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Out of sight, but not out of mind: Zoster sine herpete case study and survey of Zoster Eye Disease Study (ZEDS) Group

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1. Introduction

Herpes zoster (HZ), resulting from localized, unilateral reactivation of latent varicella zoster virus (VZV) in the dorsal ganglia of sensory nerves, is characterized by the presence of a vesicular dermatomal rash, and can often be complicated by postherpetic neuralgia in patients with previous varicella infection.[1] The median age of HZ onset is 56 years old.[2] Herpes zoster ophthalmicus (HZO) is the viral reactivation occurring along the ophthalmic division of the trigeminal nerve.[2,3] HZO accounts for about 10–25 % of all cases of HZ.[4] HZO presents with the characteristic rash, but due to its reactivation site, also demonstrates various ocular manifestations, including neurotrophic, epithelial, and stromal keratitis, uveitis, glaucoma, and sectoral iris atrophy.[5] Furthermore, a rare complication of HZO is acute retinal necrosis (ARN), which typically manifests as an arterial vasculitis and prominent inflammatory reaction that can lead to devastating vision loss.[2,6,7] Due to the morbidity associated with zoster, the United States Centers for Disease Control and Prevention recommends that all adults over 50 years old receive the recombinant zoster vaccine, which is also approved by the United States Food and Drug Administration for immunosuppressed adults aged 18 years and older.[8]

Most cases of HZO can be diagnosed by the presence of the vesicular dermatomal rash in the distribution of the first division of the trigeminal nerve, which is often a predictor of ocular inflammation and viral keratitis. Many patients also present with a prodromal period consisting of fever, malaise, headache and eye pain prior to this skin eruption. However, some patients present with pain in a particular dermatomal distribution along with other signs and symptoms suggestive of zoster, but no rash, a condition known as zoster sine herpete (ZSH). First described by Schwab, ZSH can result in delayed diagnosis and treatment given the lack of the characteristic rash, potentially leading to increased rates of complications and poorer patient outcomes.[9] Rather than relying on clinical findings alone, confirmation of this diagnosis is done via tissue testing such as Polymerase Chain Reaction (PCR) analysis of intraocular fluids revealing VZV DNA or elevated anti-VZV antibody titers.[2,10] Once identified, ocular ZSH is treated like typical HZO, which usually involves either oral or intravenous antiviral therapy combined with topical steroids and glaucoma medications, if the patient's intraocular pressure (IOP) is elevated.[11]

We present a case of PCR-confirmed ZSH that demonstrated unilateral retinal phlebitis without the common manifestations of HZO mentioned previously. The discovery of this unexpected posterior segment complication prompted a literature review and survey of clinicians in the Zoster Eye Disease Study (ZEDS) group to further characterize the diagnosis and management of ZSH, as well as the incidence of retinal phlebitis in ZSH cases.

2. Methods

This study complied with the Declaration of Helsinki, the Health Insurance Portability and Accountability Act (HIPAA) as well as the federal regulations of the United States. The protocol was approved by an institutional review board (Dartmouth-Hitchcock IRB, Protocol Number: STUDY02001101).

2.1. Case presentation

A chart review was performed for a 54-year-old female patient who presented with ocular pain, ocular hypertension and blurred vision in her right eye. She was initially diagnosed with acute unilateral hypertensive anterior uveitis. Quantitative polymerase chain reaction (PCR) eventually led to the diagnosis of zoster sine herpete after identification

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of retinal phlebitis on fluorescein angiography.

2.2. Literature review

A literature review was conducted on the OVID Medline, Scopus, and PubMed databases. The search term 'zoster sine herpete' was individually combined with 'phlebitis', 'arteritis', 'vasculitis', and 'acute retinal necrosis'. The number of search results were counted for each search, and relevant results were then reviewed in-depth.

2.3. Survey

Our voluntary survey was sent out via Google Forms to all the ophthalmologists in the Zoster Eye Disease Study Group (ZEDS) (n = 76). The ZEDS Group is an international multicenter collaboration amongst cornea specialists interested in evaluating treatment options in herpes zoster ophthalmicus. Throughout our study, an emphasis was placed on maintaining patient anonymity. The 14-question survey (Table 1) asked participants whether they have ever diagnosed ZSH and, if so, which diagnostic methods, clinical findings, and treatment plans were involved.

3. Results

3.1. Case presentation

A 54-year-old white female initially presented to the ophthalmology service with head and neck pain, conjunctival injection, and decreased vision in her right eye, but without symptoms of cranial nerve involvement. She first noticed conjunctival injection three days prior to presentation. She had no significant ocular history other than prior LASIK surgery in both eyes. Her medical history was notable for a tuberculosis (TB) exposure during her teenage years. In the same week as she had the onset of ocular symptoms, she had a positive screening QuantiFERON TB gold test but a negative chest CT. She was diagnosed with latent TB infection (LTBI) by an infectious disease specialist and began treatment with rifampin.

