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Vogt-Koyanagi-Harada (VKH) Disease

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Abstract

Background: Vogt-Koyanagi-Harada (VKH) disease is a rare, multisystem autoimmune disorder characterized by bilateral granulomatous panuveitis, with or without extraocular manifestations. Although its exact etiology and pathogenesis remain unclear, it is hypothesized to involve T-cell dysregulation targeting melanocyte-containing tissues, including the CNS, eye, ear, and skin. VKH predominantly affects pigmented groups, such as Asians, Hispanics, Indians, Native Americans, and Mediterranean ethnicities, accounting for 7-22.4% of uveitis cases. Retrospective analyses indicate a higher incidence among female patients, with most cases occurring in the second and fifth decades of life. Aim: This case report discusses a patient with probable VKH who exhibited ocular, neurologic, and auditory symptoms typical of the prodromal or acute uveitic phase and responded well to prompt management. Case Presentation: A young female in her late 20s presented with low-grade fever, severe headache, neck pain, and neck stiffness. She had received symptomatic treatment at another hospital without relief. She was empirically started on intravenous antibiotics and dexamethasone for suspected pyogenic meningitis and was discharged upon symptom relief. However, she returned two days later due to symptom recurrence. Ophthalmic examination revealed decreased visual acuity bilaterally (6/24), sluggish pupil reaction, optic disc edema, and bilateral macular serous detachments. Mild vitritis with anterior chamber cells and iris pigment on the anterior lens capsule was noted in the left eye. Systemic examination was unremarkable, except for fine crepitations in the bilateral lower lung fields. Management: Considering VKH disease, the patient was started on intravenous methylprednisolone pulse therapy (1 gram/day) for 3 days, followed by oral steroids and topical steroid drops for the eyes. She was discharged with oral prednisolone and prednisolone acetate 1% eye drops. At follow-up, her vision improved, and there was resolution of papillitis and serous retinal detachments. Conclusions: VKH is a significant cause of bilateral vision loss. This case of probable VKH syndrome underscores the importance of early recognition and aggressive treatment in achieving a favorable visual prognosis.

Keywords

VKH Syndrome, VKH Disease, Vogt Koyanagi Harada

1. Introduction

Vogt-Koyanagi-Harada (VKH) disease is a rare, multisystemic, autoimmune disorder of inflammatory origin. It is defined as a bilateral granulomatous panuveitis with or without extraocular manifestations. It is defined as a bilateral granulomatous panuveitis with or without extraocular manifestations. Though the exact aetiology and pathogenesis is yet to be cleared, it is hypothesised as a dysregulation of T-cells targeting the melanocyte-containing tissues including the CNS, eye, ear and the skin [1] [2].

In the 19th century, this disease was termed by Professor Babel of the Department of Ophthalmology at Geneva University Hospital based on previously independently recognised manifestations of the same disease process by Swiss physician Vogt and Japanese researchers Koyanagi and Harada. VKH characteristically affects pigmented groups, such as Asian, Hispanic, Indian, Native American and Mediterranean ethnicities, and constitutes for 7% - 22.4% of uveitis patients [3].

Several retrospective analyses based on medical records of VKH patients conducted in the Middle East have revealed a higher incidence among female patients compared to males, which is evident in studies conducted in Egypt [4], Saudi Arabia [5], Türkiye [6], as well outside of Middle East such as in India [7]. This gender disparity aligns with the broader pattern observed in many systemic autoimmune diseases. However, studies in certain countries like China [8] and Singapore [9] indicate no significant gender-based variations in VKH incidence. The majority of cases appear to be around the second and fifth decades of life.

We encountered one patient with probable VKH who exhibited ocular, neurologic and auditory symptoms as commonly observed in the prodromal or acute uveitic phase with prompt management.

2. History

Our patient is a young female in her late 20s who initially complained of sudden-onset headache, low-grade fever and neck stiffness on August 28th, 2023. The headache was characterized as band-like, spanning the entire head and scalp, with radiation to the neck and spine. It was associated with pronounced neck stiffness exacerbated by movement. She reported generalized weakness of her body, predominantly affecting the upper and lower limbs, along with a decreased appetite due to severe nausea. The patient also complained of constant dizziness which increased with any positional changes.

