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DOI: 10.4103/2211-5056.353129

Ocular inflammatory manifestations following COVID-19 vaccinations in Taiwan: A case series

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Abstract:

As a continuing demand for booster shots against SARS-CoV-2, ocular adverse events following the coronavirus disease-2019 (COVID-19) vaccines can cause significant visual impairment, and they warrant a high awareness and detailed documentation of possible ocular inflammatory manifestations. We present a case series of 11 patients presenting with ocular manifestations relevant to vaccine-associated autoimmune response within 6 weeks after the vaccination of the Oxford–AstraZeneca, the Moderna, and Pfizer–BioNTech vaccines at the main tertiary referral center in the most populated and most vaccinated city in Taiwan. Their diagnosis included five acute anterior uveitis, two multiple evanescent white dot syndrome, one probable Vogt–Koyanagi–Harada disease, one anterior scleritis, one relapsed idiopathic panuveitis, and one autoantibody-related central retinal artery occlusion. This report presented a broad spectrum of the ocular inflammatory events following the vaccination of COVID-19. Early recognition of the clinical manifestations mentioned herein with prompt management is crucial in recovering the patients' vision.

Keywords:

Choroid, intraocular inflammation, retina, uveitis, vascular occlusion

Introduction

The coronavirus disease-2019 (COVID-19) pandemic is an ongoing global threat to public health. Several vaccines against SARS-CoV-2 have been developed on various platforms. Due to the short observation period of these vaccines and the continuing demand for booster shots, monitoring the adverse events (AEs) after vaccinations is crucial.

As of February 2022, more than 19 million people in Taiwan (82.8% of the population) had been vaccinated for the first dose and 76.8% of the population had completed the second dose. Of the first dose, 41.6% received the Oxford–AstraZeneca COVID-19 vaccines (AZD1222, AZ),

21.0% received the Moderna COVID-19 vaccines (mRNA-1273, Moderna), and 32.9% received the Pfizer–BioNTech COVID-19 vaccines (BNT162b2). New Taipei City, which is the most populated city in Taiwan, is not only the epicenter of the pandemic (with 45.6% of domestic cases) but also one of the most vaccinated areas in Taiwan. As the main tertiary referral center in New Taipei City, our hospital is responsible for COVID-19-related critical care and the management of postvaccination AEs.

In this report, we present a case series of ocular inflammatory manifestations after the COVID-19 vaccination.

Methods

In this retrospective consecutive case series, patients presenting with ocular

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How to cite this article: Wang LU, Chen FT, Wang JK, Huang TL, Chang PY, Chen YJ, *et al.* Ocular inflammatory manifestations following COVID-19 vaccinations in Taiwan: A case series. Taiwan J Ophthalmol 2022;12:465-71.

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Submission: 15-03-2022
Accepted: 12-06-2022
Published: 01-08-2022

inflammation within 6 weeks of undergoing the first dose of COVID-19 vaccinations between May 2021 and October 2021 were enrolled. This study was approved by the Institutional Review Board of (Far Eastern Memorial Hospital-IRB No.: 110251-E) and was conducted following the tenets of the Declaration of Helsinki. The requirement for consent was waived by the institutional review board.

Ophthalmic examinations, namely a Snellen best-corrected visual acuity (BCVA), slit-lamp biomicroscopy, and indirect fundoscopic examination, were performed on each patient. Imaging examinations were performed at each physician's discretion. The image modalities comprised ultra-widefield fundus photography (Optos California, Optos PLC), spectral-domain optical coherence tomography (OCT, Cirrus 5000; Carl Zeiss Meditec Inc.), automated visual field (VF) test (Humphrey® Field Analyzer 3, Carl Zeiss Meditec Inc.), and angiography imaging (HRA Spectralis®, Heidelberg Engineering) for fluorescein angiography (FA), indocyanine green angiography (ICGA), and fundus autofluorescence (FAF).

A stepwise diagnostic approach was performed on every uveitis patient seen in our clinic. In general, the anatomical location was defined per SUN criteria.^[1] Meanwhile, the clinical dimensions were determined according to Jabs' scheme.^[2] The relevant systemic evaluation was heavily based on our regional epidemiological data in Taiwan.^[3,4] Instead of a "shotgun" approach, only targeted laboratory tests were performed according to the pretest likelihood.

