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Case Report	: Central Serous Chorioretinopathy in Fourty Nine Years Old Woman
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Central Serous Chorioretinopathy in Fourty Nine Years Old Woman.

Abstract:

Purpose: To report a case about central serous chorioretinopathy (CSC) in fourty nine years old woman.

Case report: A -49-years old woman came to Cicendo Eye Hospital Center with chief complain bilateral blurred vision since 5 years ago, disturbance in central vision, object appear distorted, and decreased contrast sensitivity. Visual acuity of right eye and left eye are finger counting in three meters. Funduscopy examination reveals a serous neurosensory detachment which appears as a well-delineated transparent blister at the posterior pole with absent foveal reflect on both eyes. Optical Coherence Tomography (OCT) examination are seen a retinal nerve fiber layer (RNFL) is 25 microns in the right eye and 41 microns in the left eye, subretinal fluid, abnormality of Retinal Pigment Epithelium (RPE), and suggestive choroidal neovascularization on both eyes. The typical angiographic finding in Fluorescein Angiography (FA) is the presence of several hyperfluorescence leaks in the level of RPE. Patient was diagnosed with central serous chorioretinopathy (CSC) on both eye.

Conclusion : . Some eyes with CSC may however have a poor visual outcome due to retinal pigment epithelium atrophy, persistent pigment epithelial detachment, subretinal fluid, recurrences, and submacular choroidal neovascularization (CNV).

I. Introduction.

In 1866, von Graefe first describe a disease of the macula characterized by Recurrebt serous macular detachment and named it *recurrent central retinitis*. Almost 100 years later, in 1955, Bennet applied the term *central serous retinopathy*. In 1967, Gass provided the classic description of the pathogenesis and clinical features of the condition which he called *idiopathic central serous choriodopathy*. Since the disease appears to involve both the choroid and the retina accepted name is *central serous chorioretinopathy* (CSC).[1,2,3]

One theory suggested that affected RPE cells lose their normal polarity and pump fluid from the choroid toward the retina, causing retinal detachment. This theory unable to explain why the cells pumping in the wrong direction or how Pigmen Epithelial Detachments (PEDs) form, since pigment epithelial cells pumping in the wrong direction should drive the form it. Excessive tissue hydrostatic pressure within

the choroid form vascular hyperpermeability may lead to mechanical disruption of RPE barrier, damage of RPE cells, and abnormal egress of fluid under the retina. Functional loss of contiguous RPE cells may allow the fluid to accumulate in the subretinal space, causing neurosensory detachment.[1]

Clinically, patient with CSC heve frequently had a proceeding stressful event and more likely have type A personalities. CSC has also been associated with cortisol levels, thus implying higher incidence in patients with Cushing's diseaseCSC is common in Caucasians, Asians, and Hispanics, and rare in African Americans.[1-5]

In the past, CSC has been considered predominantly a disease of males between 30 and 50 years of age. The overall incidence in men versus women in numerous reports was approximately 8-9:1, but the incidence in compared with ages 21-30. Very little is known about age specific prevalence and clinical findings of CSC in older adults. Neurosensory macular detachment in an adult over 50 years of age, in fact, suggest the presence of choroidal neovascularization (CNV) secondary to age-related macular degeneration (AMD). Most patients are asymptomatic unless the central macula is affected, and most common unilateral.[1-5]

Many patient first notice a minor of vision followed by various degrees of methamorphopsia, micropsia, dyscromatopsia, hypermetropization, scotoma, as well as loss of contrast sensitivity. In some patients the onset of symptoms is preceded or accompanied by migraine like headaches.[1-6]

Visual disturbances are usually mild and spontaneous recovery occurs over a few months. Some eyes with CSC may however have a poor visual outcome due to retinal pigment epithelium atrophy, persistent pigment epithelial detachment, subretinal fluid, recurrences, and submacular choroidal neovascularization (CNV). Probable reasons for development of CNV following CSC are decompensation of the pigment epithelium/Bruch's membrane complex by disease process itself..[7]

