasculopathy
VKH	Vogt – Koyanagi – Harada
CNV	Choroidal Neovascularization
PNV	Pachychoroid pigment neovasculopathy
NSD	Neurosensory retinal detachment
SRD	Serous retinal detachment
PPE	Pachychoroid pigment epitheliopathy
ONL	Outer nuclear layer
SD	Spectral domain
EDI	Enhanced depth imaging
SS	Swept source

PROFORMA

SS OCT findings of pachychoroid spectrum of diseases in patients with unilateral acute CSCR

Study number: **Date: / /**

Name:
Mobile No:

MR No:_____

Age :_____years

Gender : **1. Male** ☐ **2. Female** ☐

Affected eye: **1. RE** ☐ **2. LE** ☐

Duration of defective vision(in days): ☐

Any previous history of CSCR: **1. YES** ☐ **2.NO** ☐

Systemic diseases

1.Hypertension ☐ **2.Diabetes** ☐ **3.Allergy** ☐

4.Asthma ☐ **5.Steroids usage** ☐ **6.Smoking** ☐

7.Type A personality ☐ **8.Others** ☐

Specify_____

OCULAR EXAMINATION
Vision
Snellens chart

	RE	LE
UCVA		
BCVA		

LogMAR chart

UCVA		
BCVA		

IOP(mm of Hg)	**RE**	**LE**

SLIT LAMP EXAMINATION:

Findings	**Right eye**	**Left eye**
Anterior segment		
+90D findings		
Indirect ophthalmoscope		

INVESTIGATIONS:

SS OCT FINDINGS : **YES** [1] **NO** [2]

		RE	**LE**
1	**Subretinal fluid**		
2	**Pigment epithelial detachment** **i Dome shaped** **ii Notched** **iii Tall peaked**		
3	**RPE irregularity**		
4	**Subretinal fibrin**		
5	**IS OS disruption**		
6	**Double layer sign**		
7	**Thinning of choroidal capillaries**		
8	**Pachy vessels**		

Measurements (in µm)	RE	LE
Choroidal thickness		
Height of tallest PED		
Neurosensory detachment height		

CONSENT FORM

Informed Consent form to participate in a clinical study.

Study Title: **Swept source OCT findings of pachychoroid spectrum of diseases in patients with unilateral acute Central Serous Chorioretinopathy**

Protocol Number:

Subject's Name: _____

Subject's Initials: _____

Subject ID No: _____

		Please put initial in the box (Subject)
(i)	I confirm that I have understood the information about the study, procedures and treatments for the above study and have had the opportunity to ask questions and I received satisfactory answers to all of my questions. I have been given a copy of the informed consent form to take home	[ ]
(ii)	I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. However, this is may not be possible for certain surgical procedures	[ ]

(iii)	I understand that the Investigator of the study to access my health records for the research purpose. However, I understand that my identity will not be revealed in any information released to third parties or published.	[ ]
(iv)	I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s)	[ ]
(v)	I agree to take part in the above study.	[ ]

Signature (or Thumb impression) of the Subject:

Subject's Name:

Date:

Signature (or Thumb impression) of

Legally Acceptable Representative (LAR):

Date:

Signature of the Investigator:

Investigator's Name:

Date:

Signature of the Witness:

Name of the Witness:

Date:

ARAVIND MEDICAL RESEARCH FOUNDATION
Institutional Ethics Committee
(REGISTRATION NO. ECR/182/INST/TN/2013 DATED 20.04.2013)

20th December 2017

To
Dr.V.R.SARANYA
MS Resident
Aravind Eye Hospital
Madurai

Dear Dr.Saranya,

Thesis Title: Evaluation of the efficacy of Botulinum Toxin in the Treatment of Acute Sixth Nerve Palsy (Prospective observational study)

IEC Code: IEC201800261

Thank you for submitting your thesis and seeking the approval from the ethics committee. The documents provided by you for consideration which include the thesis protocol and informed consent forms were reviewed for the research methodology and scientific content. The Ethical committee did not find any correction and has recommended the thesis to go ahead in the present form.

Thanking you

Yours Sincerely,

Dr.R.Sharmila
Member Secretary
Institutional Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
ARAVIND MEDICAL RESEARCH FOUNDATION
No.1, Anna Nagar, Madurai-625 020

