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Case Series

Exudative Retinopathy Revisited

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Abstract

Exudative retinopathy is not a disease by itself but a manifestation of a wide range of local and systemic conditions affecting the blood vessels. These conditions can be developmental, degenerative, infectious and inflammatory. The inflammatory conditions such as CSC, APMPPE and VKH which represent different spectrum of presentation. Exudative retinopathy is a sign of an underlying disease but not a disease in itself just like headache. The present cases support that there is an overlap between them; but the severity of the underlying cause may explain the difference in presentation.

Keywords: *Exudative Retinopathy; VKH; APMPPE; CSC*

Introduction

Exudative retinopathy is usually resulting from accumulation fluid in the sub retinal and/or sub retinal pigment epithelium spaces. It is of vascular origin. The aetiology may vary. It may include degenerative, Inflammatory, infections and developmental and hereditary conditions. The severity of the exudative retinopathy may vary reflecting the degree of the underlying pathology. We are presenting 12 cases as summarised in table 1. They represent different spectrum of presentation of the exudative retinopathy.

No	Age and sex	DX	RX	Response to Rx	SRF.	Recurrence	Follow-up	Comment
1. Case	42 M	CSC	Avastin. X2	Recurrence. Once	+	+	18 mo	Did well
2. Case	35 F	CSC	Short course of steroid	SRF disappear.	+.		6 mo	Did well.
3a. Case	47 M	CSC OD.	Lucentis X3 times	SRF disappear	+	+	2 years	Didi well
3b. Case	47 M	RPE detachment. OS	Lucentis OS. 2 times	No response	+.	Persist	2years	No response
Case # 4,	30 M	CSC OS	Lucentis	SRF disappear	+	-	4 mo	Did well
Case #5,	22 M	CSC? VKH? OU	Steroids		+.	-	-	Lost F/U
case # 6,	26 M	VKH OU.	Steroid.	Responded well	+	-	4 mo	Did well

Case # 7,	19 M	APMPPE. Ou	Steroid	Responded well	+	-	6 mo	Well
Case # 8,	32 M	APMPPE. Ou.	steroid.	Responded well.	No OCT	-	6 mo	Did well
Case # 9,	15 M	VKH OD	steroid.	responded well	+	-	4 mo	Did well
Case # 10,	25 F	VKH	Steroid	Responded well.	+	-	6 mo	But needed extended low dose of steroid
Case # 11,	42 F	VKH. Ou	Steroid.	Responded well.	+	-	6 mo	Did well
Case 12,	30 F	VKH	Steroid	Responded well first then turn to chronic condition	+	+	10 y	Extensive pigmentary changes in both fundi

Table 1: *Summarises the information about the present cases.*

Case Series

Case 1: Forty-two years old male was seen on April because of decreased vision in the left eye. Patient had refractive surgery for myopia of minus 4D a while back. His vision was 0.8 in the right and 0.7 in the left. Anterior segment was ok. Fundus revealed elevation of the centre of the macula in the left eye. Right eye was normal. OCT of the right eye was normal, while the left revealed elevation of the neuro-sensory retina at centre of the macula. He received intravitreal Avastin in the left eye. He did very well and SRF disappeared and his vision was 1.0 in both eyes. Patient lost the follow up and return one year later with recent recurrence in the same eye as confirmed with OCT. again; he received intravitreal Avastin injection which led to disappearance of the SRF and his vision return to 1.0 in both eyes without any complains (Figure 1).

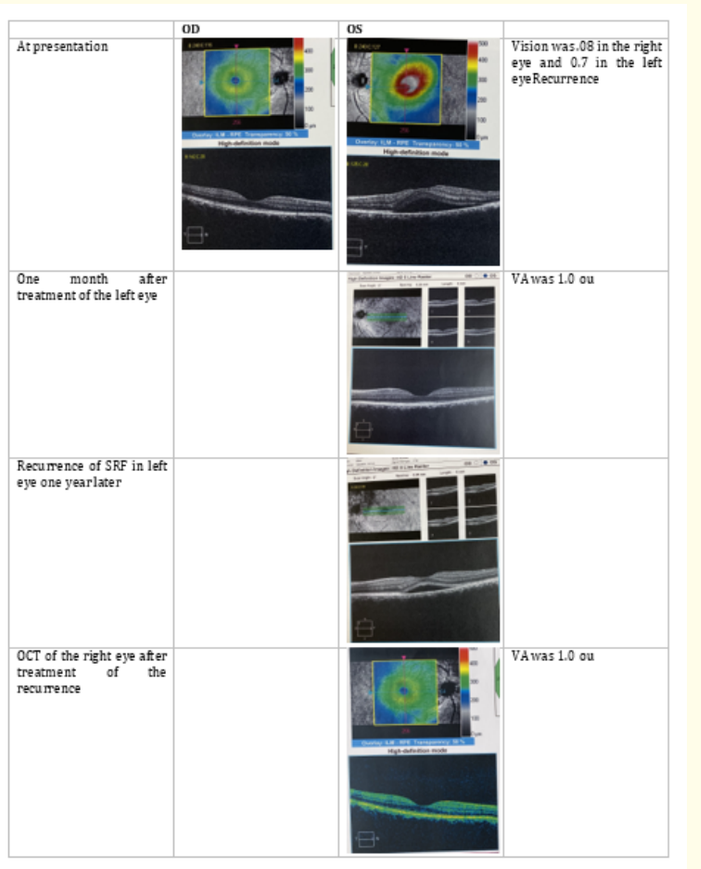

Figure 1: *Showing the OCT of both eyes during the follow up period, 1st row showed SRF left macula. 2nd row showed the resorption of the SRF 1 month after treatment. 3rd row showed the recurrence in OCT of the left eye. 4th row showed the resolution of the SRF after treatment.*

Case 2: Thirty-five years old female presented on July with marked decreased vision in the right eye for one week. No other complaints except on treatment for hypertension. Her best corrected Vision 0.05 in the right eye and 1.0 in the left eye. Tension was 22 mm hg in both eyes. Anterior segment was normal in Both eyes. Right fundus revealed elevated macula and normal in the left. Fluorescein angiogram revealed multiple leaking spots in the right macula. Patient received short course of steroid for one week. Her vision improves to 0.8 in the right eye. Repeated fluorescein angiogram in august, revealed resolution of the sub-retinal fluid (SRF) and disappearance of the hot spots. On Nov, she was on no treatment her vision was 1.0 in Both eyes (Figure 2).

