Clinical Case Reports International

Case Report
Published: 05 Jun, 2023

Vogt-Koyanagi-Harada Disease One Week after the COVID-19 Onset: A Case Report

Muto T[1,2], Sakamoto M[1], Machida S[1] and Imaizumi S[2]*

[1]Dokkyo Medical University Saitama Medical Center, Saitama, Japan

[2]Imaizumi Eye Hospital, Japan

Abstract

Purpose: Herein, we report a case of Vogt-Koyanagi-Harada (VKH) disease in a patient after the Coronavirus Disease 2019 (COVID-19) infection.

Case and Findings: A 57-year-old male patient presented to our hospital with a visual disturbance in both eyes that had started 3 weeks ago, accompanied by a headache. Four weeks prior to the presentation, the patient had tested positive for severe acute respiratory syndrome coronavirus-2, and the cough continued as an aftereffect. The Best-Corrected Visual Acuity (BCVA) was 1.0 and 0.6 in the right and left eyes, respectively. Fundoscopy demonstrated bilateral serous retinal and choroidal detachments in the left eye. Optical coherence tomography revealed bilateral serous retinal detachment and choroidal thickening. Fundus fluorescein angiography revealed multiple pinpoint leaks. This patient was diagnosed with a serous retinal detachment type of VKH disease after COVID-19. Steroid pulse therapy was immediately started, which had a good treatment response in the patient. The BCVA improved to 1.2 in the both eyes 2 months after starting the therapy.

Conclusion: COVID-19 infection could be a possible trigger factor for VKH disease. Because the exact mechanism for the development of VKH disease remains unclear today, it should be elucidated urgently.

Introduction

Vogt-Koyanagi-Harada (VKH) disease occupies 8.1% of uveitis in Japan, ranking second only to sarcoidosis [1]. The exact etiology of VKH disease remains unknown. However, T cell-mediated autoimmune responses against melanocyte-related antigens are thought to be involved in VKH development [2]. In the acute stage, bilateral panuveitis caused by choroidal inflammation, inner ear disorder, and meningitis are frequently observed. The chronic or late stages are characterized by granulomatous uveitis, sunset glow fundus, vitiligo, and gray hair.

The Coronavirus Disease 2019 (COVID-19) caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is the latest pandemic infection, which lasted for approximately three years in Japan. The ocular manifestations of SARS-CoV-2 infection include chemosis, epiphora, and conjunctival hyperemia [3]. Optic neuritis, uveitis (Benito BP), [4]. and VKH disease [5-8] have been reported after SARS-CoV-2 infection as well. However, under the circumstances, we cannot determine whether the onset of VKH disease after COVID-19 was caused by chance. Santaramria et al. assumed that SARS-CoV-2 might be a trigger for developing VKH disease [6].

Herein, we report a case of a middle-aged male with symptoms of VKH disease, probably triggered by SARS-CoV-2, 1 week after COVID-19 affection, which, to the best of our knowledge, has not been previously reported in Japan.

Case Presentation

A 57-year-old male patient presented with headache and bilateral diminution of vision for 3 weeks. He had no history of tinnitus. Four weeks before presentation, he was diagnosed with COVID-19, as confirmed with a positive reverse transcriptase polymerase chain reaction of a nasopharyngeal swab.

The initial examination revealed the Best-Corrected Visual Acuity (BCVA) to be 1.0 and 0.6 in the right and left eyes, respectively. The intraocular pressure was 14 mmHg bilaterally. Slit-

OPEN ACCESS

Correspondence:
Tetsuya Muto, Department of Ophthalmology, Imaizumi Eye Hospital, 20-9 Domaecho, Koriyama, 963-8877, Japan, Tel: +81 24 922 0665; Fax: +81 24 935 5550;
E-mail: ueda.castle@gmail.com

Received Date: *15 May 2023*
Accepted Date: *31 May 2023*
Published Date: *05 Jun 2023*

Citation:
Muto T, Sakamoto M, Machida S, Imaizumi S. Vogt-Koyanagi-Harada Disease One Week after the COVID-19 Onset: A Case Report. Clin Case Rep Int. 2023; 7: 1554.

