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Case Report: Case of Bilateral Panuveitis Due to Probable Vogt Koyanagi Harada Disease

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Abstract: ***The rare multisystem inflammatory disease known as Vogt - Koyanagi - Harada syndrome is typified by panuveitis with serous retinal detachment. It is frequently linked to cutaneous and neurologic symptoms such as vitiligo, headache, hearing loss, and poliosis. A widespread autoimmune reaction against melanocyte - associated antigens that begins in the choroidal stroma causes VKH illness. Although it is always bilateral, it might be asymmetrical. We are presenting a case report of 33years old female with painless diminution of vision and associated with headache since 15days. On slit lamp examination, Conjunctival congestion and cells present. Fundus examination showed, bilateral multifocal exudative retinal detachment and hyperemic and edematous disc. B scan examination revealed multifocal serous retinal detachment. On fundus fluorescein angiography, multiple pinpoint foci giving a starry sky appearance is seen.***

Keywords: Vogt Koyanagi harada, Exudative retinal detachment, Pan uveitis

1. Introduction

The autoimmune condition known as Vogt - Koyanagi - Harada (VKH) syndrome affects multiple systems. It is a non - necrotizing diffuse granulomatous uveitis condition that frequently affects the skin, inner ear, eyes, and central nervous system[1]. Though the exact etiology is uncertain, ideas suggest that a T - cell - mediated autoimmune response against one or more antigens linked to melanocytes, melanin, and retinal pigment epithelium (RPE) could be a significant culprit. Although there is no established cause, cutaneous damage or viral infection have been mentioned as potential contributing causes in certain instances.

Ocular symptoms like panuveitis, serous or exudative retinal detachment, "sunset glow fundus" with optic disk hyperemia, neurologic symptoms like headache, tinnitus, meningitis, and cranial nerve palsies, and dermatological symptoms like vitiligo, poliosis, and alopecia are some of its defining characteristics. [1]

There are four distinct clinical stages of the VKH syndrome:

1) A prodromal stage marked by neurological symptoms including headaches and muscle weakness as well as nonspecific symptoms like fever, nausea, and vertigo;
2) Individuals who complain of central scotoma (bilateral in 80% of cases), photophobia, ocular discomfort, or blurred vision; Hearing problems may also be present, and bilateral serous retinal detachment frequently occurs
3) The convalescent phase, which lasts for a few months after the commencement and is marked by vitiligo, hair loss, and poliosis affecting the eyelashes and eyebrows
4) Chronic recurrent stage: recurring eye problems and uveitis[2]

Other issues that frequently occur during the recurrent phase include retinal pigment epithelial proliferation, subretinal fibrosis, subretinal neovascular membranes, posterior sub - capsular cataract, posterior synechiae, and open and closed angle glaucoma. Recurrences frequently occur when steroid therapy is stopped too soon or too abruptly, or when tapering is done too quickly. [3]

2. Case Report

A 33years old female patient came with chief complaint of diminusion of vision in both eyes which was gradual in onset progressive and painless in nature and associated with headache since 15days.

On ocular examination, best corrected visual acuity in both eyes is 6/12. On Slit lamp examination, conjunctival congestion and ciliary congestion, active anterior chamber inflammation with cells 1+. Intraocular pressure measured using Non contact tonometry was 18.4mmhg and 16.7mmhg. On dilated fundus examination of Both eye Disc appears hyperemic and edematous, vessels appear tortuous and dilated, ILM folds were present around the macula, mutli focal serous retinal detachments were present.

OCT scan showing a hyperreflective foci in the vitreous and multifocal exudative retinal detachment with hyperreflective foci in the subretinal fluid. B - Scan showing similar findings of multifocal retinal detachments and increased chorodial thickness in RE 2.53mm and in left eye 2.48mm. On Fundus Fluorescein Angiography of both eye, in early phase multiple pinpoint foci of hyperfluorescene and disc hyperfluorescene and in late phase increase in pinpoint hyperfluorescence and coalescence resulting in pooling of areas of RD areas and optic disc leakage.

ENT examination and Dermatological examination was within normal limit

Laboratory Examination: High inflammatory markers (Complete blood count including white blood cells count, erythrocyte sedimentation rate, and C - reactive protein)

Patient was started on systemic corticosteroid therapy with pulse dose I. V Methylprednisolone 1gram for 3days and then oral Prednisolone 1mg/kg on tapering doses with Oral Azathioprine 50mg and Folinic acid 5mg. Topical drops with a fixed combination of Gatifloxacin - Prednisolone, mydriatic drops.

On 1month follow up, patient is symptomatically better with BCVA 6/12 in both eyes. On Slit lamp examination, anterior segment is quiet and on fundus examination disc appears normal with no serous retinal detachment in booth eyes. On OCT examination confirmed the same.