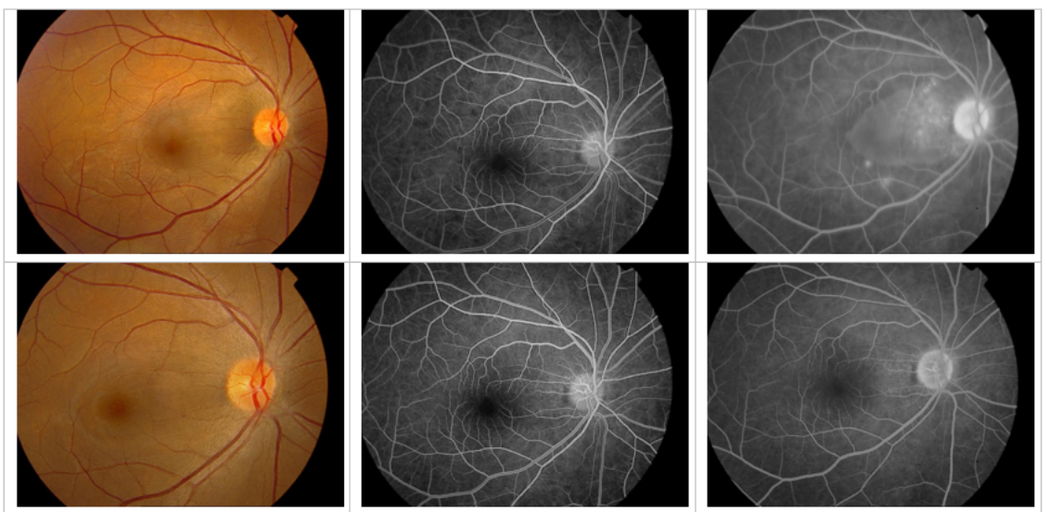

Figure 2: *Showing clinical and fluorescein angiogram of right fundus. Top row showed the photos of right eye at presentation. It shows SRF in the right macula with multiple areas of leakage. The second row showed the resorption of the SRF from right macula after treatment.*

Case 3: Forty seven years old male first seen on April because of blurred vision in the right eye. He had refractive surgery few years back. Two years back had similar episode of decreased vision in the right eye and he was treated elsewhere and he recovered. His vision was 1.00 ou and tension was 14 mmHg both eyes. Anterior segment was ok in both eyes. Fundus examination revealed elevation in the fovea in the right eye and minimal pigmentary changes in the fovea more in the left. OCT confirm presence of fluid in the sub-retinal space and sub RPE in the right eye and minimal localised sub RPE fluid in the left. Patient received 3 injection of Lucentis in the right eye and 3 injections in the left eye. Patient had complete resolution of the SRF and Sub RPE fluids from the right eye (10 months follow up) but the left eye fail to respond after the 3 injections of Lucentis. Patient preferred to observe the condition in the left eye. His last visit in Nov his vision was 1.0 both eyes and tension was 15 mm Hg in both eyes and funds revealed some pigmentary changes in the fovea more in the left eye. OCT was normal in the right eye and but small amount of sub RPE fluid in the left (Figure 3).

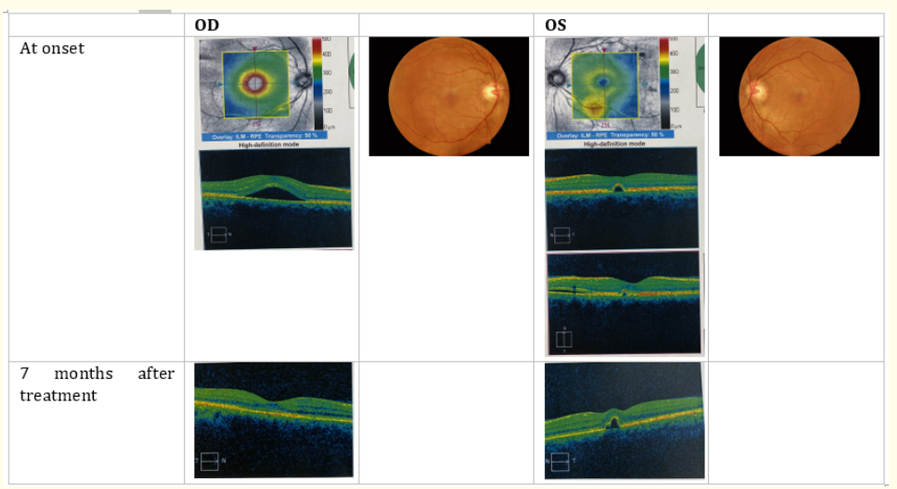

Figure 3: *Showed clinical and OCT Pictures of both fundi. 1st row at presentation showing clinical and oct pictures with SRF and sub RPE in OD and sub-RPE in the left. 2nd row showed the OCT of both eyes showing disappearance of fluid in right eye and persist of the subRPE fluid in the left eye.*

Case 4: 30 years old male seen in October because recent drop in vision LE. No other associated problems, his vision was 1.0 in the right eye and 0.3 in the left eye. Anterior segment examination was normal. Examination of the right funds was normal while there was elevation of the center of the macula in the left eye which was confirmed with OCT. Patient received intravitreal lucentis. Follow up visit confirmed marked improvement. His vision improved to 1.0 in both eyes and the sub retinal fluid disappeared (Figure 4).

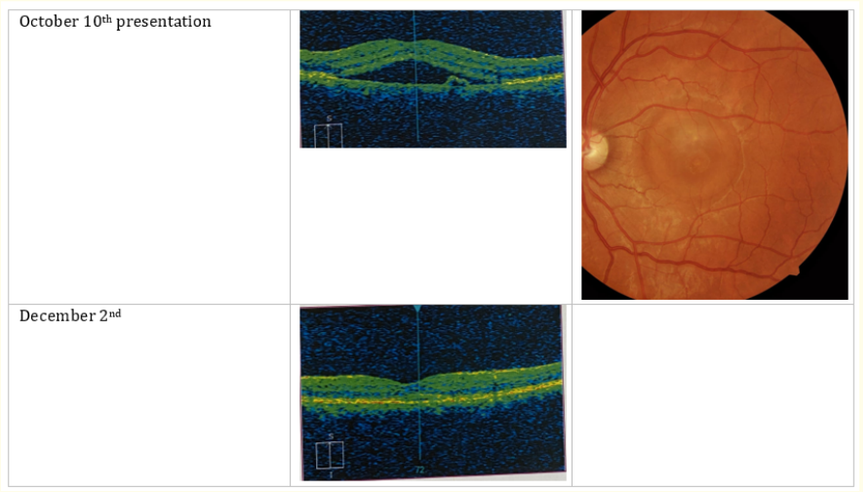

Figure 4: *Showing clinical and oct pictures of the left fundus. 1st row sowed clinical and OCT pictures at presentation showing sub retinal and sub retinal pigment epithelium fluids in the center of macula. 2nd row showing the OCT of the left eye with complete resolution of the sub retinal fluids.*

Case 5: This 22 years old male was first seen on Feb because of recent and marked drop in vision of the right eye more than in the left. No systemic problem. Best corrected vision in the right eye was 0.05 and in the left 0.8. anterior segment was normal in Both eyes. intraocular pressure was normal in Both eyes. Fundus exam revealed marked accumulation of sub-retinal fluid occupying the whole macula in the right eye and to a lesser extent in the left eye. Fluorescein angiogram confirm multiple hotspots of leakage in the right macula and multiple spots of leakage in the left but to a lesser extent. Patient fail to show up for management (Figure 5).