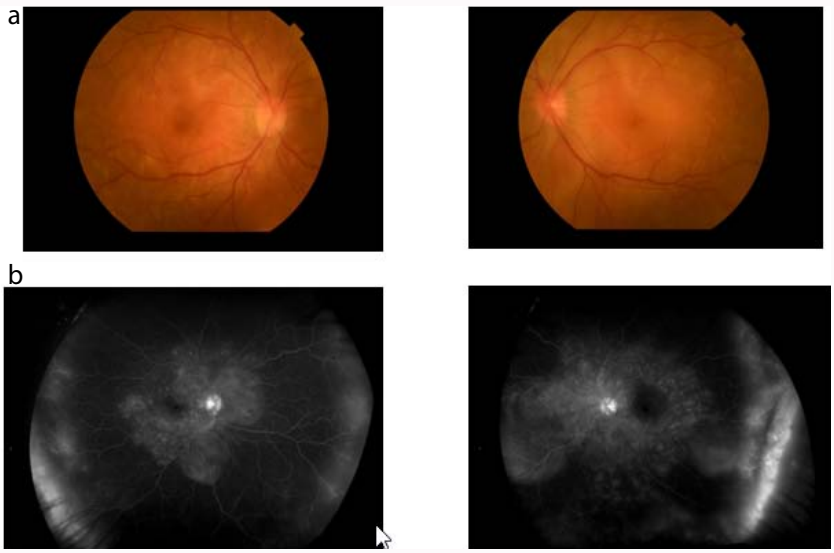

Figure 1: Eye examination images from a 57-year-old male patient with decreased vision in both eyes and a headache 1 week after COVID-19 infection, showing (a) retinal white bulges caused by serous retinal detachment are seen in both eyes and (b) serous retinal detachments with fluorescein angiography demonstrate late leakage with multiloculated pooling in both eyes. Choroidal detachment is found temporal direction in the left eye.

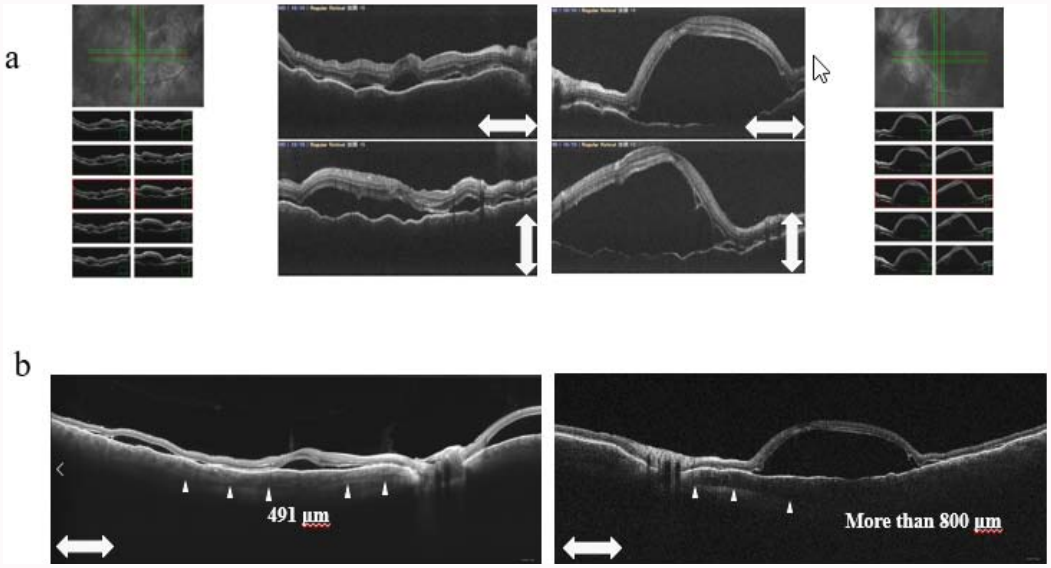

Figure 2: Optical coherence tomography images from a 57-year-old male patient with decreased vision in both eyes and a headache 1 week after COVID-19 infection, showing (a) serous retinal detachments and retinal pigment epithelium undulation in both eyes and (b) choroidal thickenings in both eyes. The borderline between choroid and sclera is unclear in the left eye because of choroidal thickening.