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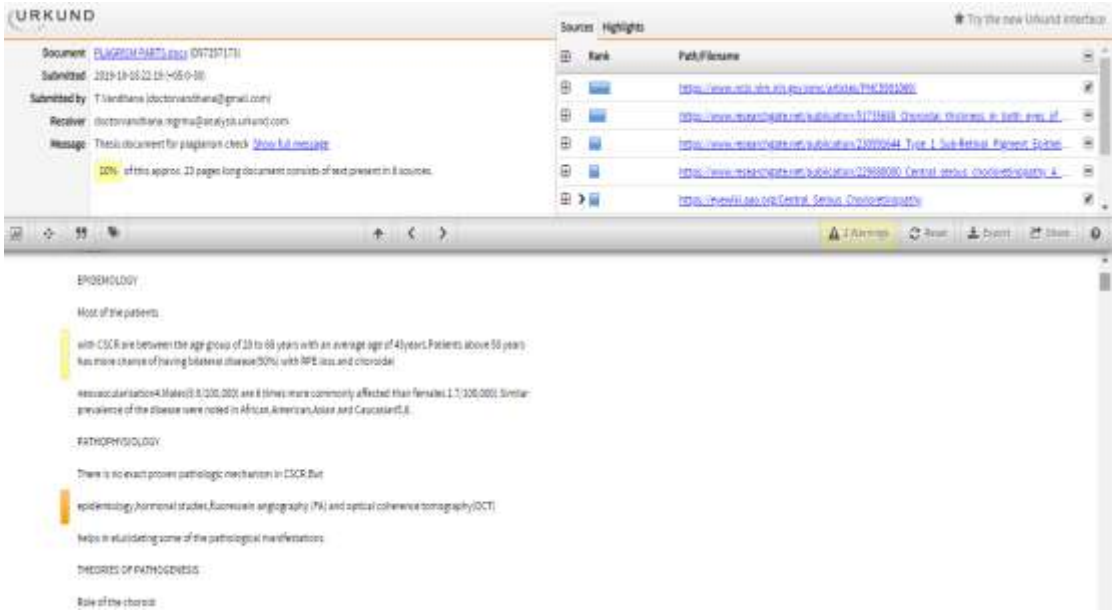
Significance:	10 %

Sources included in the report:

https://eyewiki.aao.org/Central_Serous_Chorioretinopathy
https://latinamericanjo.com/pachychoroid-as-a-biomarker-using-optical-coherence-tomography-swept-source-in-central-serous-choroidopathy
https://www.researchgate.net/publication/51735688_Choroidal_thickness_in_both_eyes_of_patients_with_unilaterally_active_central_serous_chorioretinopathy
https://www.researchgate.net/publication/230591644_Type_1_Sub-Retinal_Pigment_Epithelial_Neovascularization_in_Central_Serous_Chorioretinopathy_Masquerading_As_Neovascular_Age-Related_Macular_Degeneration
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5991069/
https://www.researchgate.net/publication/229080000_Central_serous_chorioretinopathy_A_review_of_epidemiology_and_pathophysiology

Instances where selected sources appear:

35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Rank
Path/Filename
EPIDEMIOLOGY
Most of the patients
with CSCR are between the age group of 20 to 60 years with an average age of 40 years.Patients above 50 years has more chance of having bilateral disease(50%) with RPE loss and chloroidal
neovascularisation.4.Males(3.8/100,000) are 8 times more commonly affected than females (1.7/100,000).Similar prevalence of the disease were noted in African,American,Asian and Caucasian(5,6
PATHOPHYSIOLOGY
There is no exact proven pathologic mechanism in CSCR.But
epidemiology,hormonal studies,fluorescein angiography (FA) and optical coherence tomography (OCT)
helps in elucidating some of the pathological manifestations
THEORIES OF PATHOGENESIS
Role of the choroid