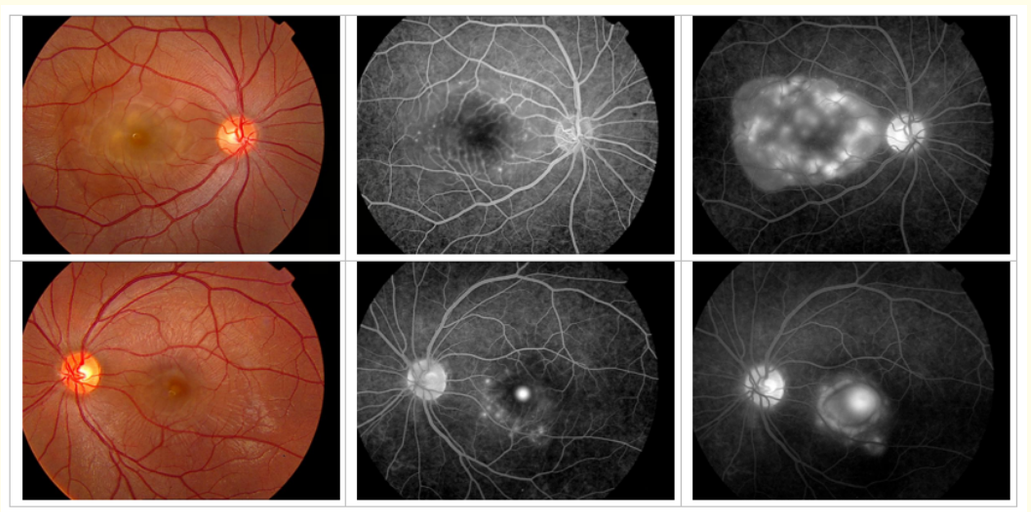

Figure 5: *Showed the clinical and fluorescein angiogram of both fundi. There was sub retinal fluid in the Right macula more than in the left with multiple spots of leakage in both eyes.*

Case 6: Twenty years old male was seen for first time on Feb because of sudden marked loss of vision of both eyes for the last few days. No history of headaches or mucous membrane ulcers. Best corrected vision on the right was 0.05 and on the left 0.2. Tension was 10 mm hg in both eyes. Anterior segment was quite in both eyes. fundus exam revealed some cells in the vitreous and sub retinal fluid involving the center of the right macula and to a lesser extent in the left. Work up was negative. Patient responded well to intensive steroid therapy. His vision was recovered and the retinal fluid disappeared. He was off medication for 5 weeks; his vision on the right eye was 1.0 and left eye was 0.63 without correction fundus and fluorescein angiogram revealed resorption of the sub-retinal fluid and RPE changes in both maculas (Figure 6).

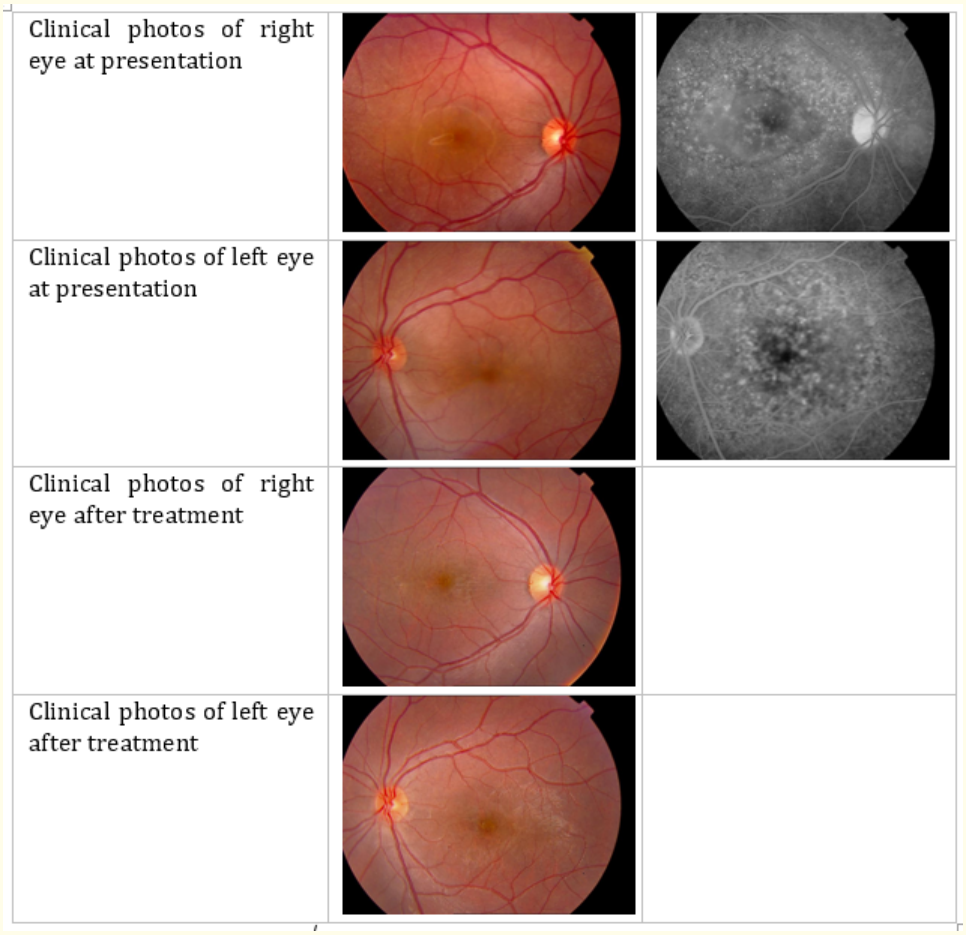

Figure 6: *Showing clinical and fluorescein angiogram of both fundi. 1st and 2nd rows showing the clinical and fluorescein angiogram pictures at presentation with SRF more in the right eye. 3rd and 4th rows showed resorption of the SRF from both maculas after treatment.*

Case 7: Nineteen years old male came because of sudden marked drop in vision in Both eyes on Dec. He denied any systemic problem. His vision 0.05 in both eyes. Findings were limited to fundus in both eyes showing multiple white elevations involving the entire posterior pole of both eyes. OCT showed thickened retina with macular edema and some sub retinal fluids and areas of hyper-reflective lesions in both eyes. Systemic investigations were negative. Patient was started on systemic steroid and zantac. One month later his vision recovered to 0.8 in both eyes. The fundus revealed resolution of the retinal edema and the sub retinal fluid (Figure 7).