The patient further reported ocular symptoms, including mild, constant, dull pain, blurry vision, redness, and the presence of floaters in both eyes. Mild hearing loss was also reported without ear pain or tinnitus. She denied any history of

trauma but had complaints of flu-like symptoms preceding the onset.

The patient initially visited another hospital on August 28th, where she was given symptomatic treatment with acetaminophen, premosan and pantoprazole. However, no improvement was observed and her symptoms continued to deteriorate. On September 3rd, she was admitted to our hospital. A lumbar puncture was performed which was equivocal for pyogenic meningitis, and the patient received empiric antibiotics, including Ceftriaxone 2 gm and intravenous amikacin 500 mg twice daily. She was also given steroids in the form of intravenous dexamethasone. Following symptom relief, she was discharged on September 09th. She re-presented to our Emergency Department on September 11th due to the recurrence of neck stiffness, headache, severe nausea and vomiting. Additionally, she reported mild dull pain in her abdomen, exacerbated by inspiration.

3. Clinical Course

On admission, she was afebrile, pulse rate 102 beats per minute and blood pressure 112/60 mmHg. Besides decreased visual acuity bilaterally 6/24, ophthalmologic examination revealed bilateral sluggish pupil reaction. Further fundoscopic examination revealed bilateral optic disc edema with bilateral serous detachments at macula of pigment epithelium and mild viritis on left eye with anterior chamber cells and iris pigment on anterior lens capsule. Lung examination was positive for fine crepitations on both lung bases. Range of neck movement were full with pain but no rigidity. Neurological examinations revealed no abnormalities. Laboratory investigations remarkable for high white blood cell count of 18.5 and therefore started on intravenous ceftriaxone 2 g twice daily. The lumbar puncture initially performed due to suspicion of meningitis might have shown elevated white cell count and protein, but these are nonspecific. Based on the findings from history, examination, and investigations, alternative diagnoses such as infectious meningitis, other autoimmune or inflammatory conditions (e.g., SLE, sarcoidosis), and idiopathic intracranial hypertension were systematically ruled out. The constellation of symptoms (meningismus, ophthalmic findings) and the lack of complete response to empirical treatment prompted consideration of VKH syndrome. We then collaborated with ophthalmologists and drawing from their expert clinical judgement, we reviewed the diagnostic criteria for VKH syndrome. Upon consideration of VKH disease, antibiotics were stopped and she was commenced intravenous methylprednisolone pulse therapy (1 gram/day) for 3 days followed by oral steroids with topical steroids for eyes. Cerebrospinal fluid (CSF) analysis showed pleocytosis with lymphocytic predominance. The patient was clinically improving with no neck pain or headache on discharge. She was discharged with oral prednisolone and prednisolone acetate 1% eye drops.

We used OCT imaging to monitor our patient's response to corticosteroid therapy. Initially, when our patient was admitted, the OCT showed bilateral serous macular detachments and optic disc swelling (papillitis), as seen in **Figures 1-4**.

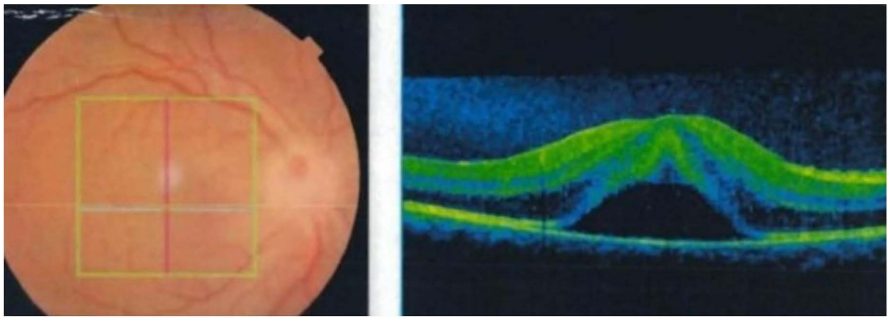

Figure 1. Optical coherence tomography showing right eye serous macular detachment.