S.NO	NAME	AGE (IN YEARS)	SEX	MR NUMBER	FECTED E	DURATION OF DEFECTIVE VISION IN DAYS	OUS HISTORY OF	SYSTEMIC DISEASE/STEROID USAGE/SMOKING	SNELLENS CHART UCVA (RE)	SNELLENS CHART BCVA(RE)	LOGMAR CHART UCVA(RE)	LOGMAR CHART BCVA(RE)	SNELLENS CHART UCVA (LE)	SNELLENS CHART BCVA(LE)	LOGMAR CHART UCVA(LE)	LOGMAR CHART BCVA(LE)	IOP		SLIT LAMP FINDINGS				SS OCT FINDINGS																MEASUREMENTS(µ)					
																			ANTERIOR SEGMENT		90 D and IO FINDINGS		JBRETINAL FLUI		PIGMENT EPITHELIAL DETACHMENT		RPE IRREGULARITY		SUBRETINAL FIBRIN		OS DISRUPTI		JBLE LAYER S		THINNING OF CHORODIAL CAPILLARIES		PACHY VESSELS		FOCAL CHOROIDAL EXCAVATION		CHOROIDAL THICKNESS		HEIGHT OF TALLEST PED		NEUROSENSORY DETACHMENT HEIGHT	
																	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
1	RAMESH	33	M	P4841195	RE	30	NO	SMOKING	6|9	6|9	0.20	0.20	6|6	6|6	0.00	0.00	14	14	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	1	2	2	2	2	2	2	1	2	1	2	2	2	527	489	..	..	261	..
2	PALANI KUMAR	46	M	P4841480	RE	7	NO	HTN,DM	6|18	6|12	0.50	0.30	6|6	6|6	0.00	0.00	17	19	NORMAL	NORMAL	FR-ABSENT-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2	2	468	405	160	..	682	..
3	SUJITH	40	M	P4846745	RE	10	NO	GOUT	6|12	6|12	0.30	0.30	6|6	6|6	0.00	0.00	16	16	NORMAL	NORMAL	FR-SRF-CSCR	FR-PRESENT	1	2	1	2	2	2	2	2	2	2	2	2	1	2	1	2	2	2	441	322	53	..	282	..
4	SARANYA	32	F	P4841835	LE	21	NO	STEROID USAGE	6|6	6|6	0.00	0.00	6|36	6|36	0.80	0.80	16	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	1	2	2	1	2	2	2	2	2	2	2	1	2	2	394	399	..	..	70	..	400
5	GURUNATHAN	38	M	P4846745	LE	3	NO	ASTHMA/STEROID U	6|6p	6|6p	0.00	0.00	6|12p	6|12p	0.30	0.30	17	15	NORMAL	NORMAL	PED	FR-DULL-CSCR	2	1	1	1	2	2	2	2	2	2	2	2	1	1	1	1	2	2	521	574	170	173	..	..	399
6	JYYANAN	34	M	F1965731	RE	10	NO	NIL	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	13	12	NORMAL	NORMAL	FR-DULL-CSCR	NORMAL	1	2	2	2	1	1	2	2	2	2	2	2	1	2	1	2	2	2	378	389	..	..	271	..	
7	MURALIDHAR	51	M	P4833867	LE	30	NO	NIL	6|12P	6|6	0.30	0.00	6|36P	6|18P	0.80	0.50	15	14	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	426	476	..	108	..	..	298
8	KRISHNARAJ	44	M	P4854587	RE	15	NO	NIL	6|18P	6|18P	0.50	0.50	6|12P	6|6	0.30	0.00	12	13	NORMAL	NORMAL	FR-absent-CSCR	PED	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	581	400	112	128	789	..	
9	GURUSAMY	33	M	P4844384	LE	2	NO	HTN	6|6	6|6	0.00	0.00	6|9	6|9	0.20	0.20	18	19	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	436	378	..	40	..	351	
10	BOOPATHIRAJ	33	M	F1967782	RE	25	NO	SMOKING	6|24	6|6P	0.60	0.00	6|18	6|6P	'0.50	0.00	11	14	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	463	452	..	..	346	..	
11	LAKSHMANAN	39	M	P4846163	RE	20	NO	RHEUMATOID ARTH	6|12P	6|12P	0.30	0.30	6|6	6|6	0.00	0.00	16	14	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	442	426	38	..	240	..	
12	ALEXANDER	34	M	F1933591	LE	30	NO	SMOKING	6|12P	6|6	0.30	0.00	6|24	6|6P	0.60	0.00	17	14	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	474	482	..	70	..	378	
13	POOMURUGAN	31	M	F1699421	RE	7	NO	NIL	6|9P	6|9P	0.20	0.20	6|6	6|6	0.00	0.00	15	14	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	470	453	74	..	229	..	
14	THANGARAJ	42	M	F1969873	RE	10	NO	CARDIAC/SMOKING	5|60	5|60	1.00	1.00	6|6	6|6	0.00	0.00	15	15	NORMAL	NORMAL	FR-absent-CSCR	PED	1	2	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	391	378	50	108	662	..	
15	CHANDRA SEKARAN	37	M	P4853259	RE	20	NO	SMOKING	6|9	6|9	0.20	0.20	6|6	6|6	0.00	0.00	15	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	1	1	2	2	2	2	2	1	2	2	2	2	2	468	447	113	..	303	..	
16	SARAVANAN	39	M	P4364734	LE	15	NO	SMOKING	6|6	6|6	0.00	0.00	6|6P	6|6p	0.00	0.00	10	13	NORMAL	NORMAL	RPE CHANGES	FR-ABSENT-CSCR	2	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	406	415	..	..	..	240	
17	NARAYANAN	36	M	P4730146	RE	1	NO	NIL	6|6P	6|6P	0.00	0.00	6|6	6|6	0.00	0.00	15	14	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	347	394	84	..	149	..	