3. Discussion

The clinical diagnosis of VKH, a multisystem autoimmune illness, is based on the presence of ocular symptoms and the lack of ocular damage or surgery. The international updated criteria of VKH, which include complete VKH, incomplete VKH, and probable VKH, are the most widely used for diagnosing it.

Complete VKH is defined as bilateral involvement with neurologic or auditory findings and integumentary findings

Incomplete VKH is defined as bilateral involvement with neurologic or auditory findings or integumentary findings

Probable VKH is defined as bilateral ocular involvement only. These new diagnostic criteria were established by the First International Workshop on VKH syndrome. [4]

The patient's diagnosis of likely VKH syndrome was made because there was no neurological or dermatological involvement, only ocular involvement.

Our patient's BCVA was after a proper diagnosis and tailored treatment. It is rare to diagnose probable VKH syndrome, but complete VKH syndrome with integumentary signs is frequently observed and treated. Consequently, we would like to report this instance. Since the patient had a headache and blurred vision, prompt identification and treatment of likely VKH syndrome were crucial for symptomatic therapy and obtaining BCVA because the patient was younger.

In another case report by Kervi n mehta et al, 32years female presented with diminution of vision since 6months associated with headache. On slit lamp examination presence of multiple old mutton fat keratic precipitates and loss of iris colour and pattern and posterior synechiae present. Fundus examination showed depigmentation with bright red - orange choroid and pale disk suggestive of sunset glow fundus. [2]

In the past, our patient had no clinical signs of immunological recovery uveitis or ocular damage. However, the duration of this disease was shortened by our early identification and effective therapy.

4. Conclusion

VKH syndrome is a common cause of chronic ocular inflammation. Extensive physical and Ophthalmologic examination is essential to diagnose this entity, because it encompasses not only eyes but also the nervous and the integumentary systems. Ocular inflammation in this multisystemic immune disorder may lead rapidly to irreversible blindness if adequate sufficient therapy is not initiated promptly. Corticosteriods should be used to achieve inflammatory control.

Disclosure
The Authors report no conflict of interest in this work.

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Nil

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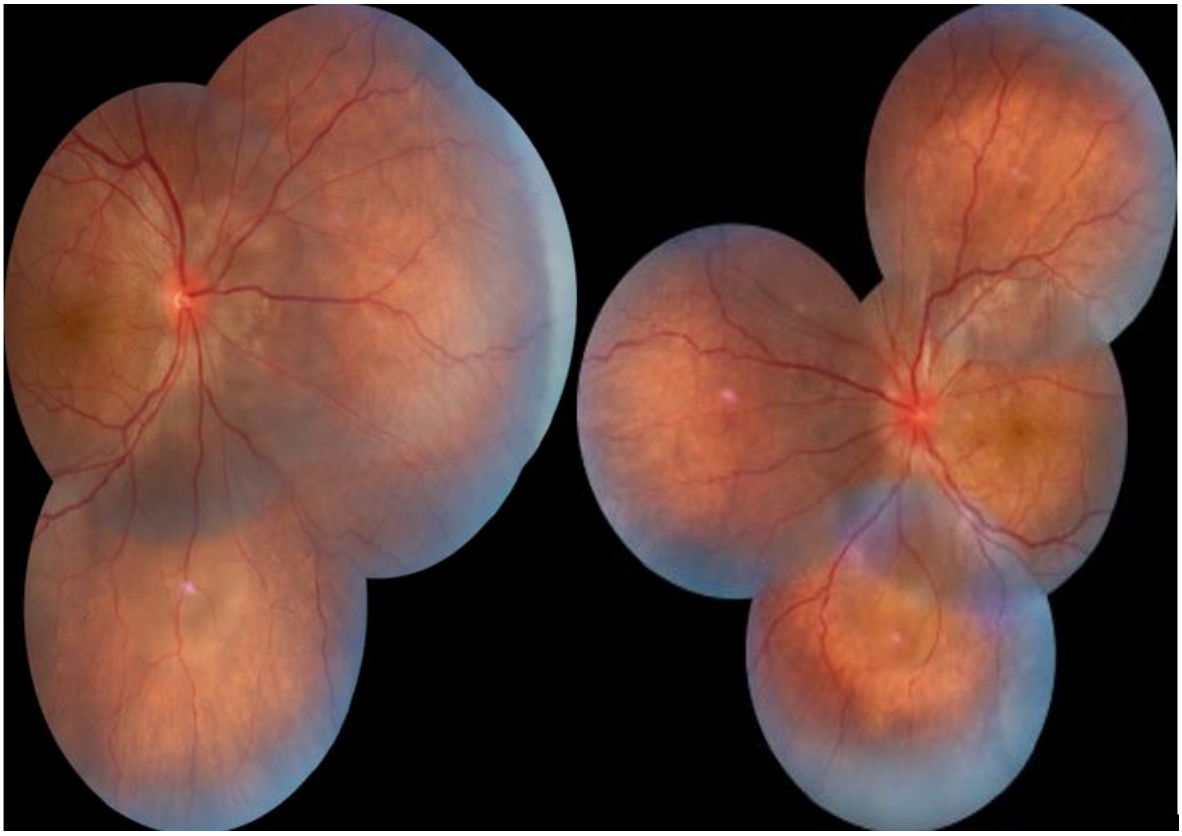