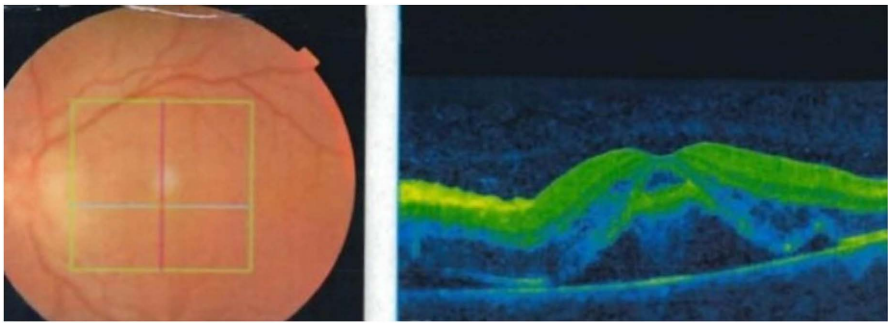

Figure 2. Optical coherence tomography showing left eye serous macular detachment.

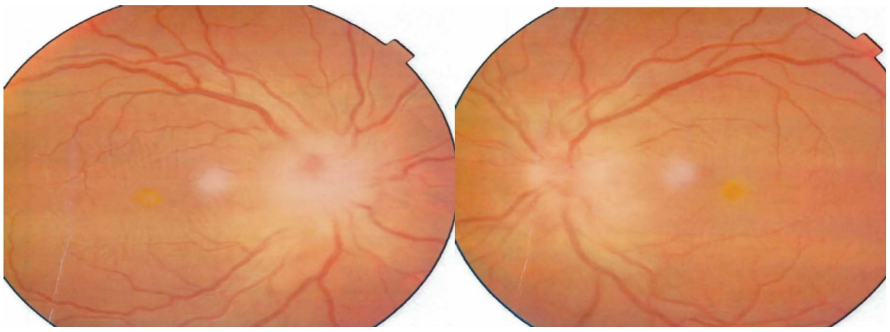

Figure 3. Fundus picture showing bilateral disc swelling-papillitis.

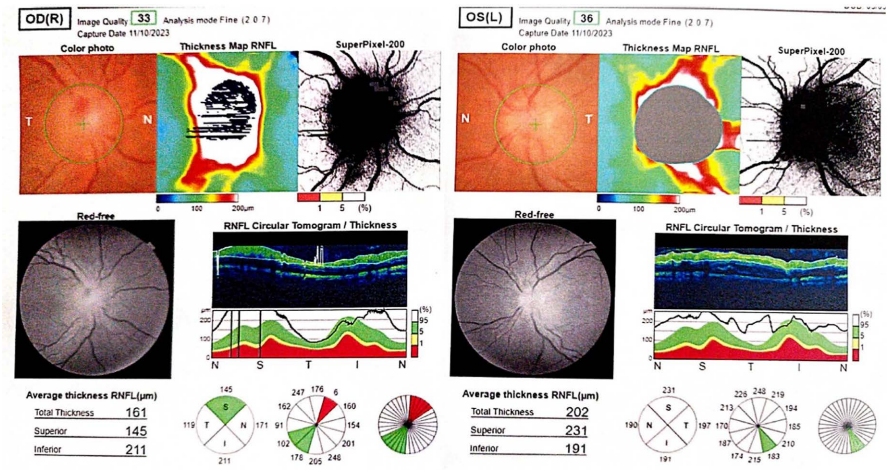

Figure 4. Optical coherence tomography showing bilateral disc swelling—papillitis.

Three weeks post-admission, during follow-up, the patient was afebrile, free of headaches, and had improved vision with occasional episodes of blurring. Visual acuity improved to 6/9 for both the eyes, and the OCT findings showed bilateral resolution of serous retinal detachment as well as papillitis, as seen in **Figures 5-8**, indicating a successful response to our steroid therapy. The patient's positive response to steroid therapy, including the resolution of neurological symptoms and improvement of ocular manifestations, is a hallmark feature of VKH syndrome, further solidifying that the correct diagnosis was made.

At this point we lost contact with the patient as she left to continue treatment elsewhere. However, we provided the patient with a summary of her condition, treatment received, and recommendations for ongoing management, to ensure continuity of care.