18	SELVARANI	41	F	P4858052	LE	20	NO	NIL	6|12P	6|6	0.20	0.00	6|36	6|24	0.80	0.60	19	20	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	415	458	..	178	..	591	
19	MEYGNANAMURTHY	30	M	P4859036	RE	3	NO	NIL	6|9	6|6	0.20	0.00	6|6	6|6	0.00	0.00	13	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	532	490	207	..	415	..	
20	ESWARAN	39	M	P4866966	LE	30	NO	NIL	6|12	6|6P	0.20	0.00	6|24P	6|24P	0.60	0.60	11	13	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	421	490	..	..	..	436	
21	ALAGU	40	M	F1975275	RE	14	NO	SMOKING	6|12P	6|6P	0.20	0.00	6|6	6|6	0.00	0.00	17	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	620	542	..	..	362	..	
22	MURUGESAN	49	M	P4865134	LE	30	NO	HTN/DM	6|9	6|6	0.20	0.00	6|6	6|6	0.00	0.00	13	14	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	2	2	2	2	2	2	2	2	2	2	1	2	1	2	2	325	442	96	..	..	225	
23	HAJA ALAVUDEEN	60	M	P4865179	LE	10	NO	EPILEPSY	6|18	6|6	0.50	0.00	6|60	6|36	1.00	0.80	14	12	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	399	453	90	130	..	505	
24	SOMASUNDARAM	42	M	P4859059	LE	20	NO	NIL	6|18	6|6	0.50	0.00	6|18	6|9	0.50	0.20	15	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	1	2	2	2	2	2	2	2	1	1	1	1	2	2	482	537	..	80	..	356	
25	RAMAKRISHNAN	43	M	F1917311	LE	10	NO	NIL	6|6	6|6	0.00	0.00	6|12	6|12	0.30	0.30	16	16	NORMAL	NORMAL	NORMAL	FR-SRF-CSCR	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	426	454	..	..	..	234	
26	RAJAKUMARI	37	F	P4740244	RE	10	NO	NIL	6|24	6|24	0.60	0.60	6|6	6|6	0.00	0.00	12	14	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	1	2	2	2	2	2	1	1	1	2	2	2	340	340	..	..	551	..	
27	GEETHA	36	F	P4711819	LE	15	NO	NIL	6|6	6|6	0.00	0.00	6|12	6|12	0.30	0.30	16	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	504	511	59	27	..	142	
28	POTHURAJA	39	M	P4732907	LE	7	NO	SMOKING	6|6	6|6	0.00	0.00	6|9	6|9P	0.20	0.20	12	11	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	426	403	..	40	..	312	
29	BOOMINATHAN	45	M	P4735579	LE	10	NO	DM	6|9P	6|6	0.20	0.00	6|24	6|9	0.60	0.20	17	14	NORMAL	NORMAL	PED	PED,FR-ABSENT-CSCR	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	364	469	145	143	..	362	
30	SAKAYAM	43	M	P4767442	RE	15	NO	NIL	6|12	6|9	0.30	0.20	6|12P	6|12P	0.30	0.30	11	9	NORMAL	NORMAL	FR-SRF-CSCR	ERM,RPE CHANGES	1	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	462	519	..	..	121	70	
31	ARUNTHATHI	33	F	P4733345	LE	4	NO	NIL	6|9P	6|6	0.20	0.00	6|36	6|36	0.80	0.80	13	12	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	434	378	..	..	..	260	
32	JANARTHANAN	40	M	P4771480	LE	30	NO	NIL	6|6	6|6	0.00	0.00	6|9	6|9	0.20	0.20	15	13	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	1	1	1	1	2	2	461	486	..	..	..	429	
33	SHANAVAS	36	M	P4742653	RE	7	NO	HTN	5|60	5|60	1.00	1.00	6|6	6|6	0.00	0.00	13	11	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	570	467	82	..	436	..	
34	FATHIMA BEEVI	41	F	P4759495	RE	21	NO	NIL	6|36	6|6P	0.80	0.00	6|36	6|6	0.80	0.00	14	16	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	504	408	..	..	334	..	
35	SARAVANAN	44	M	P4734070	RE	30	NO	SMOKING	6|9P	6|9P	0.20	0.20	6|6	6|6	0.00	0.00	14	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	340	327	86	..	91	..	
36	PALANIVEL	46	M	P4730195	LE	30	NO	NIL	6|6P	6|6	0.00	0.00	6|36	6|18P	0.80	0.50	14	12	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	419	479	92	336	..	568	
37	SENTHIL KUMAR	33	M	P4753805	RE	15	NO	NIL	6|12P	6|12P	0.30	0.30	6|6	6|6	0.00	0.00	15	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	553	484	42	..	256	..	
38	THANGAMRAJENDRAN	36	M	P4715777	RE	10	NO	NIL	6|9	6|9	0.20	0.20	6|6	6|6	0.00	0.00	12	15	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	496	461	21	..	142	..	
39	PACHAI PERUMAL	33	M	P4768505	LE	3	NO	NIL	6|6	6|6	0.00	0.00	6|6P	6|6	0.00	0.00	20	18	NORMAL	NORMAL	NORMAL	FR-DULL-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	277	460	..	54	..	192	
40	ILAYARAJA	31	M	P4751366	RE	7	NO	NIL	6|12P	6|9P	0.30	0.20	6|6	6|6	0.00	0.00	15	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	1	2	1	2	2	2	480	383	77	..	546	..	