Results

We enrolled 11 patients (4 men and 7 women) with a mean (\pm standard deviation [SD]) age of 47.7 (\pm 16.2) years [Table 1]. The mean (\pm SD) time to the onset of ocular symptoms was 14.5 (\pm 12.4) days after vaccination. The mean follow-up time (\pm SD) was 125.7 (\pm 89.4) days. The ocular inflammatory disorders diagnosed included acute anterior uveitis (five patients), multiple evanescent white dot syndrome (MEWDS) (two patients), probable Vogt-Koyanagi-Harada (VKH) disease (one patient), and anterior scleritis (one patient). A patient presented with a recurrent attack of bilateral idiopathic panuveitis. Another patient (Case 11) was diagnosed with unilateral central retinal arterial occlusion (CRAO). Treatment was described in Table 1. Most of the patients presented with the complaint of blurred vision and exhibited improved or recovered vision after the treatment. During subsequent follow-up, no patients presented with postvaccination ocular inflammation again. While case 11 was reluctant to receive the second dose, case 4 switched from Moderna mRNA-1273 to BNT162b2 uneventfully. Meanwhile, other patients received the

second shots with the same vaccine brands as their first ones without recurrent ocular inflammation.

Selected cases

Case 6: Multiple evanescent white dot syndrome

A 32-year-old healthy woman presented with central scotoma in her right eye 34 days after Oxford - AstraZeneca COVID-19 (AZ) vaccination. She had no flu-like prodromes and denied other physical complaints. The presenting BCVA in the right eye was 10/20. The fundus examination revealed grey-white lesions in the peripapillary area and posterior deep retina. These lesions exhibited hyperautofluorescence on the FAF and corresponded to interrupted ellipsoid zones (EZ) on the OCT [Figure 1]. Meanwhile, OCT also demonstrated some intraretinal hyperreflective lesions above the EZ line. The EZ was relatively preserved than that at the peripapillary region. Besides, there was no hyperautofluorescence change in the central foveal region; therefore, we speculated that these lesions were an acute accumulation of inflammatory depositions without significant retinal pigment epithelium dysfunction. The VF examination revealed an enlarged blind spot. A low-dose oral steroid of 10 mg prednisolone per day for 4 weeks was prescribed. The patient's BCVA recovered to 20/20 after 7 weeks.

Case 8: Vogt-Koyanagi-Harada

A 52-year-old woman complained of bilateral progressive blurred vision for 3 days with preceding vaccination at 13 days after AZ vaccination. She experienced mild dizziness but denied headache, tinnitus, or flu-like

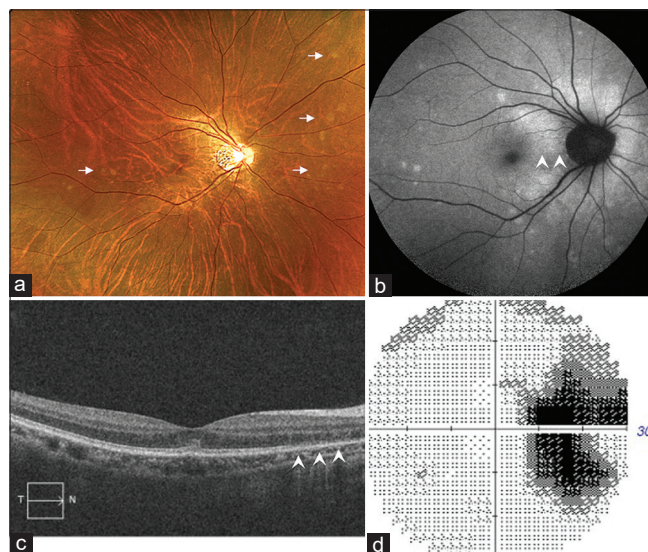


Figure 1: Clinical presentations: Case 6 of MEWDS in the right eye. Ultra-widefield fundus photography (a) showed multiple grey-white lesions at the mid-peripheral and peripapillary area (arrows). OCT (c) and FAF (b) showed an interrupted ellipsoid zone (arrowheads) at the area of corresponding peripapillary hyper-autofluorescence (arrowheads). OCT (c) also demonstrated intraretinal hyperreflective lesion above the EZ line in the central foveal region. VF (d) revealed an enlarged blind spot. MEWDS = Multiple evanescent white dot syndrome, OCT = Optical coherence tomography, FAF = Fundus autofluorescence