In terms of long-term management, we recommend gradual tapering of steroids once inflammation is controlled and some patients may require long-term maintenance with immunosuppressive therapy. The length of treatment and subsequent taper must be individualized for each patient. Patients should undergo regular ophthalmic monitoring to monitor for recurrence of uveitis and assess visual acuity. They should also be monitored for complications due to long-term steroid therapy including glaucoma and cataract. Continued vigilance is necessary as VKH syndrome can have a relapsing-remitting course, requiring ongoing evaluation and adjustment of treatment based on disease activity and patient response.

4. Discussion

VKH disease is an important cause of noninfectious uveitis. Immunological and histopathological studies suggest that VKH is an autoimmune inflammatory condition aimed at proteins associated with melanocytes. Histopathologic findings and in vitro experiments demonstrated the role of CD4+ T lymphocytes. These T cells likely initiate the inflammatory process through generation of cytokines, IL 17 and IL 23, in individuals with altered tolerance to melanocytes from deficient T regulatory cells. In vitro, uveal pigment inhibited leukocyte migration of peripheral blood mononuclear cells (PBMC) from patients with VKHD, and both CD4+ and CD8+ T lymphocytes were cytotoxic against melanocytes in vitro. Moreover, Norose *et al.* described cytotoxicity displayed by lymphocytes from PBMC and CSF of patients with VKHD against the B-36 melanoma cell line [10]. The trigger for this autoimmune response, although still not known, may be a combination of genetic susceptibility and viral infection. Several studies have demonstrated that HLA-DR1, DR4 and DRB1 is associated with VKHD patients of different ethnic groups with susceptibility varying from 11.76 to 45.1 [11].

The clinical manifestations of VKH syndrome can be categorized into four main stages, namely the prodromal stage, the anterior uveitic phase, the convalescent or chronic phase, followed by the chronic recurrent phase. The prodromal phase mostly constitutes of flu-like illness, where patients primarily present with headache, nausea, vertigo, fever, meningismus and orbital pain. Furthermore,

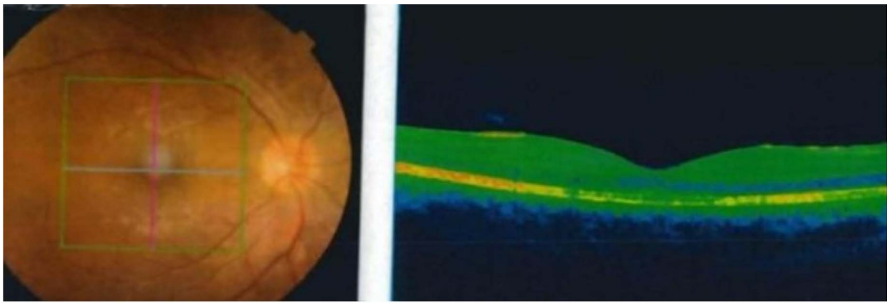

Figure 5. Optical coherence tomography showing right eye resolution of serous retinal detachment.

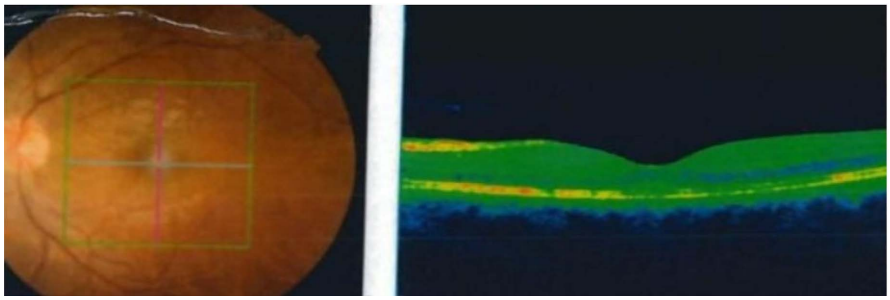

Figure 6. Optical coherence tomography showing left eye resolution of serous retinal detachment.