41	AMARNATH	34	M	P4750207	RE	10	NO	NIL	6|6P	6|6P	0.00	0.00	6|6	6|6	0.00	0.00	14	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	405	355	140	..	424	..	
42	SEKAR	52	M	P4746182	LE	7	NO	HTN	6|6	6|6	0.00	0.00	6|12	6|12P	0.30	0.30	15	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	411	419	..	63	..	243	
43	NAGANATHAN	23	M	P4760440	LE	30	NO	NIL	6|6	6|6	0.00	0.00	6|9	6|9	0.20	0.20	14	16	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	397	402	..	..	..	..	160
44	CHANDRAMOHAN	42	M	P4763180	RE	30	NO	NIL	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	16	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	2	2	1	1	2	2	511	447	..	..	326	..	
45	SEVUGA PERUMAL	43	M	P4743185	RE	10	NO	NIL	6|9P	6|6	0.20	0.00	6|6	6|6	0.00	0.00	16	18	NORMAL	NORMAL	FR-SRF-CSCR	RPE DEGENERATION	1	2	1	1	2	2	2	2	2	2	2	2	1	2	1	2	2	2	560	442	80	142	182	..	
46	RAJ KUMAR	40	M	P4761310	LE	7	NO	NIL	6|6	6|6	0.00	0.00	6|12	6|12	0.30	0.30	14	14	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	406	461	57	130	..	342	
47	SINOJ	33	M	P4750816	RE	10	YES	SMOKING	6|60	6|60	1.00	1.00	6|9	6|6	0.20	0.00	12	14	NORMAL	NORMAL	FR-absent-CSCR	POST CSCR-RPE CHANGES	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	532	446	..	..	574	..	
48	SIVA KUMAR	37	M	F1917505	RE	30	NO	NIL	6|24	6|24	0.60	.0.60	6|6	6|6	0.00	0.00	16	15	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	1	1	2	2	2	2	2	2	2	2	1	2	1	2	2	420	411	..	146	539	..	
49	SURENDRAN	37	M	F1925822	LE	10	NO	NIL	6|6	6|6	0.00	0.00	5|60	5|60	1.00	1.00	15	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	472	567	54	59	..	589	
50	PREETHI SAKARIYA	45	F	P4750572	LE	21	NO	NIL	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	13	11	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	362	376	..	70	..	461	
51	KANNAN	42	M	P4872981	RE	10	NO	TYPE A PERSONALIT	6|6P	6|6P	0.00	0.00	6|6	6|6	0.00	0.00	15	19	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	1	1	1	1	2	2	339	317	67	..	210	..	
52	MUTHU PANDI	44	M	F1997536	LE	7	NO	SMOKING	6|6	6|6	0.00	0.00	6|36P	6|12	0.80	0.30	12	14	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	401	449	96	158	..	549	
53	MUTHALAMMAL	40	F	F1997704	RE	3	NO	NIL	6|12P	6|6P	0.30	0.00	6|9	6|6	0.20	0.00	12	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	354	316	217	..	121	..	
54	VIKRAMAN NAIR	53	M	P4926444	LE	15	NO	ASTHMA/STEROID U	6|6P	6|6	0.00	0.00	6|12P	6|12P	0.30	0.30	15	20	NORMAL	NORMAL	PED	FR-ABSENT-CSCR,PED	2	1	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	357	421	40	115	..	321	
55	KALANDER	54	M	P4905350	LE	10	NO	NIL	6|18P	6|6	0.50	0.00	6|24	6|9	0.60	0.20	14	18	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	350	407	..	63	..	324	
56	SENTHIL KUMAR	40	M	P4877003	RE	7	NO	NIL	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	10	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	390	340	..	..	215	..	
57	KARUPPASAMY	38	M	P4740843	LE	2	NO	NIL	6|6	6|6	0.00	0.00	6|24P	6|24P	0.60	0.60	14	16	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	1	2	2	2	1	2	2	2	2	1	2	1	2	2	2	312	480	112	182	..	472	
58	BABU	32	M	F1980905	LE	20	NO	NIL	6|12P	6|6	0.30	0.00	6|12P	6|9	0.30	0.20	20	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	1	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	449	571	97	74	..	559	
59	SELVI	42	F	P4879184	LE	5	NO	TYPE A PERSONALIT	6|6	6|6	0.00	0.00	6|12P	6|12P	0.30	0.30	16	15	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	362	431	..	111	..	158	
60	SIVARAMAN	43	M	P4564333	LE	30	NO	STEROID USAGE/SK	6|6	6|6	0.00	0.00	6|12P	6|12P	0.30	0.30	14	14	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	1	1	1	1	2	2	324	366	..	..	..	..	401
61	PARAMASIVAM	49	M	P4903488	LE	30	NO	SMOKING,DM	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	15	13	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	1	1	2	2	2	2	2	2	1	2	1	2	2	2	553	531	..	90	..	197	