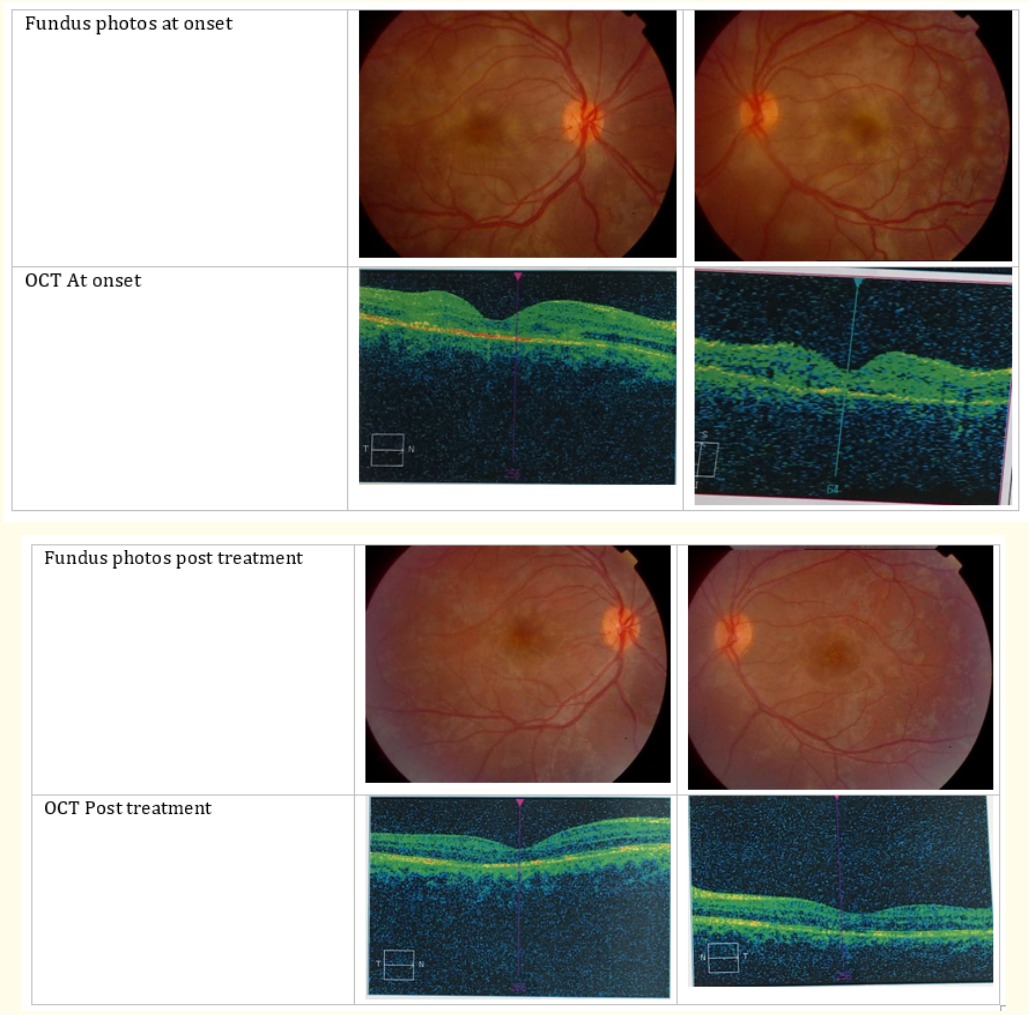

Figure 7: *Showing clinical and OCT pictures of both fundi. 1st and 2nd rows showed the pictures at onset showing thickening of the retina and sub retinal fluid in both eyes. 3rd and 4th rows showing resolution of retinal changes after treatment.*

Case 8: Thirty-two years old female was seen because of sudden severe drop of her vision in Both eyes. She denied any systemic problem. Best corrected vision on the right eye was counting fingers at 1 meter and 0.05 in the left. Intraocular pressure was 15 mmHg in the right and 14 mmHg in the left. Anterior segment was quiet in Both eyes. The findings were limited to the fundus of both eyes revealing exudative retinal detachment and retinal edema in both eyes with multiple areas of leakage in Fluorescein angiogram of both fundi. Systemic work up was negative. Systemic steroid and Zantac were given. Last seen 2 months after discounting the medication; her vision was 1.0 both eyes. Tension was 12 mm Hg in both eyes. Both fundi revealed resolution of the retinal edema and the retinal detachment. On her last visit; she was off treatment for 6 months and continued to do well. This case represents an overlap between APMPPE and VKH (Figure 8).

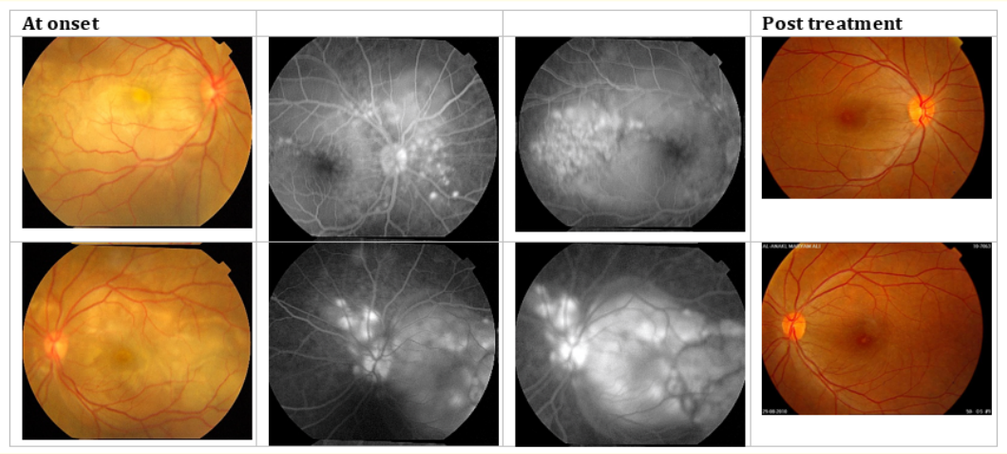

Figure 8: *Showing clinical and Fluorescein angiogram of both fundi photographs at onset and after treatment. First row showed clinical and fluorescein angiogram the right eye pre-treatment and the clinical picture post-treatment there is a resolution and of the retinal edema and inflammation which was present at presentation. 2nd row showed the same in the left eye.*

Case 9: Fifteen years old boy came on June, with sudden drop of vision on the right eye more than left. Medical history was negative. His best corrected vision on the right eye was 0.3 and on the left was 0.9. Anterior segment was ok. Fundus dull reflex on the right and normal on the left. OCT on the right confirm thick retina and presence of subretinal fluid and the left OCT was normal. He received intensive course of steroid and Zantac. Two weeks later his vision was 1.0 in both eyes and the sub retinal fluid resorbed and steroid was being tapered and then it was discontinued (Figure 9).