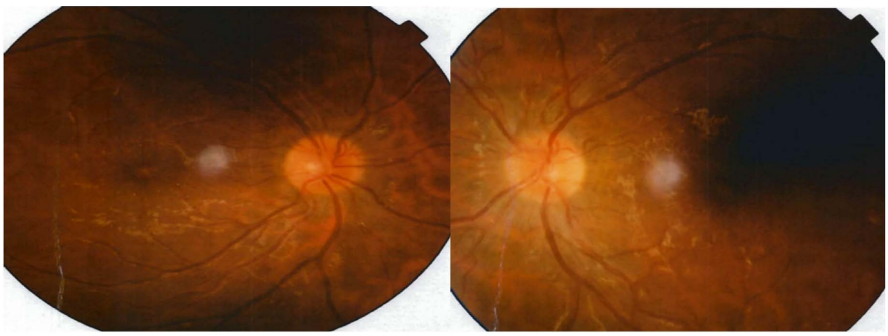

Figure 7. Fundus picture showing resolution bilateral disc swelling—resolved papillitis.

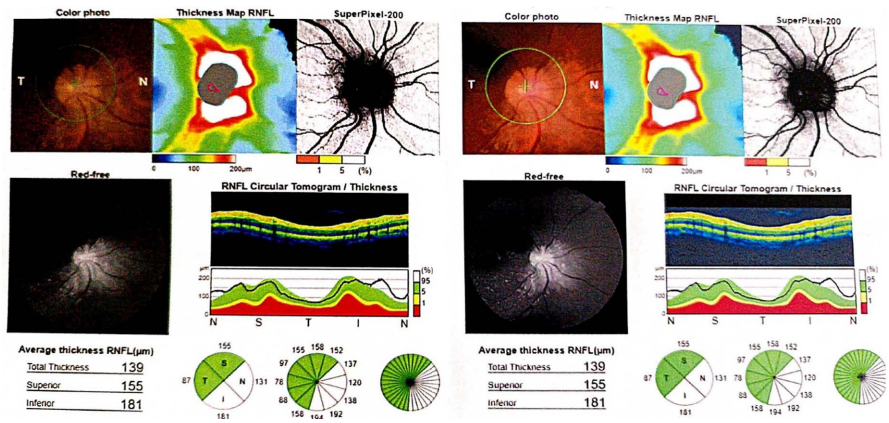

Figure 8. Optical coherence tomography showing resolution bilateral disc swelling—resolved papillitis.

pleocytosis may be observed upon CSF analysis in this stage. The uveitic phase is where the early ocular manifestations are seen, including serous retinal detachments multifocally and thickening of the choroid. If left untreated, this can quickly progress into granulomatous uveitis of the anterior chamber, followed by progressive choroidal depigmentation in the posterior segment, also termed as "sunset-glow fundus" [12].

The diagnosis is primarily based on clinical features. Several criteria have been proposed, including the American Uveitis Society (AUS) in 1978 and the Sugiura's Criteria in 1976. AUS criteria requires: 1) the absence of any history of ocular trauma or surgery; and 2) the presence of at least three of the following four signs: a) bilateral chronic iridocyclitis; b) posterior uveitis, including exudative retinal detachment, forme fruste of exudative retinal detachment, disc hyperemia or edema and "sunset-glow" fundus; c) neurologic signs of tinnitus, neck stiffness, cranial nerve, or central nervous system disorders, or cerebrospinal fluid pleocytosis; and d) cutaneous findings of alopecia, poliosis, or vitiligo [13]. Our patient denied any history of ocular trauma or surgery and met the first three of the four signs listed. VKH affects pigmented structures, such as the eye, inner ear, meninges and skin. The initial presentation of meningism was remarkably more common than expected, with meningism being the most common extraocular manifestation in approximately 64% of cases according to one study conducted in South India [14]. Hence, it was unsurprising that our patient presented similarly. Changes in the inner ear, such as hearing loss, vertigo, tinnitus, have also been observed especially during the prodromal phase.

Although the diagnosis is principally clinical, newer imaging modalities allow for quantifying inflammation and improved assessment of treatment efficacy. Multimodal imaging has provided crucial information in the diagnostic approach in typical, atypical presentations of VKH, and excluding conditions mimicking VKH. This encompasses non-invasive methods like fundus photography, OCT, B-scan ultrasonography, and invasive methods like fundus fluorescein angiography (FFA) and indocyanine green angiography (ICGA).