Table 1: Clinical features of ocular inflammatory manifestations

Case	Gender	Age	Vaccine	Time from vaccination to symptoms (days)	Affected eye	Diagnosis	Targeted systemic evaluation	Symptoms	Presenting BCVA	Final BCVA	Treatment	Outcome
1	Male	28	Moderna ^a	11	OD	Acute anterior uveitis	Positive HLA-B27	Pain, Blurred vision	20/50	20/28.6	Topical steroids	Resolved
2	Male	36	AZ ^b	5	OD	Acute anterior uveitis	HLA-B27 not tested	Red eye, Foreign body sensation	20/25	20/22.2	Topical steroids	Resolved
3	Female	56	AZ	13	OD	Acute anterior uveitis	Negative HLA-B27	Blurred vision	20/400	20/50	Oral steroids	Improved
4	Male	58	Moderna	30	OS	Acute anterior uveitis	Positive HLA-B27	Blurred vision	20/50	20/33.3	Topical steroids, oral steroids	Improved
5	Female	29	BNT162b2 ^c	4	OS	Acute anterior uveitis	Positive HLA-B27	Blurred vision	20/25	20/25	Topical steroids, oral steroids	Improved
6	Female	32	AZ	34	OD	MEWDS ^d	Not necessary	Central scotoma	20/40	20/20	Oral steroids	Resolved
7	Female	59	AZ	7	OS	MEWDS	Not necessary	Blurred vision	20/25	NA ^e	Topical steroids	NA
8	Female	52	AZ	13	OU	Probable VKH ^e	Unremarkable for occult infectious diseases, CSF study not performed	Blurred vision	OD 20/200 OS 20/100	OD 20/25 OS 20/20	Oral steroids, cyclosporin	Improved
9	Female	62	AZ	3	OU	Relapsing of idiopathic panuveitis	Sarcoidosis-like, but unremarkable ACE and chest CT	Pain, blurred vision	OD 20/50 OS 20/200	OD 20/40 OS 20/40	Topical steroids, oral steroids	Improved
10	Female	50	AZ	35	OS	Anterior scleritis	Negative ANA, RF, anti-ENA, and ANCA	Red-eye, pain	20/28.6	20/25	Topical steroids, oral steroids	Resolved
11	Male	70	Moderna	5	OD	CRAO	Positive anti-PF4, no contributing findings on the echocardiography and the carotid duplex study	Blurred vision	Counting fingers at 15 cm	Counting fingers at 15 cm	Hyperbaric oxygen therapy, topical anti-glaucoma drops	Stationary

^aModerna COVID19 vaccine (mRNA-1273), ^bOxford-AstraZeneca COVID-19 vaccine (AZD1222, ChAdOx1 nCoV-19, ChAdOx1-S), ^cPfizer-BioNTech COVID-19 vaccines (BNT162b2), ^dMultiple Evanescent White Dot Syndrome, ^eVogt-Koyanagi-Harada disease, CRAO, ^fThe patient did not return to our clinic. ACE=Angiotensin-converting enzyme, ANA=Antinuclear antibody, ANCA=Antineutrophil cytoplasmic antibodies, anti-ENA=Anti-extractable nuclear antigens, anti-PF4=Anti-platelet factor 4 antibodies, CRAO=Central retinal artery occlusion, CSF=Cerebrospinal fluid, CT=Computed tomography, HLA-B27=Human leukocyte antigen B27, MEWDS=Multiple evanescent white dot syndrome, NA=Not acquired, RF=Rheumatoid factor, VKH=Vogt-Koyanagi-Harada disease, BCVA=Best-corrected visual acuity, OD=Right eye, OS=Left eye, OU=Both eyes, AZ=Oxford - AstraZeneca COVID-19 vaccines