lamp examination of the anterior segment was within normal limits bilaterally, including the absence of cells and flare. Dilated fundoscopy demonstrated multifocal serous retinal detachments without vitritis in both eyes (Figure 1a) and choroidal detachment in the left eye (Figure 1b).

Fluorescein angiography revealed dot hyperfluorescence and multiple areas of pinpoint hyperfluorescent foci with later-phase pooling in both eyes. Spectral-domain Optical Coherence Tomography (OCT) (RS-3000 advance, Nidek Corporation, Japan) and swept-source OCT (PLEX Elite 9000, Carl Zeiss Meditec AG, Jena, Germany) confirmed a series of morphological changes (bullous serous retinal detachment, Retinal Pigment Epithelium [RPE] undulation, and significant choroidal thickening and the development of a membranous structure on the RPE beneath the foveal cystoid space in both eyes (Figure 2a, 2b).

The diagnosis was acute onset of VKH disease after COVID-19 infection. Steroid pulse therapy was started by infusing methylprednisolone (Solu-Medrol; Pfizer, New York, NY, USA) at 1000 mg/day for 3 days, followed by oral prednisolone (Predonine; Shionogi, Osaka, Japan) administration that was started at 40 mg/day and tapered by 5 mg/day every 4 weeks. The response was very good and BCVA reached 1.2 in both eyes 2 months after the therapy. RPE undulation and significant choroidal thickening improved 2 months after the therapy (Figure 3a, 3b).

Discussion

SARS-CoV-2, which is a novel coronavirus that causes COVID-19, is associated with a multitude of adverse systemic effects beyond

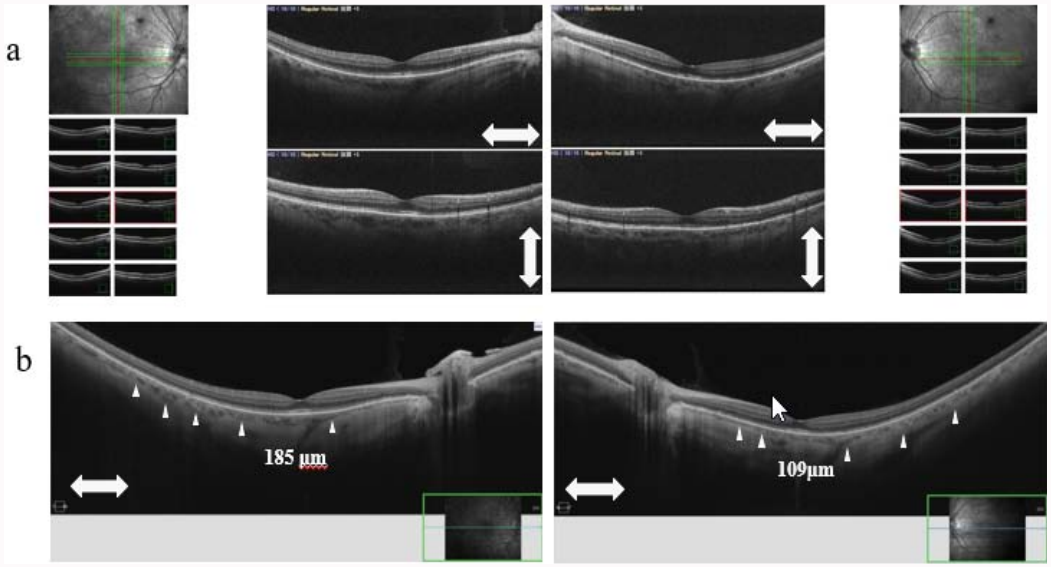

Figure 3: Optical coherence tomography images, 2 months after the therapy from a 57-year-old male patient with decreased vision in both eyes and a headache 1 week after COVID-19 infection, showing (a) serous retinal detachments and retinal pigment epithelium undulation vanished through steroid therapy in both eyes and (b) choroidal thickening improved to almost normal in both eyes.