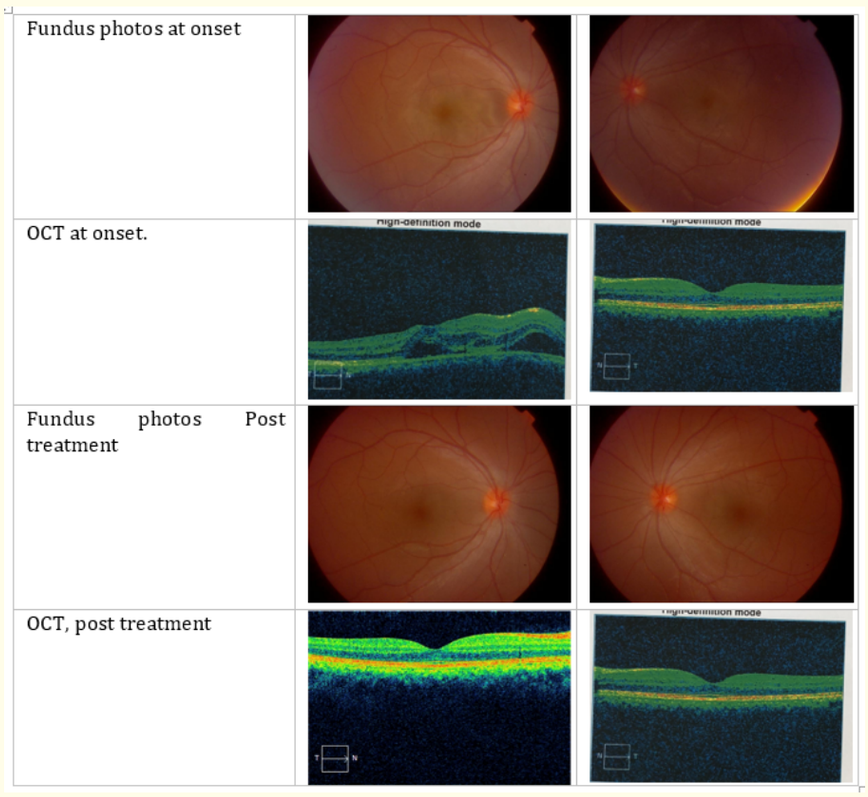

Figure 9: *Showing clinical and OCT picture of both fundi at presentation and after treatment. First and second row showed presence of sub retinal fluid in the right fundus. 3rd and 4th rows showed the disappearance of the sub retinal fluid from the right fundus.*

Case 10: Twenty-five years old female came on sept with sudden decreased vision in both eyes for the last few days. She had no other complains. Vision was 0.25 in both eyes. Tension was normal both eyes. Findings were limited to fundus in both eyes with minimal vitritis in both eyes. There were diffuse thickening of the retina with white elevated patches more in the left eye. OCT confirmed presence of sub-retinal fluids in both eyes. She received an intensive course of steroid treatment and she responded well to the treatment. Her vision improved to 1.00 in both eyes and the sub retinal fluid disappeared and the fundus findings improved as seen in clinical and OCT photos (Figure 10).

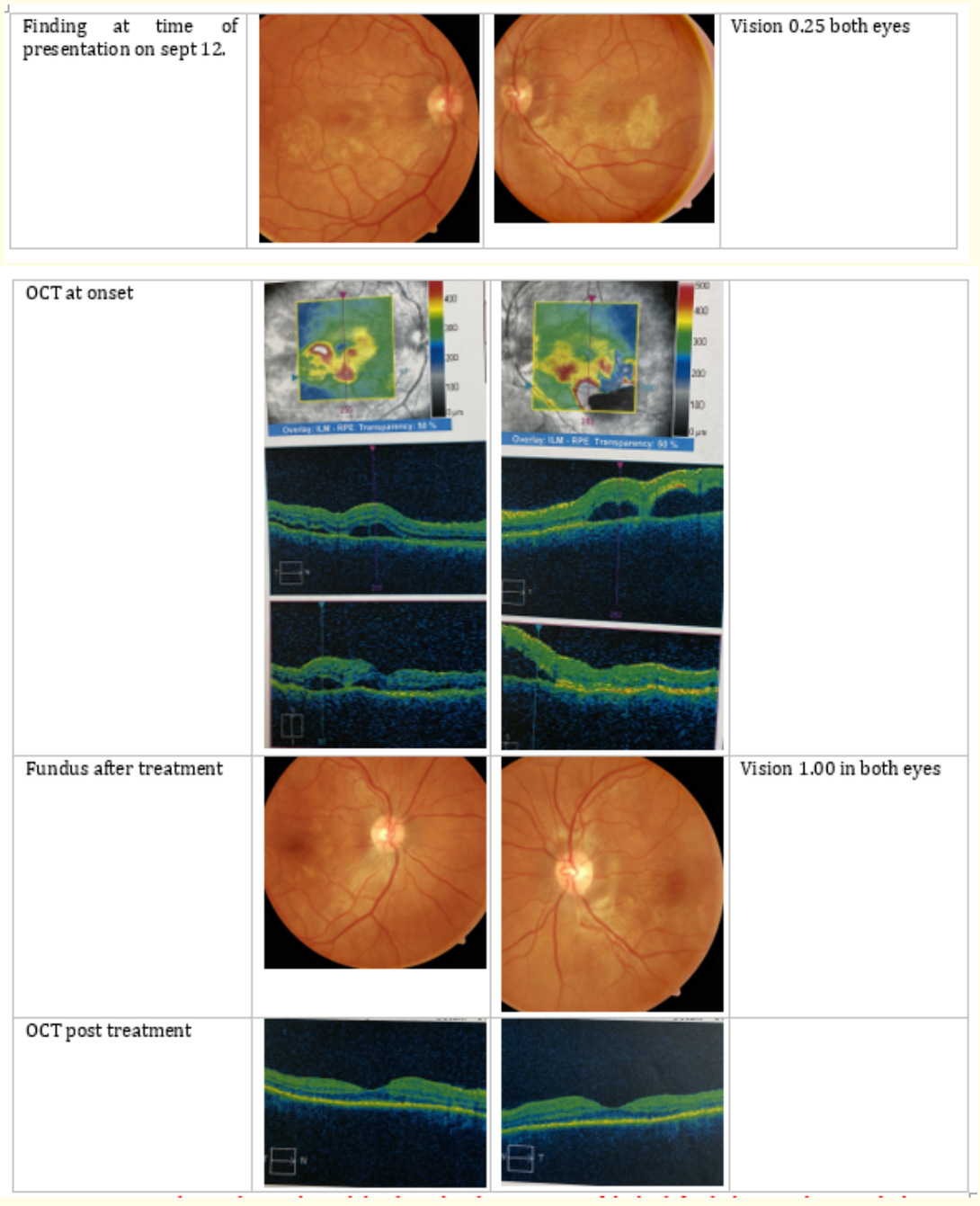

Figure 10: *First and second rows showed the clinical and OCT picture of the both fundi showing the retinal edema and retinal detachment at onset. The 3rd and 4th rows showed the clinical and OCT pictures of both fundi after treatment showing resolution of the retinal detachment.*

Case 11: Forty-two years old female was seen on Nov, because of severe headache more on the left side with decreased vision in both eyes. Blood pressure was normal, recent MRI was reported to be normal. Best corrected Vision was 0.63 in both eyes. tension was 12 mm Hg in both eyes. Motility was normal. Anterior segment was normal Fundus exam revealed bilateral exudative retinal detachment and papilledema. OCT revealed bilateral retinal detachment with bilateral disc edema. She was diagnosed as a case of VKH. She received an intensive course of steroid therapy and she responded well. On March, her best corrected vision was 1.0 in both eyes and OCT returned to normal (Figure 11).