prodromes. No other neurological deficits were found. The presenting BCVA was 20/200 in the right eye and 20/100 in the left eye. The fundus examination showed subretinal fluid at the macula and inferior retina [Figure 2]. OCT presented bilateral fibrinous subretinal and intraretinal fluid at the macula with profound retinal pigment epithelial folds. The FA and ICGA revealed typical pinpoint leakage and choroidal dark spots on the mid phase of 3 min in both eyes. After a month of combination therapy with oral prednisolone with an initial dose of 1 mg/kg per day and cyclosporine A 200 mg per day, the subretinal fluid completely resolved. We gradually tapered oral prednisolone to 5 mg per day for 4 months and discontinued it after 4 months due to disease quiescence. We also substituted azathioprine 100 mg per day for cyclosporine A due to the development of hirsutism in 3 months. The patient's final vision recovered to 20/25 in the right eye and 20/20 in the left eye.

Case 11: Central retinal arterial occlusion

A 70-year-old man presented with sudden-onset painless visual loss in his right eye 5 days after Moderna vaccination. Physical examinations revealed previously unknown stage I hypertension with systolic blood pressure of 160 mmHg and diastolic blood pressure of 85 mmHg. The presenting BCVA in the right eye was counting fingers at 15 cm. The funduscopy revealed a typical diffuse whitened retina with a cherry-red spot [Figure 3]. The OCT indicated inner retinal hyper-reflectiveness and thickening. Due to the typical clinical presentation with compatible OCT findings, we did not arrange FA/ICGA for this patient. Besides, choroidal circulation was presumed to be preserved in the presence of cherry-red spots, which made posterior ciliary artery or ophthalmic artery occlusion less likely. The internist's evaluation revealed no additional cardiovascular or neurovascular abnormalities. The

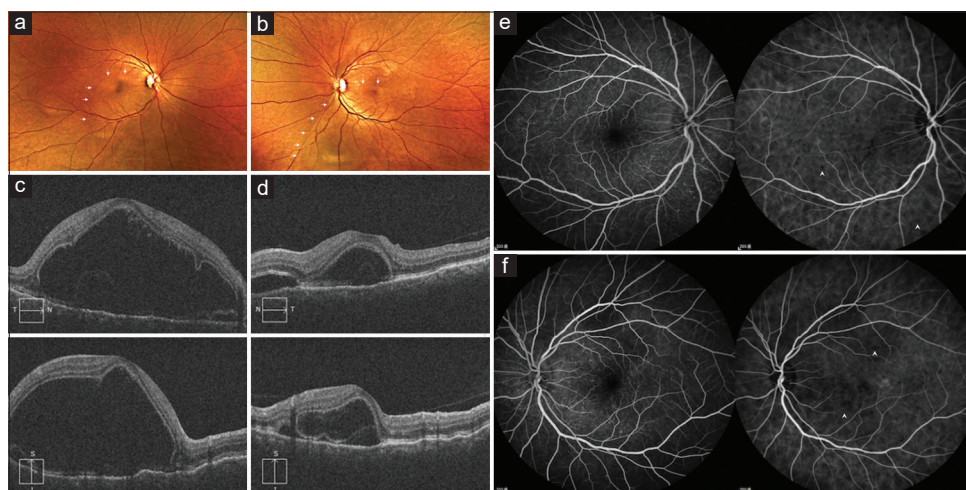


Figure 2: Clinical presentations: Case 8 of probable VKH. Color fundus photography (a: right eye, b: left eye) showed subretinal fluid at the macula and inferior retina (arrows). OCT (c: right eye, d: left eye) showed fibrinous intraretinal and subretinal fluid at the macula and profound retinal pigment epithelial folds. FA and ICGA (e: right eye; FA on the left and ICGA on the right, f: left eye; FA on the left and ICGA on the right) mid phase (3:07 in the right eye and 3:15 in the left eye) presented typical pinpoint leakage and choroidal dark spots (arrowheads). VKH = Vogt-Koyanagi-Harada, OCT = Optical coherence tomography, FA = Fluorescein angiography, ICGA = Indocyanine green angiography