During the acute phase, OCT imaging may show exudative retinal detachment, undulations of inner retinal layers, a thickened choroid, fibrinous septa in the subretinal space, and folds of the retinal pigment epithelium (RPE) without RPE detachments. Enhanced depth imaging spectral domain OCT, which visualizes choroidal thickness, can evaluate disease stage, monitor treatment efficacy, and indicate disease recurrence. The choroid is thinner during the convalescent phase, in longstanding disease, and after initiating corticosteroid treatment. In the chronic phase, progressive choroidal thinning with loss of small choroidal vessels and stromal scarring occurs. Eyes with RPE undulations are more likely to develop posterior recurrences and have worse vision at 12 months. Thus, OCT is a valuable technology for understanding the disease process [11]. Optical coherence tomography angiography (OCTA) is another useful non-invasive tool for detailed reconstructions of retinal-choroidal microvasculature, using intraluminal blood

flow as inherent contrast [15]. Recent evidence suggests that OCTA effectively identifies choriocapillary-level changes in acute VKH, making it valuable for monitoring disease progression, regression, and relapse alongside clinical exams and other imaging modalities [16].

Treatment is based on reducing inflammation with prompt use of systemic corticosteroids at a dose of 1 - 1.5 mg/kg per day for a minimum of 6 months. Lai TY *et al.* reported that patients receiving treatment for less than 6 months were much more likely to have recurrences compared to those treated for 6 months or more [17]. Chee and colleagues observed that the majority of patients who received high-dose corticosteroids within the first two weeks of disease onset experienced complete resolution of inflammation, in contrast to patients who received treatment between two to four weeks, all of whom developed chronic or recurring disease [18].

Despite systemic corticosteroids being the main therapy, studies have explored alternative or additional treatments for VKH disease. Recent research highlights the potential of subtenon triamcinolone injections as primary VKH treatment for patients lacking systemic signs, though their long-term application requires further investigation [18]. Paredes *et al.* found improved visual outcomes with prompt initiation of immunomodulatory therapy (IMT) compared to steroid monotherapy or delayed IMT commencement [19], while Urzua *et al.* noted significant benefits from early IMT initiation in a specific subset of corticosteroid-resistant patients [20]. For VKH patients unresponsive to standard immunosuppression, biological response modifiers like adalimumab, rituximab, and infliximab have demonstrated good efficacy in several trials [21].

The development of complications is associated with the chronicity and greater number of recurrences which is linked with worse visual outcomes. Various studies unanimously identified cataract as the most common complication in VKH patients (30% - 47%), followed by glaucoma (6% - 29%) and subretinal neovascularization or fibrosis (2% - 17%) [11].

Recently, similar cases have been reported in female patients who developed VKH secondary to COVID-19. In a report published in the Indian Journal of Ophthalmology, a 23-year-old female presented with VKH characterized by bilateral serous retinal detachment and disc hyperemia after a recent COVID-19 infection. She responded well to oral corticosteroids and was classified as probable VKH based on revised international diagnostic criteria, without neurological or integumentary findings [22]. Another case involved a 29-year-old female with incomplete Vogt-Koyanagi-Harada disease associated with COVID-19, presenting with gradual vision loss, bilateral tinnitus, and ocular pain.

Exacerbated by eye movements. Examination revealed optic nerve swelling, retinal folds, and bilateral serous retinal detachment. Treatment with intravenous methylprednisolone (1 gram daily for 3 days), followed by oral prednisolone (1 mg/kg), successfully resolved her symptoms and ophthalmological findings [23].

Our case report stands out as there haven't been any published instances of

VKH syndrome in UAE patients, making its diagnosis challenging due to its rarity in this region. The rarity of this condition may lead to a lack of awareness among healthcare providers which can result in delayed diagnosis or misdiagnosis, as the condition may not be initially considered in the differential diagnosis of patients presenting with characteristic symptoms. Our goal is to increase awareness among healthcare providers, leading to improved rates of diagnosis and better outcomes for patients.

5. Conclusion

Our patient was a case of probable VKH syndrome, presenting in the prodromal or anterior uveitic phase, as verified by the clinical criteria and supported by imaging. With meticulous treatment with corticosteroids and regular follow up, she has shown great improvement. She must continue to be followed up closely as there is a high chance of recurrences and complications which can potentially be sight-threatening.

Ethics Statement

A written informed consent for the publication of the case details and any accompanying images were obtained from the patient involved in the case.

Conflicts of Interest

The authors confirm that they have no conflicts of interest pertaining to the publication of this paper.

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