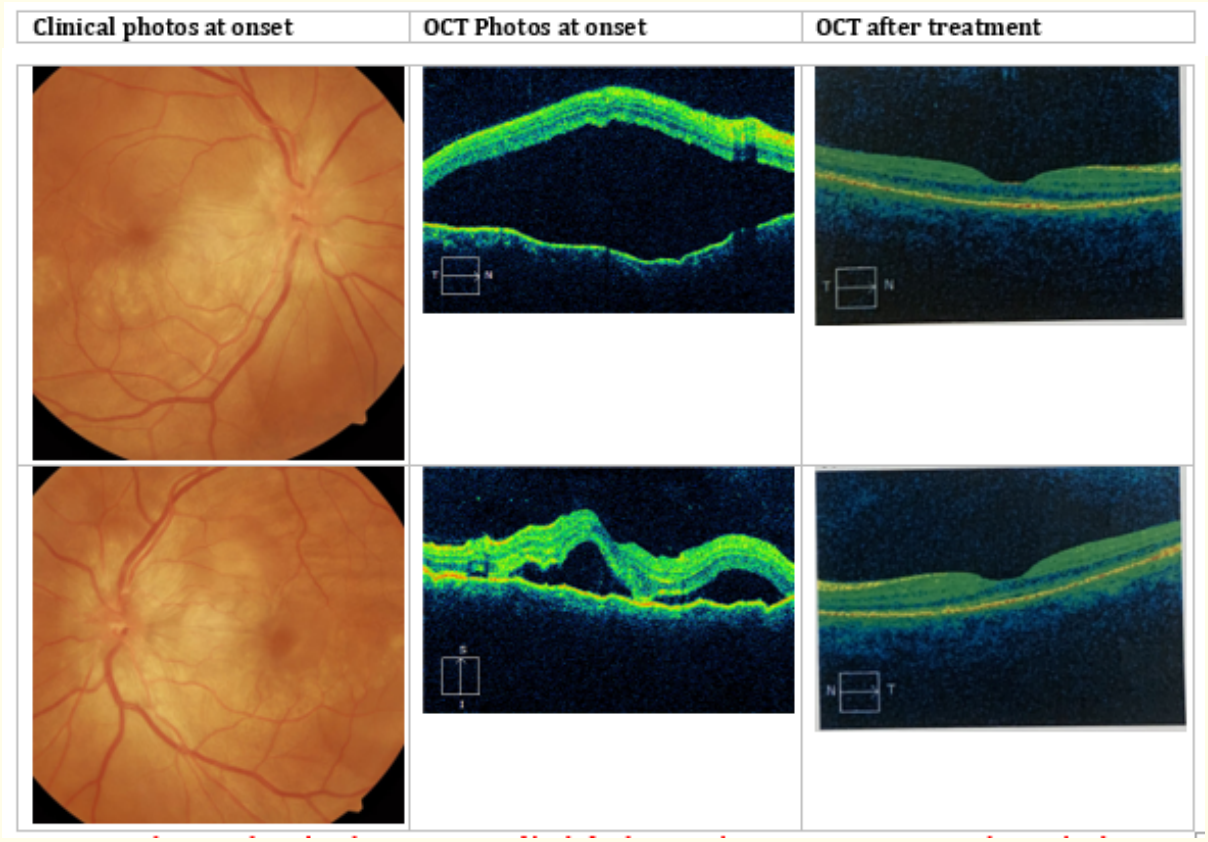

Figure 11: *FShowing Clinical and OCT pictures of both fundi Pre and post treatment. First and second columns showed Retinal detachment and disc edema in Both eyes. Third column showed the resolution of the detachment in OCT after treatment.*

Case 12: Thirty years old female was first seen and treated in 2005 for episcleritis OS. Her vision at that time was 0.8 in the right eye and 0.63 in the left eye. She lost the follow up till March 2008, at that time she was seen because of deterioration of her vision in both eyes for the last few days. She was pregnant and due to deliver. She was found to have exudative detachment in both eyes. Her visual acuity was counting fingers in both eyes. Treatment with high dose of steroid was given after she delivered her baby. She responded to treatment her VA improved to 0.63 both eyes with correction. Tension was 18 mm Hg in both eyes, anterior segment was ok, but fundus revealed resolution of the retinal detachment from both eyes. There was an evidence of pigmentary changes in both fundi and early degenerative changes involving the macula of both eyes being right eye was affected more than left (Figure 12A). On Dec 2015 she was seen again at this

time her vision was 0.5 in the right eye and 0.7 in the left eye. Tension was 20mm Hg Both eyes. Fundus revealed marked degenerative and pigmentary changes in both fundi (Figure 12A). Recent OCT revealed marked RPE and adjacent retinal layers distortion with undulation of the RPE and suggestion of sub retinal fibrosis and pigmentary changes in both eyes. Last seen on 2018 because of decreased vision in the right eye. It was found to have recurrence in the right eye. Recent OCT of the right eye revealed accumulation of sub retinal fluid in the right macula. Again, oral steroid was given, and her vision improved and SRF disappeared. This case represents the progression of VKH disease over a period of 10 years (Figure 12B).