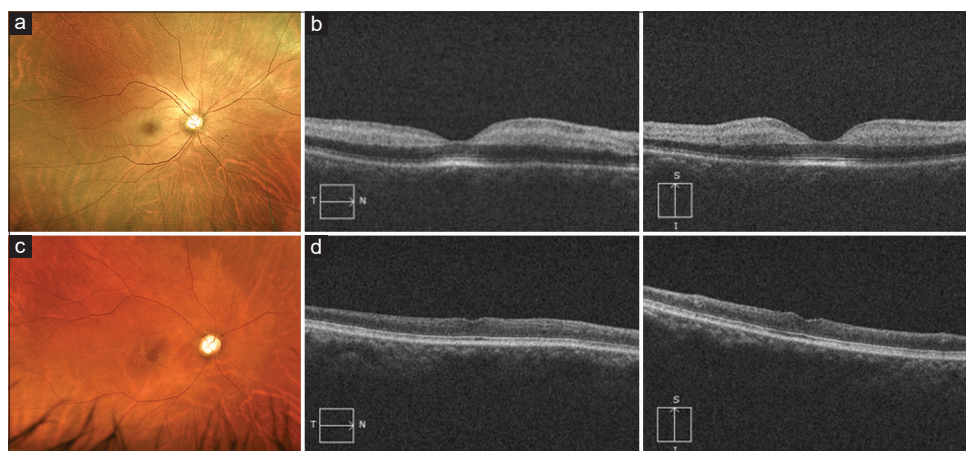


Figure 3: Clinical presentations: Case 11 of CRAO in the right eye. Presenting images and follow-up images after 7 months were shown. Presenting color fundus photography (a) showed a diffusely whitened retina with a typical cherry-red spot and OCT (b) showed inner retina hyper-reflectiveness. Following color fundus photography (c) and OCT (d) showed attenuated vessels and atrophic inner retina. CRAO = Central retinal arterial occlusion, OCT = Optical coherence tomography

laboratory data confirmed a normal level of cholesterol, triglyceride, low-density lipoprotein cholesterol, and HbA1c. The echocardiography was unremarkable for plaques or valvular abnormality. The carotid duplex study revealed only mild-to-moderate stenosis (<50%) at common and internal carotid arteries. Five weeks after the disease onset, the serum test revealed a positive anti-platelet factor 4 (anti-PF4) of 73.34 ng/mL (enzyme-linked immunosorbent assay method, upper limit: 49.99 ng/mL), with a normal platelet count of 170,000/ μ L, and a normal D-dimer level of 279 ng/mL fibrinogen equivalent units. Due to the poor visual prognosis of CRAO, following ineffective traditional management such as hypotensive topical medications and ocular massage, the patient agreed to undergo hyperbaric oxygen therapy (HBOT) for 20 sessions about 36 h after the disease onset. Although HBOT was commenced after 24 h, it was the only possible treatment modality to rescue the ischemic retinal tissue in this devastating condition.^[5] The patient experienced a widened VF after the therapy. The final BCVA after 7 months was counting fingers at 15 cm. Consequently, we presumed this case as an autoantibody-related CRAO and a possible presentation of thrombosis with thrombocytopenia syndrome (TTS).^[6]

Discussion

Our study investigated ocular inflammatory AEs following COVID-19 vaccinations. According to the Naranjo scale and the causality assessment of AEs following immunization, all of our cases were interpreted as “possible” adverse reactions to the vaccines.^[7,8] Based on the definition of TTS,^[9] we included patients who experienced ocular events within 6 weeks of vaccination. This relative longer interval allowed us a chance to observe the occurrence of delayed autoimmune response, as some of the uveitis developed in 6 weeks.^[10] Although a certain causality cannot be demonstrated by our study, this ocular inflammation cases series provides crucial clinical information.

Several studies have recently explored the occurrence of uveitis following different COVID-19 vaccines. Because vaccine peptide fragments and adjuvants are both possible triggering antigens, various immune mechanisms have been postulated. Rabinovitch *et al.* conducted a study involving 21 patients who developed acute anterior uveitis and two patients who developed MEWDS following the administration of an mRNA-based vaccine, BNT162b2. The authors suggested a mechanism of adjuvant-induced type I interferon secretion.^[11] VKH following COVID-19 vaccinations has also been reported.^[12-15] Papisavvas and Herbort presented a case of chronic VKH reactivation 6 weeks after administration of the second dose of BNT162b2; the