acute respiratory syndrome. Molecular mimicry has been proposed as a contributing factor to some ocular complications, including conjunctival hyperemia [3], optic neuritis, uveitis [4], VKH disease [5-8], and ampiginous choroiditis [9]. The clinical manifestations of VKH disease are caused by an autoimmune response directed against melanin-associated antigens in SARS-CoV-2 may become a trigger for VKH disease, as have the target organs, i.e., the eye, inner ear, meninges, and skin. Sensibilization of melanocytes by virus antigens is considered one of the causes of VKH disease [10]. Flu-like symptoms antecedent to VKH disease would make sense as a virus infection. SARS-CoV-2 may become a trigger for VKH disease, as have several other viruses been considered triggers for the onset of VKH disease [10-13].

Bassili et al. reported that the Epstein-Barr virus was detected from a vitreous sample in a 24-year-old Korean woman with VKH disease [11]. The Epstein-Barr virus was not detected although they checked the vitreous sample in nine patients with uveitis and two patients with proliferative diabetic retinopathy [11]. Taken together, they concluded the strong association between VKH disease and the Epstein-Barr virus [11]. Their research is very important because it can be direct proof that the Epstein-Barr virus exists in a vitreous body in a patient with VKH disease.

Sugita et al. stated that T cells from the peripheral blood and intraocular fluid in patients with VKH disease cross-reacted with tyrosinase protein as well as with highly homologous cytomegalovirus-specific sequences [12].

Yoshino et al. reported a case of a 31-year-old Japanese male patient who simultaneously had a positive influenza A virus antigen test result and VKH disease [13]. He was treated with peramivir for influenza A at first and with steroid pulse therapy for VKH disease next [13]. They speculate that influenza A virus infection contributes to the onset or exacerbation of VKH disease [13].

Chronic Hepatitis C Virus (HCV) infection is estimated to infect 3% of the world population [14]. Additionally, HCV infection is implicated in the development of various autoimmune diseases [15]. Toitou et al. speculated a possible association between HCV infection and/or interferon treatment and the development of VKH disease [14]. Several case reports of VKH-like diseases after Interferon-Alpha (IFN-α) administration in patients with hepatitis C were presented [16,17]. Duan et al. reported that IFN-α treatment of hepatitis C can trigger VKH disease, and the cause of IFN-α-related VKH disease might be its T cell stimulating properties [17].

COVID-19 is an infectious disease caused by the new coronavirus, SARS-CoV-2. Galeotti et al. reported that COVID-19 infection can trigger several autoimmune and inflammatory disorders [18]. Previous reports support the role of viral infection as an immunological trigger for VKH disease [4-18].

Conclusion

Altogether, COVID-19 infection could be a possible trigger factor for VKH disease. However, the exact mechanism for the development of VKH disease remains unclear today. Therefore, the possibility of the development of VKH disease after COVID-19 infection by chance cannot be denied. The mechanism for the development of VKH disease should be elucidated urgently.

References

1. Sonoda KH, Hasegawa E, Namba K, Okada AA, Ohguro N, Goto H, et al. Epidemiology of uveitis in Japan: A 2016 retrospective nationwide survey. Jpn J Ophthalmol. 2021;65(2):184-90.
2. Urzua CA, Herbort CP Jr, Takeuchi M, Schlaen A, Concha-Del-Rio LE, Usui Y, et al. Vogt-Koyanagi-Harada disease: the step-by-step approach to a better understanding of clinicopathology, immunopathology, diagnosis, and management: A brief review. J Ophthalmic Inflamm Infect. 2022;12(1):17.
3. Wu P, Duan F, Luo C, Liu Q, Qu X, Liang L, et al. Characteristics of ocular findings of patients with Coronavirus disease 2019 (COVID-19) in Hubei province, China. JAMA Ophthalmol. 2020;138(5):575-8.
4. Benito-Pascual B, Gegúndez JA, Díaz-Valle D, Arriola-Villalobos P, Carreño E, Culebras E, et al. Panuveitis and optic neuritis as a possible initial presentation of the novel Coronavirus disease 2019 (COVID-19). Ocul Immunol Inflamm. 2020;28(6):922-5.