II. Case Report.

A 49-years-old woman came to Cicendo Eye Hospital Centre on July, 18th 2011 with chief complain blurred vision on both eyes since 5 years ago. She had developed disturbance in central vision, object appear distorted, and decreased contrast sensitivity. No history of pain, headache, denies episode of stress, hypertension, diabetic, using drugs for long-term especially steroids, family history, denies of tobacco or alcohol use. The examination visual acuity revealed finger counting in three-meters on both eyes. Best corrected visual acuity in the right eye 0.1 snellen with spheres +2.00 diopters, and in the left eye 0.1 snellen with spheres +1.75. Amsler-grid test reveals scotoma, and methamorphopsia on both eyes, contrast sensitivity is decreased on both eyes. Ophthalmology examination in segment anterior within normal limit. Funduscopy indirect examination reveals reveals a serous neurosensory detachment which appears as a well-delineated transparent blister at the posterior pole with absent foveal reflect on both eyes. Patient was initially diagnose with CSC in both eye and had roborantia once a day as therapy. Patients was planned for OCT examination for next visit.

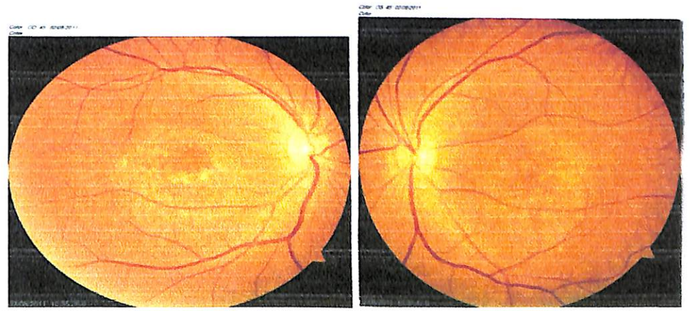

Image 1. Funduscopy on both eye

Second visit on July, 26th 2011 was found similar complain with previous examination. The examination visual acuity revealed finger counting in three-meters

on both eyes. Ophthalmology examination in segment anterior within normal limit..Funduscopy indirect examination reveals a serous neurosensory detachment which appears as a well-delineated transparent blister at the posterior pole with absent foveal reflect on both eyes. OCT examination are seen a retinal nerve fiber layer (RNFL) is 25 microns in the right eye and 41 microns in the left eye, detachment of the neurosensory, subretinal fluid, and abnormality of Retinal Pigment Epithelium (RPE) on both eyes, and suggestive choroidal neovascularization on both eyes. Patient was diagnosed with CSC on both eyes. Patients was planned for Fundus Angiography examination for next visit.

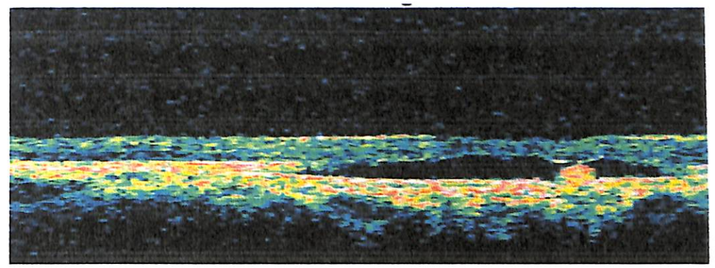

Image 2. OCT in the right eye.

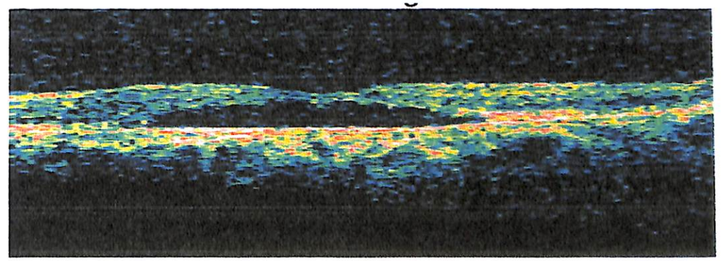

Image 3. OCT in the left eye.