Figure 1: Right Eye & Left Eye: showing mutifocal serous RD with hyperemic disc

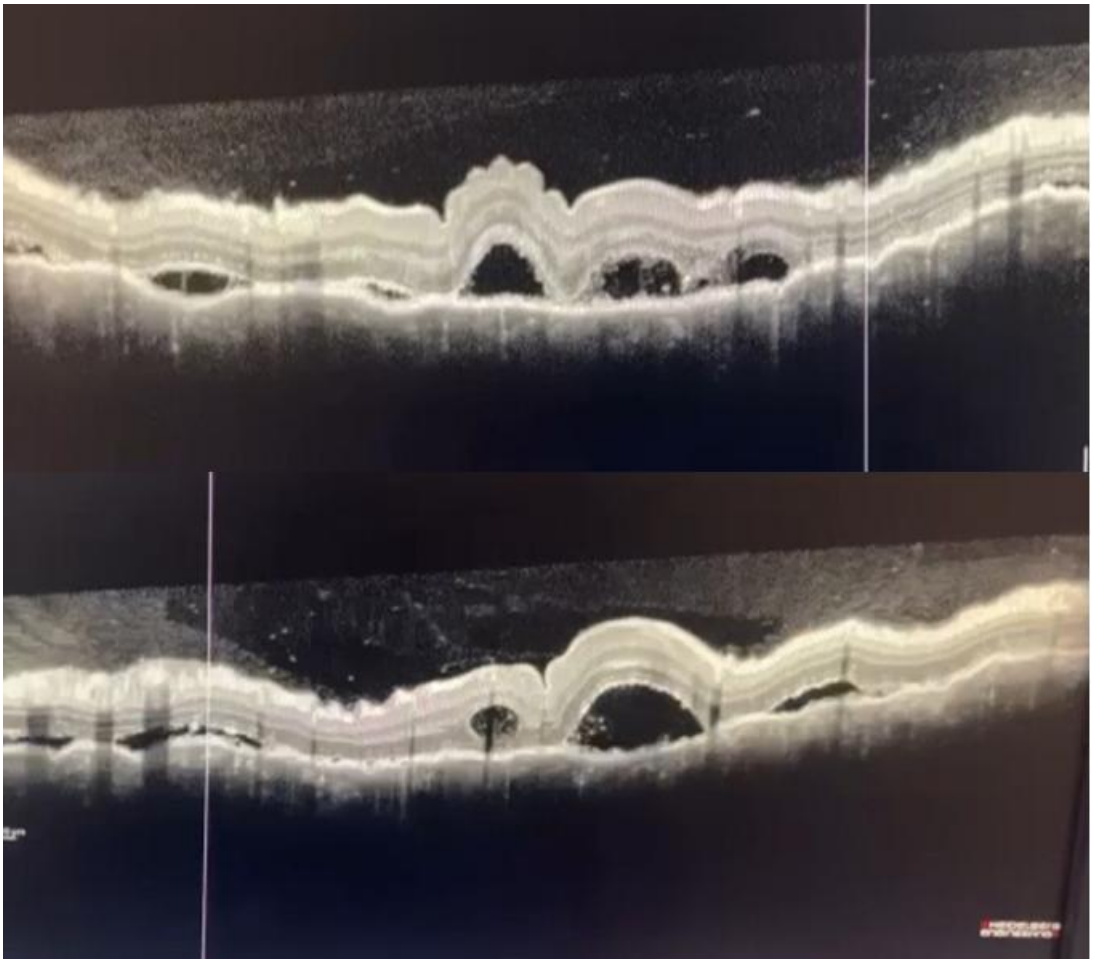

Figure: 2: 1. Right Eye, 2. Left eye OCT showing a hyperreflective foci in the vitreous and multifocal exudative retinal detachment with hyper - reflective material within the subretinal fluid.