62	KULANTHAIVEL	35	M	P4906029	RE	40	NO	DM,ASTHMA	6|6P	6|6P	0.00	0.00	6|6	6|6	0.00	0.00	18	20	NORMAL	NORMAL	FR-ABSENT-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	1	2	1	2	2	2	430	364	63	_	177	_
63	NAGARAJAN	43	M	P4908307	RE	3	NO	DM	5|60	5|60	1.00	1.00	6|12	6|6	0.30	0.00	17	16	NORMAL	NORMAL	FR-ABSENT-CSCR	PED	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	513	335	82	76	897	_
64	DEEN ASAN	51	M	P4909287	RE	20	NO	HTN	5|60	6|9P	1.00	0.20	5|60	6|6	1.0	0.00	15	15	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	431	437	_	_	258	_
65	ASARUDEEN	31	M	F1994742	RE	30	NO	TYPE A PERSONALIT	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	14	19	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	578	472	_	_	220	_
66	GURUSAMY	56	M	P4120146	RE	10	NO	DM	6|24	6|18	0.60	0.50	6|6	6|6	0.00	0.00	17	19	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	468	339	194	_	568	_
67	SANJAY GANDHI	37	M	P4901102	RE	7	NO	TYPE A PERSONALIT	6|24	6|9	0.60	0.20	6|9	6|6	0.20	0.00	14	16	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	369	332	_	_	243	_
68	SARAVANAN	40	M	P4873988	LE	15	NO	SMOKING	6|6	6|6	0.00	0.00	6|60	6|60	1.0	1.0	17	13	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	440	463	_	157	_	826
69	PERUMAL	59	M	P4904946	RE	20	NO	NIL	5|60	6|60	1.00	1.00	5|60	6|36	1.00	0.80	13	13	NORMAL	NORMAL	FR-absent-CSCR	RPE DEGENERATION	1	2	2	2	1	2	2	2	2	1	2	2	2	2	2	2	2	2	432	344	_	_	477	_
70	RAMU	42	M	P4892839	RE	42	NO	NIL	6|12	6|12	0.30	0.30	6|6	6|6	0.00	0.00	15	12	NORMAL	NORMAL	FR-SRF-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	361	377	_	_	129	_
71	CHANDRASEKARAN	43	M	F1982118	RE	10	NO	NIL	6|18	6|18	0.50	0.50	6|6	6|6	0.00	0.00	14	12	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	1	2	2	2	2	2	2	1	2	1	2	2	2	370	285	89	_	535	_
72	UTHAMAPUTHIRAN	33	M	P4899930	RE	10	NO	NIL	6|6P	6|6P	0.00	0.00	6|6	6|6	0.00	0.00	13	15	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	352	373	_	_	248	_
73	RAGUNATHAN	40	M	F1996553	RE	10	NO	NIL	6|18	6|18	0.50	0.50	6|6	6|6	0.00	0.00	14	19	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	482	361	110	_	530	_
74	THAYALARAJ	35	M	P3525270	LE	3	NO	TYPE A PERSONALIT	6|12	6|6	0.30	0.00	6|12	6|12	0.30	0.30	14	13	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	303	397	_	_	_	338
75	MANIKANDAN	38	M	P4918748	LE	7	NO	HTN,HYPERLIPIDEM	6|9P	6|6	0.20	0.00	6|9P	6|6P	0.20	0.00	17	15	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	267	330	_	_	_	377
76	GANESHAN	40	M	P4911552	RE	7	NO	NIL	6|6P	6|6	0.00	0.00	6|6	6|6	0.00	0.00	16	20	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	416	400	90	_	310	_
77	PALANI	28	M	P4907473	RE	7	NO	NIL	6|24	6|24	0.60	0.60	6|6	6|6	0.00	0.00	15	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	361	278	_	_	338	_
78	SANKAR	42	M	P4912213	RE	7	NO	TYPE A PERSONALIT	6|24	6|9	0.60	0.2	6|24	6|6	0.60	0	17	15	NORMAL	NORMAL	FR-ABSENT-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	446	486	_	_	248	_
79	SHEIK AMIR	46	M	P4921689	LE	3	NO	HTN,SMOKING	6|6	6|6	0.00	0.00	6|12	6|12	0.30	0.30	19	19	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	340	341	_	105	_	473
80	SELVAM	39	M	P4922588	RE	30	NO	NIL	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	15	16	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	330	288	_	_	264	_
81	KARTHI	32	M	P4926520	LE	2	NO	SKIN DISEASE/STER	6|6	6|6	0.00	0.00	6|6P	6|6	0.00	0.00	19	19	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	314	357	_	_	_	234
82	SARASU KUMAR	43	M	P4881820	RE	14	NO	TYPE A PERSONALIT	6|12P	6|12P	0.30	0.30	6|6	6|6	0.00	0.00	13	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	1	2	2	2	2	2	2	2	2	1	1	1	1	2	2	474	451	91	50	391	_
83	GOVINDHARAJ	49	M	F1982923	RE	3	NO	ASTHMA/STEROID U	5|60	6|18P	1.00	0.50	6|18P	6|6	0.50	0.00	19	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	1	2	2	2	2	2	1	2	1	2	1	2	2	2	425	297	132	_	517	_
84	KARUPPAIAH	42	M	P4882644	RE	42	NO	STEROID USE/SMOI	6|60	6|60	1.00	1.00	6|6	6|6	0.00	0.00	14	13	NORMAL	NORMAL	FR-absent-CSCR	PED	1	2	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	297	323	120	221	783	_