Third examinationon August, 18th 2011 was found similar complain with previous examination. The examination visual acuity revealed finger counting in three-metrees on both eyes. Ophthalmology examination in segment anterior within normal limit.Funduscopy indirect examination revealsa serous neurosensory detachment which appears as a well-delineated transparent blister at the posterior pole with absent foveal reflect on both eyes. OCT examination are seen a retinal nerve fiber layer (RNFL) is 25 in the right eye and 41 in the left eye, subretinal fluid, abnormality of Retinal Pigment Epithelium (RPE), and suggestive choroidalneovascularization on both eyes. Fluorescein Angiography (FA) is the presence of several hyperfluorescent leaks in the level of RPE. Patient was diagnosed with choroidal neovascularization complicating CSC on both eye, and patient was planned for anti vascular endothelial growth factor (VEGF) intravitreal injection.

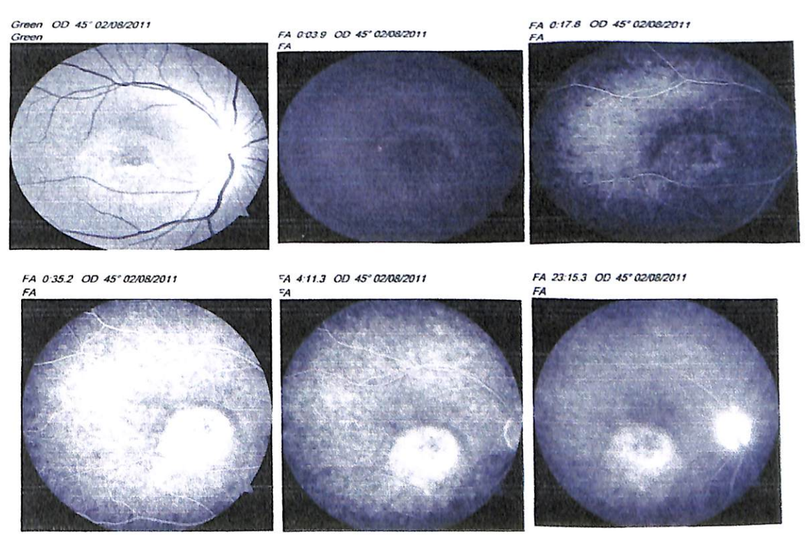

Image 4.FA in the right eye.

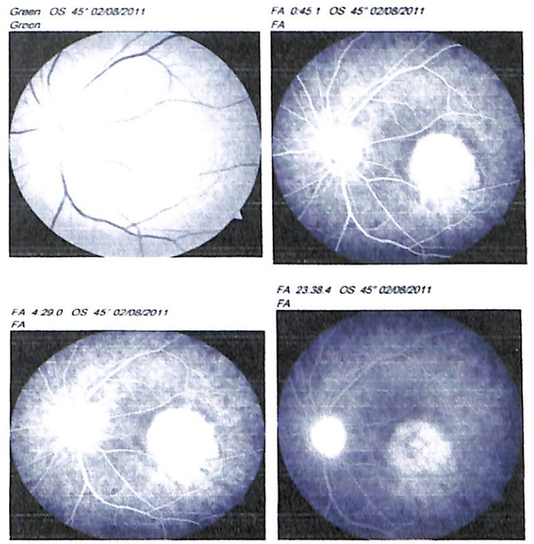

Image 5.FA in the left eye.

III. Discussion.

Many patient with first notice a minor blurring of vision followed by various degrees of methamorphopsia, micropsia, dyscromatopsia, hipermetropization, and central scotoma, as well as loss of contrast sensitivity. In some patients the onset of symptoms is preceded or accompanied by migrane like headaches, certain personality type including type A personality, hypochondria, hysteria, conversional neurosis, using steroid medication, pregnancy, stress, psychiatric medication are at in increased risk of developing CSC. Visual acuity in acute stages from 20/20 to 20/200. Visual can usually be improved with a small hyperopic correction, but complication of chronic CSC reduced visual acuity remains after resolution of subretinal fluid.[1,2,3,6]

In this patient had blurred vision since 5 years ago. She had developed disturbance in central vision, object appear distorted, and decreased contrast

sensitivity.The examination visual acuity revealed finger counting in three-metrees on both eyes. Best corrected visual acuity in the right eye 0.1 snellen with spheres +2.00 diopters, and in the left eye 0.1 snellen with spheres +1.75. Amsler-grid test reveals scotoma, and methamorphopsia on both eyes, contrast sensitivity is decreased on both eyes. The decreased vision can be corrected with hyperopic correction. In this patient other etiologic factor has not been presented.