authors proposed that the altered RNA metabolism with overactive Toll-like receptors induced the reactivation.^[13] Our case 8, on the contrary, was a *de novo* acute VKH disease. It can be the result of molecular mimicry between vaccine peptide fragments and uveal self-peptides, as postulated by Koong *et al.*^[14,16] *De novo* VKH disease was also reported to develop after the vaccination of yellow fever,^[17] Bacillus Calmette–Guérin,^[18] hepatitis B virus,^[19] and influenza.^[20,21] However, the exact mechanism for the development of VKH after the vaccination remains unclear. In most cases, inflammation was well-controlled with oral corticosteroids with excellent visual outcomes within 6 months of treatment. Although not commonly confirmed by most studies, Sood *et al.* reported a patient with negative human leukocyte antigen (HLA)-DR4 developing VKH disease following hepatitis B virus vaccination in 3 days;^[19] on the contrary, Murtaza *et al.* reported a patient with positive HLA-DR4 developing VKH disease following influenza vaccination in 2 days.^[20] Specific HLA haplotypes may thus account for the individual susceptibility of the autoimmune activation. However, HLA typing was not performed on our patient as a regular examination, and we observed a similar clinical presentation and prompt treatment response to the immunosuppressive therapy as seen in other typical VKH patients. Furthermore, anterior scleritis was reported in several patients who had received an inactivated COVID-19 vaccine.^[22] Pichi *et al.* suggested that the antigen of the virus acted as an immune trigger.

Few reports have explored retinal vascular disorders following COVID-19 vaccinations. Bialasiewicz *et al.* reported a case of central retinal vein occlusion that occurred immediately after BNT162b2 vaccination, but they did not speculate about the underlying mechanisms.^[23] Girbardt *et al.* reported six cases of retinal vascular events shortly following COVID-19 vaccinations with a branched retinal arterial occlusion and a combined arterial and venous occlusion, however, they did not provide a possible mechanism for the causality.^[24] In our patient with CRAO after receiving Moderna, the strong positive result of the anti-PF4 test suggests an autoimmune occlusive vasculopathy. Anti-PF4 is the antibody that binds the complex of heparin and platelet factor 4.^[9] Although a similar complex can also occur following exposure to compounds other than heparin, such as polyvinyl phosphonate,^[25] it remains unclear how the complex formed after the exposure to the COVID-19 vaccines, with no history of heparin use and other identifiable causes.^[26] Since our patient had no recent medical history of exposure to heparin, the emergence of anti-PF4 is most likely triggered by COVID-19 vaccination. As TTS was rarely seen in ophthalmology patients and patients receiving an mRNA vaccine, the platelet and D-dimer levels were not examined immediately but

5 weeks after the visual symptoms; therefore, the normal values may not reflect the acute blood profile changes. Nonetheless, recent study revealed that more than 70% of vaccine-induced TTS patients remained positive for anti-PF4 at 100 days.^[27] Although the patient's clinical profiles did not meet the criteria for TTS,^[9] we presumed that a mild or focal coagulation activation could result in retinal vessel occlusions without a significant change in D-dimer level. The follow-up ultra-widefield fundus photography after 7 months showed attenuation of the arteries, presumably due to the natural course of CRAO. Although HBOT was performed to alleviate the retinal ischemia, it was not possible to reverse the retinal arterial occlusions *per se* meanwhile, since FA was not performed, we could not rule out angiographic retinal vasculitis in this patient. The antibody-mediated autoimmunity induced by the mRNA vaccine may contribute to localized vasculitis, like the assumption that antibodies against spike proteins can cross-react with proteins and antigens in the retinal arteries.^[10]

The temporal association between the vaccinations and ocular manifestations was reported subjectively by the patients. Therefore, an exact causal relationship could not be determined. Meanwhile, we did not routinely acquire vaccination information for the new uveitis patients during the same time frame. Therefore, some vaccination-induced ocular inflammation could have been neglected.

In conclusion, this case series presented a spectrum of ocular inflammatory manifestations following COVID-19 vaccination. Early recognition of the clinical presentations mentioned herein with prompt management is crucial in recovering the patients' vision. Due to the complex immune reactions induced by the vaccines, more studies are required to elucidate the exact mechanisms underlying the occurrence of these manifestations.

Financial support and sponsorship

Nil.

Conflicts of interest

The authors declare that there are no conflicts of interests of this article.

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