85	VANJI MUTHU	48	M	P4883234	RE	40	NO	DM/RHEUMATOID .	6|36	6|18	0.80	0.50	6|12	6|6	0.30	0.00	15	14	NORMAL	NORMAL	FR-DULL-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	345	370	_	_	540	_
86	SELVI	48	F	P4884643	LE	7	NO	NIL	6|6	6|6	0.00	0.00	6|12	6|6P	0.30	0.00	14	13	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	1	2	1	2	2	353	349	_	_	_	400
87	PAUL RAJ	37	M	P4885337	RE	15	NO	TYPE A PERSONALIT	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	15	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	1	2	2	2	2	2	1	2	1	2	2	2	459	422	_	_	204	_
88	SABU	46	M	P4925041	RE	7	NO	SKIN DISEASE/STER	6|18	6|9P	0.50	0.20	6|6P	6|6	0.00	0.00	16	19	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	390	324	86	77	439	_
89	SIVANAMMAL	43	M	P4855475	LE	5	NO	TYPE A PERSONALIT	6|6P	6|6	0.00	0.00	6|36	6|36	0.80	0.80	14	18	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	367	421	_	_	_	248
90	DINESH	34	M	P4925431	LE	30	NO	ASTHMA/ANKYLOSI	6|6	6|6	0.00	0.00	6|9	6|6P	0.20	0.00	17	15	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	359	363	_	_	_	129
91	PRABHU	35	M	F2000260	LE	10	NO	TYPE A PERSONALIT	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	14	16	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	316	327	_	67	_	169
92	MADHAVAN	53	M	P4164100	LE	7	NO	SKIN DISEASE/STER	6|9	6|9	0.20	0.2	6|6	6|9	0	0.2	14	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	348	350	_	72	_	492
93	MALAR	32	F	F1997158	RE	7	NO	RHEUMATOID ARTH	6|9P	6|9P	0.20	0.20	6|6	6|6	0.00	0.00	12	14	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	1	1	2	2	2	2	357	365	_	_	272	_
94	UKKIRAPANDIAN	33	M	P4885520	LE	15	NO	NIL	6|6	6|6	0.00	0.00	6|6P	6|6P	0.00	0.00	18	18	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	467	582	_	_	_	213
95	SUBRAMANIYAN	56	M	F1984006	RE	30	NO	TYPE A PERSONALIT	6|6P	6|6P	0.00	0.00	6|6	6|6	0.00	0.00	16	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	1	2	1	2	2	2	350	326	_	_	208	_
96	DHANASEKARAN	38	M	P3915842	RE	10	NO	SKIN DISEASE/STER	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	14	16	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	1	2	1	2	2	2	430	439	85	_	173	_
97	AMUTHA	40	F	P4839188	LE	3	NO	NIL	6|6	6|6	0.00	0.00	6|24	6|24	0.60	0.60	14	13	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	383	389	_	124	_	442
98	VIJAYA RAGAVAN	23	M	P4888940	RE	30	NO	NIL	6|24P	6|12	0.60	0.30	6|9	6|6	0.20	0.00	19	20	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	402	353	_	_	469	_
99	MURUGAN	38	M	P4889044	LE	30	NO	SMOKING	6|6	6|6	0.00	0.00	6|12	6|12	0.30	0.30	10	10	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	302	331	_	_	_	383
100	KALAMANI	44	F	P4891971	RE	30	NO	HYPERTHYROID	6|60	6|60	1.00	1.00	6|6	6|6	0.00	0.00	14	12	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	328	306	123	_	846	_
101	SANKAR THAYALAN	41	M	P4902301	RE	10	NO	SMOKING	6|6P	6|6	0.00	0.00	6|6	6|6	0.00	0.00	16	18	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	1	2	1	2	2	2	302	281	_	_	208	_
102	ARUL RAMALINGAM	32	M	P4912671	LE	3	NO	TYPE A PERSONALIT	6|6	6|6	0.00	0.00	6|6P	6|6P	0.00	0.00	14	16	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	364	446	_	_	_	196
103	GANTHI	56	M	F1996735	RE	30	NO	TYPE A PERSONALIT	6|24P	6|18	0.60	0.50	6|6	6|6	0.00	0.00	16	19	NORMAL	NORMAL	FR-ABSENT-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	1	2	1	2	2	2	344	289	_	_	352	_
104	ULAGANATHAN	39	M	P4915804	LE	10	NO	SMOKING	6|6P	6|6	0.00	0.00	6|18P	6|18P	0.50	0.50	16	16	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	1	2	2	2	2	2	1	2	1	2	2	382	384	_	62	_	411
105	SOUNDARA PANDIAN	36	M	P4922689	RE	2	NO	TYPE A PERSONALIT	6|36	6|36	0.80	0.80	6|6	6|6	0.00	0.00	15	17	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	340	302	48	_	786	_
106	SUSILA MARY	52	F	P4945491	RE	10	NO	TYPE A PERSONALIT	6|24P	6|18P	0.60	0.60	6|6	6|6	0.00	0.00	12	12	PSEUDOPHAKI/	PSEUDOPHAKI/	FR-large SRF PED	MULTIPLE PED	1	2	1	1	2	2	1	2	2	2	2	2	1	2	1	2	2	2	238	269	787	100	1041	_