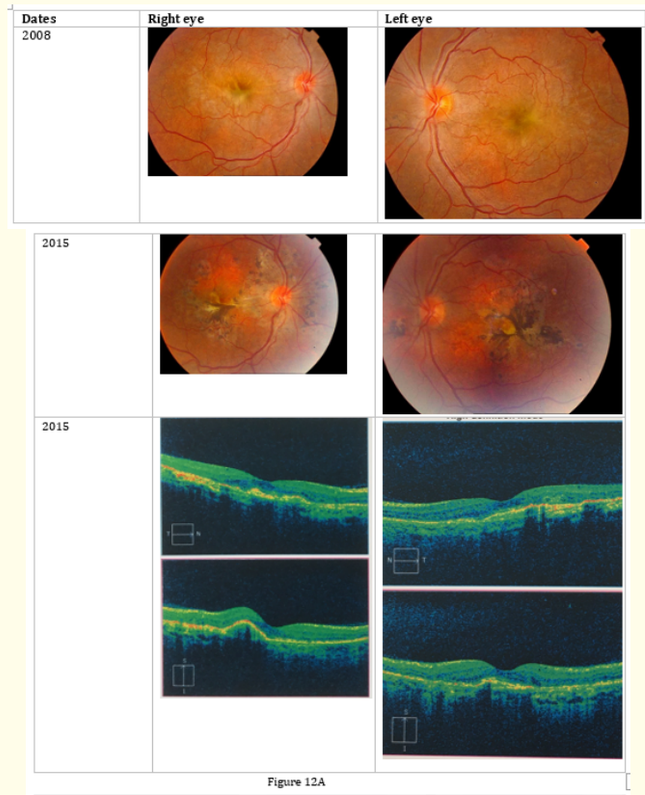

Figure 12A

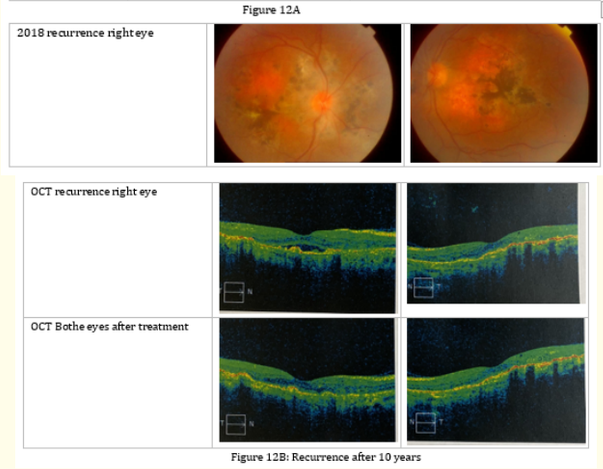

Figure 12B: Recurrence after 10 years

Figure 12: *A, B: In figure 12A 1st row showed fundus photos of both eyes after resolution of the acute stage in (2008) showing start to have pigmentary changes in both maculas. Second row showed the fundus of both eyes 7 years later (2015) showing progression of the pigmentary changes in both fundi. The third row showed the corresponding OCT 2015 of both eyes. OCT OD showed undulation and thickening of the RPE and some spots of RPE loss. OCT of the left eye showed similar changes but to a lesser extent. In figure 12B the first row showed the clinical fundus of both eyes in 2018 during the attack of recurrence in the right eye showing the continuing of progression of the pigmentary changes. The second row showed the OCT picture of both fundi beside the above mentioned RPE changes there was Subfoveal accumulation of fluid in the right eye. The 3rd row showed the OCT of both eyes showing the resorption of the SRF from right fundus after treatment.*

Discussion and Conclusion

Exudative retinopathy is a serious ophthalmic problem because of its effect on visual function. Table 2 and 3 summarise the reported conditions associated with exudative retinopathy. They include a long list of developmental, hereditary, degenerative, infectious and inflammatory conditions. The conditions that cause exudative retinopathy vary in the extent of their presentations. The inflammatory conditions cause exudative retinopathy have different degree of severity. Among the inflammatory conditions; Vogt-Koyanagi-Harada disease (VKH), Acute posterior multifocal placoid pigment epitheliopathy (APMPPE) and possible central serous retinochoroidopathy (CSC); they present as an exudative retinopathy with different degrees. They are indirectly of vascular etiology but the real underlying cause remain to be identified.

Conditions	VKH references	APMPPE references	CSC references
Autoimmune	[1-17]		[71-77]
Viral.	[18-22]	Mumps [52] Encephalitis [53,54]	
Genetics	[23-42]		[78-87]
Multiple sclerosis.	[43]		[88]
Hepatitis B vaccination	[44]		
Infection.	Tuberculosis [45]	Tuberculosis [55], Brucellosis [56], Lyme disease [57,58], Dengue fever [59]	Tuberculosis [90,91,98], anthrax vaccination [92], Helicobacter [93-104].
Ocular pathology.			Optic nerve pit [105], retinitis pigmentosa [106,107].
Vascular pathology. Including stroke cardiac, hypertension		Vasculitis [60,66], CRVO [62], stroke [63-67].	[108-110], Hypertension [111-113], stroke [114].
Ocular trauma, surgical, non surgical, laser	Posterior scleritis [46]		[115-125], Post laser [126,127]
Cancer	[203,205,207,210]		[112-115], After laser [126,127]
Drug side effect			Steroid [128-166], erectile dysfunction [167-174], deep antler spray [175], hormonal therapy [149,150,161,163]
Systemic inflammatory disease	Ulcerative colitis [50], Graves disease [51]	Wagner granulomatosis [68], Graves disease [69], Nephritis [70]	[176-183], ulcerative colitis [176,178], still disease [177], SLE [179], Crohn's disease [180], Giant cell arteritis [181], esophageal reflux [183]
Psychiatric and personality problems			[184-188]

Table 2: *Showing the associated conditions with exudative retinopathy [1-188].*

Exudative retinopathy				
Developmental	**Infectious**	**Inflammatory**	**Degenerative**	**Other conditions**

• Hereditary
• FEVR
• Norrie's disease
• Optic nerve head pit and cavitation
• Retinopathy of prematurity.
- Acute exudative polymorphous vitelliform maculopathy.
• Tumor related:
  • Malignant melanoma
  • Retinoblastoma
  • Hemangiomas
  • Haematological malignancies
  • Metastasis
	Tuberculosis
	Syphilis
	Dengue fever
	Lyme disease
	CMV retinitis
	Nematodes infections
	Fungal infections
	• Helicobacter
	• Anthrax
	• Mumps
	• Brucellosis
		• Vogt Koyonagi Harada's disease
		• Sympathetic ophthalmia
		• APMPPE
		• Posterior scleritis
		• Central serous retinochoroidopathy
		• Iatrogenic: post PRP
		• Uveal effusion
		• Serpiginous choroiditis
		• Idiopathy Unilateral acute maculopathy
		• Intermediate uveitis and other forms of uveitis
		• Relapsing poly chondrites:
		  • Vasculitis e.g. Behcet's diseases
		  • Multiple sclerosis.
			• Diabetic retinopathy
			• Age related macular degeneration
			• Hypertension retinopathy and eclampsia
			• Retinal vein occlusion
			• Coat's disease
			• Macular telangiectasia
			• Retinal schisis
				Steroid Therapy

Table 3: *Showing the list of the conditions associated with exudative retinopathy.*

In the present report; cases 1-4, represent mild form of exudative retinopathy with mild localized leaking spots involving one eye which represent what is known as central serous chorioretinopathy (CSC). CSC is a disorder characterized by serous retinal detachment and/or retinal pigment epithelial (RPE) detachment, changes most often confined to the macula. It might be presenting elsewhere in the fundus but since it will not effect vision; patients do not seek medical help and therefore they will be missed. It is usually seen in young individuals. Von Graefe (1866) in his original description; described the disease as a recurrent central retinitis [189]. Horniker in 1922 called it capillarospastic central retinitis reflecting his belief that vascular pathology was the underlying mechanism [190]. Maumenee described fluorescein angiographic (FA) characteristics of the condition as fluorescein leakage at the level of the RPE suggesting that the RPE and choroid were the primary tissues involved [191]. Gass further characterized the fluorescein angiographic findings and he gave the term central serous chorioretinopathy [192]. All agreed that vascular and RPE defect are the obvious etiology. But the underlying cause of these defects is not clear.

Table 2 revealed that CSC was found to be associated with several systemic conditions [71-188]; which include Diabetes insipidous, oral lichen platus, pituitary adenoma, intrathecal steroid for back pain, and after epidural injection, treatment of symptoms of menopaus-

al state, in renal transplant recipient, in Cushing syndrome, atopic disease, rhinitis, uveitis and use of steroid. Also, it had been reported with different infectious process (TB, Helicobacter, anthrax), systemic and pulmonary hypertension, systemic inflammatory conditions, after ocular trauma, and in cancer patients [71-188]. These will raise a suspicion that they might contribute to the occurrence of CSC in these cases. Even though; none of the present cases had an associated systemic condition but still it might be a manifestation of a systemic process which might not be clear to us, or they might be due to a local defect in RPE such as a micro rip. Many reports claim the association with the use of steroid with CSC but the mechanism was not clear [128-166]. The question can be raised that probably the under lying cause for these conditions that led to the use of steroid might be the cause of CSC. Probably the CSC cases that did not respond to steroid are the ones due to a local defect in the RPE such as micro rip.