The mechanism involved in the pathogenesis of CNV and associated CNV membrane (CNVM) formation are being elucidated but are still not fully understood. In CNV, activated endothelial cells migrate through Bruch's membrane; this process occurs by degradation of an intact Bruch's membrane break. Clinocopathologic studies suggest that classic CNVs are predominantly subretinal in location, whereas occult CNVMs are predominantly sub-RPE. Bruch's membrane may disrupted when the balance between proteolitic enzymes such as as matrix metalloproteinase (MMPs) and their inhibitors, the tissue inhibitors of metalloproteinase (TIMPs), favors a protein environment.[8]

Increased expression of VEGF in CNV suggested the controversial hypothesis that tissue hypoxia may be an etiologic factor for this order, since hypoxia stimulates VEGF expression in RPE.[8]

The hyperpermeability of the choriocapillaris may be caused by capillary and venous congestion, possibly because of choroidal ischemia. Choroidal ischemia in CSC may induce an increase in the concentration of VEGF, which has profound effects on vascular permeability. For these reasons, it is likely that choroidal hyperpermeability caused by choroidal ischemia is a nearly event in the development of symptomatic CSC, where under the appropriate circumstances, it may progressively lead to RPE detachment followed by neurosensory detachments. Theoretically, reduced levels of VEGF may improve the choroidal ischemia, thus ameliorating the choroidal hyperpermeability in CSC.[9]

CSC has been considered predominantly a disease of males between 30 and 50 years of age. The overall incidence in men versus women in numerous reports was

approximately 8-9:1, but the incidence in compared with ages 21-30. Most patients are asymptomatic unless the central macula is affected, and most common unilateral.[1,2,6]

In this patients was diagnosed with choroidal neovascularization (CNV) complicating CSC on both eye. Very little is known about age specific prevalence and clinical findings of CSC in older adults. We suggested the presence of (CNV) secondary to CSC that disrupted of Bruch's membrane and increase expression of VEGF. The hyperpermeability of the choriocapillaris may be caused by capillary and venous congestion, possibly because of choroidal ischemia. Choroidal ischemia may induce an increase in the concentration of VEGF, which has profound effects on vascular permeability.

Funduscopy indirect examination with CSC reveals a detachment of the neurosensory retina. The normal foveal light reflect is usually absent and replaced by a halo of light reflect delimiting the elevated area. The detached neurosensory retina is usually transparent and of normal thickness. In this patients, funduscopy indirect examination reveals a detachment of the neurosensory retina which appears as well-delineated transparent blister at the posterior pole and absent of foveal light reflect.

OCT is an excellent, non invasive method to use for diagnose and following the resolution of the retinal fluid in CSC. Subtle fluid accumulation beneath the sensory retna and the RPE not evident on FA and clinical examination can often be picked up by OCT. once the diagnoses is established, OCT can be used to follow and documentation the resolution of the subretinal fluid. Careful scanning the OCT revelas serous PED under the elevation more often than previously recognized. Long-standing and recurrent PEDs may present with pigment migration or atrophy. In most cases the fibrin deposits dissolve; rarely do they stimulate subretinal fibrosis and fibrotic scar formation, leading to permanent visual loss.[1,7]

OCT in this patients reveals RNFL thickness on the both eye is decreased, subretinal fluid, abnormality of Retinal Pigment Epithelium (RPE), and suggest choroidal neovascularization (CNV) on both eye.