5. Yepez JB, Murati FA, Petitto M, De Yepez J, Galue JM, Revilla J, et al. Vogt-Koyanagi-Harada disease following COVID-19 infection. Case Rep Ophthalmol. 2021;12(3):804-8.

6. Santamaria A, Chang J, Savarain C. SARS-CoV-2 among the potential viral triggers for Vogt-Koyanagi-Harada disease: First case report and literature review. Ocul Immunol Inflamm. 2022;30(7-8):1869-75.

7. Saraceno JJF, Souza GM, Dos Santos Finamor LP, Nascimento HM, Belfort R Jr. Vogt-Koyanagi-Harada syndrome following COVID–19 and ChAdOx1 nCoV–19 (AZD1222) vaccine. Int J Retina Vitreous. 2021;7(1):49.

8. Anthony E, Rajamani A, Baskaran P, Rajendran A. Vogt-Koyanagi-Harada disease following a recent COVID–19 infection. Indian J Ophthalmol. 2022;70(2):670-72.

9. Tom ES, McKay KM, Saraf SS. Bilateral ampiginous choroiditis following presumed SARS-CoV-2 infection. Case Rep Ophthalmol Med. 2021;2021:1646364.

10. Lavezzo MM, Sakata VM, Morita C, Rodriguez EE, Abdallah SF, da Silva FT, et al. Vogt-Koyanagi-Harada disease: Review of a rare autoimmune disease targeting antigens of melanocytes. Orphanet J Rare Dis. 2016;11:29.

11. Bassili SS, Peyman GA, Gebhardt BM, Daun M, Ganiban GJ, Rifai A. Detection of Epstein-Barr virus DNA by polymerase chain reaction in the vitreous from a patient with Vogt-Koyanagi-Harada syndrome. Retina. 1996;16(2):160-61.

12. Sugita S, Takase H, Kawaguchi T, Taguchi C, Mochizuki M. Cross-reaction between tyrosinase peptides and cytomegalovirus antigen by T cells from patients with Vogt-Koyanagi-Harada disease. Int Ophthalmol. 2007;27(2-3):87-95.

13. Yoshino N, Kawamura A, Ishii A, Yoshida K, Watanabe T, Yamashita T, et al. Vogt-Koyanagi-Harada disease associated with influenza A virus infection. Intern Med. 2018;57(11):1661-5.

14. Touitou V, Bodaghi B, Cassoux N, Tran TH, Rao NA, Cacoub P, et al. Vogt-Koyanagi-Harada disease in patients with chronic hepatitis C. Am J Ophthalmol. 2005;140(5):949-52.

15. Jadali Z, Alavian SM. Autoimmune diseases co-existing with hepatitis C virus infection. Iran J Allergy Asthma Immunol. 2010;9(4):191-206.

16. Kasahara A, Hiraide A, Tomita N, Iwahashi H, Imagawa A, Ohguro N, et al. Vogt-Koyanagi-Harada disease occurring during interferon alpha therapy for chronic hepatitis C. J Gastroenterol. 2004;39(11):1106-9.

17. Duan J, Wang Y, Liu D, Ma J. Induction of Vogt-Koyanagi-Harada disease by interferon-alpha and ribavirin treatment in patients with hepatitis C: A case report and review of the literature. Ocul Immunol Inflamm. 2019;27(2):229-34.

18. Galeotti C, Bayry J. Autoimmune and inflammatory diseases following COVID-19. Nat Rev Rheumatol. 2020;16(8):413-4.