Case 3 had bilateral mild form of CSC surprisingly that right eye responded well after 3 intravitreal injection of Lucentis while the left eye with sub RPE detachment fail to respond to 3 intravitreal injections of Lucentis. The reason of failure of the left eye to respond to treatment was not clear. Case 1 and 4 had unilateral pathology which responded well to intravitreal antivegf. While case 2 had unilateral pathology representing severe form which responded well to steroid therapy which might be a bridge between CSC and VKH suggesting a link between CSC and VKH.

Case 7 and 8 represent moderately sever form of inflammation of the retina with retinal edema and some SRF. They represent what is labelled as an acute posterior multifocal placoid pigment epitheliopathy (APMPPE); which is an uncommon, bilateral, idiopathic condition first described by Gass in 1968 [209]. APMPPE usually affects young, apparently healthy adults. It may start unilaterally and then progress to involve the second eye within days to weeks. A viral or flu-like illness; may precede the onset of symptoms. Visual complaints usually include blurred vision, scotomas, metamorphopsia and photopsia. On the initial examination of the fundus in APMPPE; there is marked by creamy gray-white lesions at the level of the RPE. On OCT, these lesions appear hyper-reflective in the thickened outer retinal layers and choroid [193]. On fluorescein angiography, the lesions appear hypo-fluorescent in early phases and more numerous than evident on clinical examination. In later phases, these active lesions become hyper-fluorescent from leakage and staining [208]. There are multiple overlapping features between VKH and APMPPE as noted by A Bird and colleagues [194] and Lee and colleagues [195]. The difference probably related to the severity and duration of the disease. Furthermore, both entities can present with serous retinal detachments that improve with pulse corticosteroid treatment [196-199].

Table 2 revealed that APMPPE was reported to be associated with different systemic conditions [52-70] which include: 1) infection with viral (mumps and encephalitis) and bacterial (Tuberculosis, Brucellosis, lyme disease and Dengue fever) 2) vascular pathology (vasculitis, CRVO, stroke) and 3) systemic inflammatory disease (Wagner granulomatosis, Grave's disease, nephritis). These may indicate that APMPPE is most probably secondary to a systemic process whether infectious or non-infectious conditions systemic or local. Cases # 7, 8 represent APMPPE with some SRF and respond to steroid may suggest a link to VKH as also noted by others [194,195].

Cases 5, 6,9-12 represent what is known as Vogt Koyangi Harada Syndrome (VKH) with different severity. VKH is a severe bilateral granulomatous posterior pan uveitis with serous retinal detachment, disk edema, vitritis. The first case of VKH was reported by Vogt in 1906 [200] and in 1926 Harada reported a case with bilateral uveitis, bilateral retinal detachment and meningeal irritation [201]. Y koyanagi in 1929 he hypothesized that the disease is caused by an anaphylactoid reaction against pigment (melanin) [202]. So, the name of VKH disease was given to this condition. Eye involvement in VKH presents clinically as an acute uveitis and chronic recurrent uveitis. They may present with blurring of vision develops in patients in both eyes, although involvement of one eye may be delayed as in case 9 [203]. Clinically it presents with a sudden onset, of bilateral granulomatous uveitis in up to 70% of patients, with subretinal fluid and choroidal thickening, blurred vision, and conjunctival injection. Signs may include swelling and hyperemia of the optic nerve head and retinal edema as in cases 5, 6, 9-12. It might be associated with hearing loss, neurological problems and skin depigmentation, which not seen in present cases.. The Neurologic signs, including headache, meningismus as in case # 11. Integumentary manifestations, including sensitivity of the scalp and of the skin to touch [204]. Sensory hearing loss and tinnitus may be present in some of these cases [205,206]. Immunological

and histopathological studies suggest that VKH is an autoimmune inflammatory condition mediated by CD4 toward the melanocytes [1-17]. Genetic susceptibility of persons expressing HLA DRB1*0405, combined with viral infection, may play a role in initiating the autoimmune process [5,207]. Chronic recurrent phase of intra-ocular granulomatous inflammation develops in some of the patients and is also marked by complications such as retinal pigment epithelium (RPE) proliferation, subretinal fibrosis and sub-retinal neovascular membrane. Case 12 represent the progression of changes in the posterior segment after resolved the acute stage of the inflammation over the period of 10 years. There were an extensive and progressive pigmentary and fibrous change in the retina, RPE and choroid in the macula of both eyes of the present case.

Table 2 revealed that VKH was found to be associated with: 1) autoimmune conditions, 2) with viral infections, 3) hepatitis, 4) multiple sclerosis, 5) tuberculosis, 6) cancer, 7) systemic inflammatory conditions such as ulcerative colitis and Grave's Disease. These will support that systemic problems might be involved in the development of VKH. Cases 5 and 6 it could be bilateral CSC or bilateral VKH from the appearance. Case 6 responded well to steroid. Case 9 had unilateral sever involvement responded well to steroid; this will support the overlap between VKH and CSC. Cases 10-12 represent acute bilateral severe form of VKH. Cases number 2, 5-12 represent different spectrum of presentation of exudative retinal detachment. All these cases responded to steroid therapy indicating that an inflammatory process was involved in all these cases.

Again looking at table 2 and 3 demonstrate some of the reported possible associated etiology with these conditions: 1) infectious both bacterial and viral were reported in all of the 3 conditions (CSC, APMPPE, VKH), 2. Systemic inflammatory conditions had been reported in all the 3 conditions, 3.vascular conditions such as hypertension had been reported with APMPPE and VKH, 4. Cancer in CSC and VKH, 5. Genetic predisposition in with CSC and VKH, 6. Systemic steroid use with CSC. It seems CSC, APMPPE and VKH are different spectrum of presentation of posterior segment inflammation due to local or part of systemic conditions. Based on the previous reports as shown in table 2 and their response to steroid therapy; they might be a different degree of presentation of different inflammatory conditions.

Posterior segment inflammation may start focally and progress vertically (choroid, RPE, retina) or horizontally within these layers or both in the retina and choroid depending on the etiology, duration before presentation, severity of the condition. If the defect confined to a small area of the RPE resulted in CSC (focal). While in case of local Choroiditis extending to RPE defects resulting in CSC with multiple defects which could be in the macula or outside the macula as in the three cases 1-4. While In cases of severe diffuse Choroiditis leading to wide RPE and retinal involvement resulting in severe form manifested as in APMPPE like picture. While In very severe form result in VKH picture. Therefore, these conditions (CSC, APMPPE, VKH) and other related ones are symptoms of a local or a systemic disease.

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