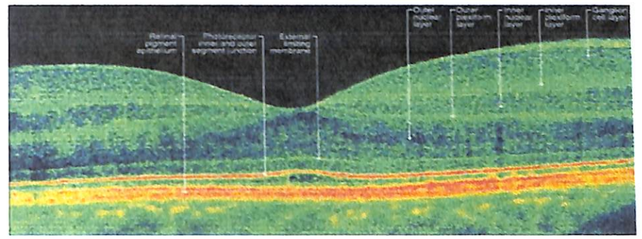

Image 6.OCT normal.

One characteristic of CNV is its tendency to leak and pool. There is an association between clinical leaking and the presence of an enlarged of subretinal space. When the first visible signs of leakage appeared on angiographic examination, newly formed vessels had spread into the subretinal space around the break in Bruch's membrane , fluid was accumulating in the subretinal space, and RPE cells were proliferating in a papillary pattern around the newly formed vessels. At the end of the involution process, when the neovascular membrane no longer demonstrated any leakage, the sub retinal vessels were found to be tighly enveloped by RPE cells and no fluid separated them from the sensory retina.[8]

In this patient, FA on both eye reveals window defect in choroidal phase, and hyperfluoresence in arteri phase, and arteriovenous phase, but decreased in late phase similar with decreased of the dye of fluorescence in arterivenous. This FA may be explained CNV, atropihic RPE tracts appear as mottled hyperfluoresence, because three characteristic fluorescein angiographic patterns, expansile dot pattern, smokestack pattern and diffuse pattern, are not seen in this patient FA.

Patient was diagnosed with CNV complicating CSC because presenting with CNV occasionally may show evidence suggestive of previous CSC, with differential diagnosed with choroidal neovascularization (CNV) secondary to age-related macular degeneration (AMD) but the incidence most common over 50 years of age. Idiopathic polypoidalchoroidalvasculopathy usually corresponds closely to the area of leakage on angiography and involve small-caliber vascular abnormalities and may present

exclusively with a neurosensory detachment of the neurosensory retina. The polypoidal lesions may resemble small PEDs clinically and by fluorescein angiography. These cases masquerade as CSC. Indocyanine Green Angiography (ICG) is important in differentiating these two disorder. Complication of CSC may present diffuse RPE damage, multifocal area of leakage, and subretinal deposit of fibrin and damage, and presented of CNV[1,2,3]

Bevacizumab is a recombinant humanized full-length monoclonal antibody that binds all isoforms of VEGF. The bevacizumab molecule can penetrate the retina and is also transported into the RPE, the choroid and photoreceptor outer segments after intravitreal injection. Intravitreal bevacizumab has been utilized to treat numerous ocular disorders, generally those which associated with neovascularization or vascular leakage as a consequence of an underlying disease. In general, the results have been positive, with numerous case series describing regression of neovascularization or resolution of leakage in response to the treatment.[9,10.]

Treatment for this patient is anti VGEF intravitreal injection to reduces of CNV and inhibitors VEGF. In 2008, Niegel et al reported the first case report of the intravitreal use of bevacizumab for CSC. The results suggest that intravitreal use of bevacizumab is safe and effective for the treatment of CSC.[1,2,9,10.]

Patient was diagnosed with chronic CSC because older patient presenting with CNV occasionally may show evidence suggestive of previous CSC, with differential diagnosed with choroidal neovascularization (CNV) secondary to age-related macular degeneration (AMD) but the incidence most common over 50 years of age. CSC in older patient may present with more diffuse RPE damage, multifocal area of leakage, and subretinal deposit of fibrin and damage. Chronic CSC may be complicated by secondary CNV. Idiopathic polypoidal choroidal vasculopathy usually corresponds closely to the area of leakage on angiography and involve small-caliber vascular abnormalities and may present exclusively with a neurosensory detachment of the neurosensory retina. The polypoidal lesions may resemble small PEDs clinically and by fluorescein angiography. These cases masquerade as CSC. Indocyanine Green

Angiography (ICG) is important in differentiating these two disorder. The yellow-white exudates of Vogt-Koyanagi-Harada syndrome can appear similar to CSC; however, the granulomatous uveitis seen in the former helps differentiate the diseases.[1,2,3]

Prognosis for this patient is *quo ad vitam bonam, quo ad functionam dubia ad malam.*

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