Vogt-Koyanagi-Harada syndrome following COVID-19 vaccination

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Uveitis fellow at Universidade Federal de São Paulo (UNIFESP)¹ Chief of the Uveitis sector of the Universidade Federal de São Paulo (UNIFESP)² Purpose: Illustrate a case of Vogt-Koyanagi-Harada syndrome as an adverse event following COVID-19 vaccination.

Methods: Ophthalmic evaluation and complementary exams were performed in the patient, and also follow-up consults in the Uveitis ambulatory.

Results: 39-year-old female presented to the Uveitis ambulatory reporting ocular pain and low visual acuity in both eyes in the past three months, which started 2 weeks following the first dose of COVID-19 Oxford-AstraZeneca vaccine. She presented headache and tinnitus, and denied a history of penetrating trauma or intraocular surgery. In the previous service, she had been treated with methylprednisolone pulse therapy for 3 days and was using regressive oral prednisone 20 mg daily. In the initial exam, visual acuity (VA) was light perception in both eyes. Biomicroscopy revealed posterior synechiae, and no anterior chamber reaction (ACR). Fundus exam revealed extensive serous retinal detachment (RD) and hyperemic disc in both eyes. Syphilis and HIV serologies were negative, and tuberculosis screening was also negative..

Due to the hypothesis of VKH syndrome, the patient was hospitalized and was treated with ivermectin followed by three-day pulse therapy with 1g intravenous methylprednisolone. One week following pulse therapy, the patient presented hand movement VA and maintained the fundoscopy aspect of the initial exam. Due to the unsatisfactory response to the methylprednisolone, pulse therapy with cyclophosphamide (1g/m² of body surface) for 3 days was prescribed, as well as 50 mg of oral prednisone daily. Following the cyclophosphamide pulse, the patient presented a discrete improvement in the extension of the serous RD and the superior retina was attached, and she also reported improvement of the headache. She also reported depigmentation in the neck.

In the following consults, there was a slow progressive improvement in the height of the serous RD, and adalimumab was introduced - 40 mg subcutaneous every 2 weeks - and the oral prednisolone was slowly regressed. Although the improvement in the fundus aspect, and the absence of signs of active inflammation (no RCA nor vitritis), the VA remained hand movement and there was also loss of external retina in the optical coherence tomography (OCT) of the macula.



Figure 1 - Patient presenting initial neck depigmentation.



Figures 2-5 - Fundus photographs revealing sunset glow fundus and atrophic aspect of the macula in both eyes, and a residual serous RD in the inferior periphery in the 4-month follow up.

Discussion: After over one year of follow-up, the patient remains without inflammatory activity using adalimumab and 5 mg oral prednisone. VKH syndrome is a granulomatous panuveitis associated with serous retinal detachment. Its physiopathology is multifactorial, involving an autoimmune T-cell mediated response against melanocytes' antigens, and genetic predisposition, such as the presence of HLA-DR4. Influenza vaccination and viral infections have been described as triggers to VKH syndrome.

The onset of VKH syndrome has been described as an adverse event following COVID-19 vaccination and most cases described occur within 1 week after vaccination. Although the mechanism is unclear, it is believed that the adenoviral vector of the Oxford-AstraZeneca vaccine can trigger antibody–mediated hypersensitivity reactions through antigen-mimicry, inducing a Th1 response in predisposed individuals, which could result in a VKH syndrome onset or its re-activation. The patient of the reported case had no prior history of ocular inflammation and developed symptoms 2 weeks following the first dose of the vaccine, and the inflammation resulted in permanent visual impairment despite treatment. It is important to study such cases of adverse events following vaccination in order to understand its pathogenesis and also identify predisposed individuals to ocular inflammation.

References:

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