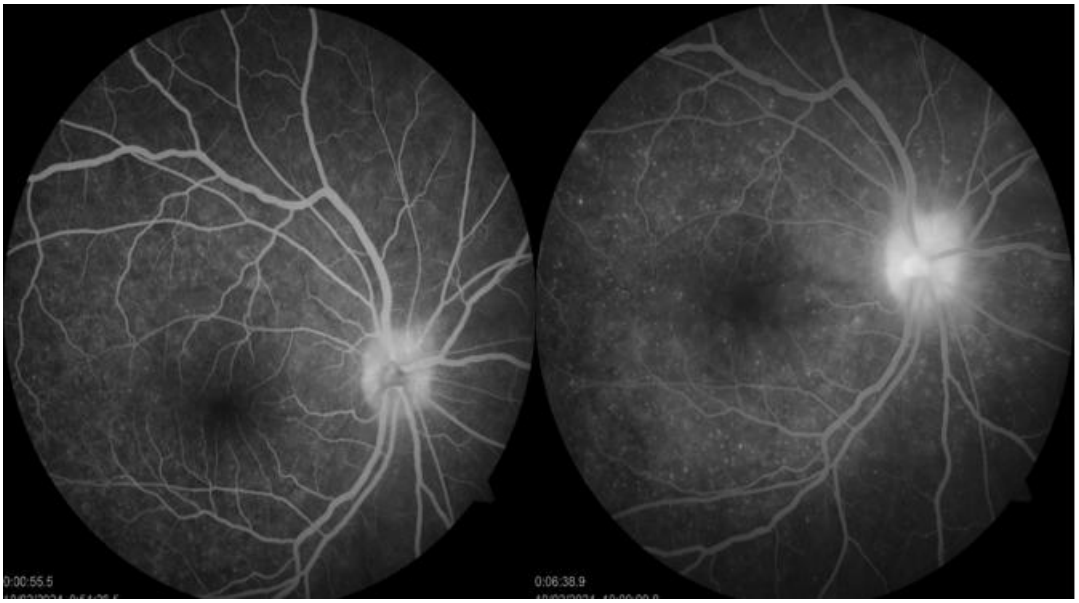

Figure 3: FFA: Right eye:
In Early phase - Multiple Pin point foci hyperfluorescence with disc hyperflourescence.
In Late stage - Increase in pinpoint hyperfluorescence and Coalescence resulting in pooling of the contrast in exudative RD areas and Optic disc leakage

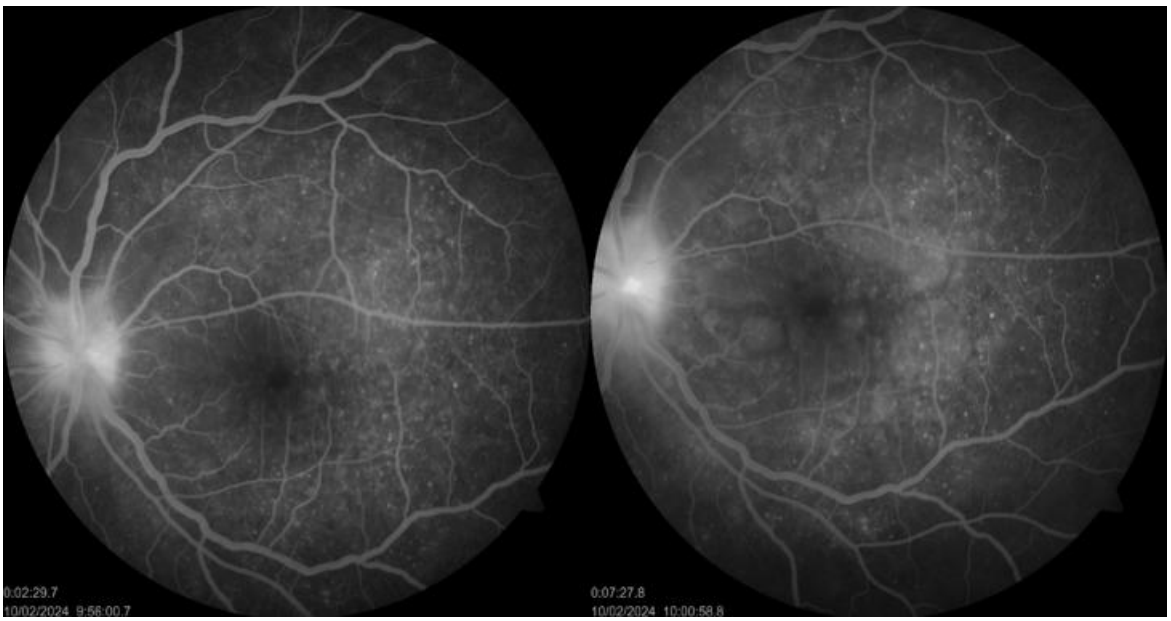

Figure: 4: FFA: LE
In Early phase - Multiple Pin point foci hyperfluorescence with disc hyperflourescence.
In Late stage - Increase in pinpoint hyperfluorescence and Coalescence resulting in pooling of the contrast in exudative RD areas and Optic disc leakage