107	SOWMIYA NARAYAN	39	M	P4922694	RE	10	NO	NIL	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	15	15	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	1	2	1	2	2	2	490	474	_	_	258	_
108	VADIVALAKAN	42	M	F1998966	RE	4	NO	NIL	6|60	6|60	1.00	1.00	6|6	6|6	0.00	0.00	15	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	526	467	_	_	611	_
109	POOPANDI	43	M	P4921253	RE	7	NO	TYPE A PERSONALIT	6|9	6|9	0.20	0.20	6|6	6|6	0.00	0.00	17	16	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	374	354	44	_	143	_
110	SURYA KUMAR	27	M	P4881387	RE	15	NO	SMOKING	6|18	6|18	0.50	0.50	6|6	6|6	0.00	0.00	13	15	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	510	489	91	_	376	_
111	SELVI	35	F	P4929340	RE	4	NO	NIL	6|36	6|24	0.80	0.60	6|9	6|6	0.20	0.00	11	12	NORMAL	NORMAL	FR-SRF-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	460	340	119	_	366	_
112	VISWANATHAN	47	M	P4929220	RE	30	NO	SMOKING/CARDIAC	6|18	6|18	0.50	0.50	6|9	6|9	0.20	0.20	19	18	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	377	367	_	_	282	_
113	LATHA	34	F	P4932978	RE	10	NO	HTN	6|12	6|6	0.30	0	6|12	6|6	0.30	0	13	11	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	425	389	_	_	444	_
114	SELVAM	38	M	P4937185	RE	8	NO	TYPE A PERSONALIT	6|36	6|18P	0.80	0.50	6|6	6|6	0.00	0.00	12	14	NORMAL	NORMAL	FR-SRF-CSCR,PED	NORMAL	1	2	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	460	401	252	_	498	_
115	RADHA KRISHNAN	36	M	P4937018	RE	4	NO	NIL	6|24	6|24	0.60	0.60	6|6	6|6	0.00	0.00	13	11	NORMAL	NORMAL	FR-SRF-CSCR	NORMAL	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	461	277	_	_	292	_
116	JOTHI	30	F	P4947507	LE	5	NO	PREGNANT	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	20	18	NORMAL	NORMAL	NORMAL	FR-SRF-CSCR	2	1	2	1	2	2	2	2	2	2	2	2	2	1	2	1	2	2	401	462	_	171	_	275
117	CHINNASAMY	39	M	P4947647	LE	5	NO	NIL	6|6	6|6	0.00	0.00	6|24	6|18P	0.50	0.50	17	15	NORMAL	NORMAL	NORMAL	FR-SRF-CSCR	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	301	343	_	_	_	302
118	RITESH SRIDHARAN	30	M	P4945458	LE	30	NO	NIL	6|6	6|6	0.00	0.00	6|12	6|12	0.30	0.30	13	14	NORMAL	NORMAL	PED	SRF-CSCR	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	434	520	81	_	_	124
119	SABURA BEEVI	46	F	P4926815	RE	30	NO	HTN,THYROID	6|6P	6|6P	0.00	0.00	6|6	6|6	0.00	0.00	14	14	NORMAL	NORMAL	FR-CSCR,PED	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	389	376	190	_	183	_
120	ANIL KUMAR	50	M	P4826459	LE	30	NO	HTN,HYPERLIPIDEM	6|6	6|6	0.00	0.00	6|6	6|6	0.00	0.00	15	16	NORMAL	NORMAL	FR-absent-PED	FR-PED,SRF	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	401	404	15	124	_	451
121	KARUPPAIAH	42	M	P4945018	RE	5	NO	SMOKING	6|12P	6|12	0.30	0.30	6|6	6|6	0.00	0.00	13	10	NORMAL	NORMAL	SRF-CSCR	NORMAL	1	2	1	2	2	2	2	2	2	2	2	2	1	2	1	2	2	2	480	461	140	_	462	_
122	SENTHIL KUMAR	39	M	P4943955	LE	4	NO	TYPE A PERSONALIT	6|6	6|6	0.00	0.00	6|18P	6|18P	0.50	0.50	14	15	NORMAL	NORMAL	PED	FR-ABSENT-CSCR	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	361	380	334	_	_	599
123	MANJULA	42	F	P4941211	LE	_	NO	NIL	6|6	6|6	0.00	0.00	6|9	6|9P	0.20	0.20	12	11	NORMAL	NORMAL	NORMAL	FR-SRF-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	331	367	_	_	_	312
124	KARTHK	36	M	P4942841	LE	10	NO	NIL	6|6	6|6	0.00	0.00	6|36	6|36	0.80	0.80	15	18	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	450	531	73	_	_	634
125	TAMIL SELVI	32	F	P2008362	LE	30	NO	NIL	6|6	6|6	0.00	0.00	6|12	6|12	0.30	0.30	16	17	NORMAL	NORMAL	NORMAL	FR-ABSENT-CSCR	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	394	408	_	_	_	285
126	MUTHURAJ	48	M	P4940809	LE	30	NO	HTN/SMOKING	6|6P	6|6	0.00	0.00	5|60	5|60	1.00	1.00	14	16	NORMAL	NORMAL	NORMAL	FR-CSCR,PED	2	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	297	383	_	70	_	984
127	NISHA	35	F	P4935019	RE	5	NO	TYPE A PERSONALIT	6|18P	6|18P	0.50	0.50	6|6	6|6	0.00	0.00	13	13	NORMAL	NORMAL	FR-absent-CSCR	NORMAL	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	404	340	_	_	417	_