Her best-corrected visual acuity was 20/60–2 of the right eye and 20/20 of the left eye with IOPs of 30 mmHg and 15 mmHg in the right and left eye, respectively. On slit lamp examination there was 2+ conjunctival injection, 1+ cell in the anterior chamber, and fine keratitic precipitates in Arlt's triangle on the cornea. On examination of the fundus, cell in the anterior vitreous was noted. Notably, her examination was negative for a vesicular dermatomal rash or any corneal inflammation, neovascularization or epithelial defects. She was initially diagnosed with unilateral acute non-granulomatous hypertensive anterior uveitis, which was possibly an extrapulmonary manifestation of TB. She was prescribed prednisolone acetate 1 % to use every 2 hours and cyclopentolate 1 % once daily as a mydriatic for photophobia in her right eye. After one week without improvement, she was prescribed difluprednate 0.05 % every 2 hours and 2 % dorzolamide hydrochloride/0.5 % timolol maleate twice daily in her right eye for increased IOP. Given her concurrent treatment for TB, a known cause of uveitis and uveitic glaucoma, the patient was prescribed isoniazid, rifampin and pyrazinamide therapy for six months.[12] Extensive laboratory workup was significant for a positive HLA-B27 as well as negative syphilis and angiotensin-converting enzyme markers. She continued to have waxing and waning anterior chamber inflammation despite titration of topical and oral corticosteroid regimens.

Following initiation of treatment, iris transillumination defects were noted in the right eye. The presence of iris atrophy raised suspicion for iris vasculitis and concomitant retinal vasculitis, prompting evaluation with fluorescein angiography, revealing right eye unilateral retinal phlebitis (Fig. 1a). As a result of this, anterior chamber paracentesis was performed and sent for VZV, HSV1, HSV2, and CMV analysis via quantitative PCR. The PCR revealed only varicella zoster virus (VZV) DNA (54,000 copies/mL), thereby confirming the diagnosis of zoster sine herpete with associated retinal phlebitis. The patient began treatment with 1000mg of oral valacyclovir three times daily. Two weeks later, the patient's retinal phlebitis was dramatically reduced as demonstrated by repeat angiography (Fig. 1b). After her difluprednate

Table 1
ZEDS investigators survey questions and responses.

Topic	Questions	Answer Options (Responses)
Diagnosis	Have you ever diagnosed Zoster Sine Herpete?	**Yes** (11/28; 39 %), **No** (17/28; 60 %)
	How many total cases of Zoster Sine Herpete have you diagnosed?	**Less than 5 cases** (9/11; 81 %), **5–9 cases** (2/11; 19 %), **10–19 cases** (0), **Greater than 20 cases** (0)
	Over what period of time have you made these diagnoses of Zoster Sine Herpete?	**1–10 years** (5/11; 45 %), **11–20 years** (2/11; 18 %), **21–30 years** (4/11; 36 %)
	Which method was used to make the diagnosis of Zoster Sine Herpete?	**Resolution of symptoms following treatment** (5/10; 50 %), **Physical exam findings** (7/10; 70 %), **Varicella Zoster Virus DNA via PCR** (5/10; 50 %), **Serologic evidence of active zoster, e.g., Elevated anti-VZV antibody titers which regress with treatment** (3/10; 30 %)
Clinical findings	Which findings were seen in association with Zoster Sine Herpete?	**Keratitis** (9/11; 81 %), **Neurotrophic cornea** (6/11; 54 %), **Uveitis** (9/11; 81 %), **Glaucoma** (4/11; 36 %), **Iris atrophy** (6/11; 54 %), **Posterior segment complication** (1/11; 9 %), **Other** (0)
	In the setting of Zoster Sine Herpete, have you ever obtained fluorescein angiography?	**Yes** (3/11; 27 %), **No** (8/11; 72 %)
	Based on fluorescein angiography (if obtained), have you diagnosed retinal phlebitis in the setting of Zoster Sine Herpete?	**Yes** (2/8; 25 %), **No** (6/8; 75 %)
Treatment & Clinical course	For previous cases of Zoster Sine Herpete, which treatments were required?	**Oral** (11/11; 100 %), **Intravenous** (0), **Intravitreal** (0), **Topical steroids** (9/11; 81 %), **Immunomodulatory therapy** (1/11; 9 %)
	For previous cases of Zoster Sine Herpete, which oral treatments were required?	**Acyclovir** (4/10), **Famciclovir** (2/10), **Valacyclovir** (9/10), **Valganciclovir** (1/10), **Brivudin** (0)
	For previous cases of Zoster Sine Herpete, which intravenous treatments were required?	**Acyclovir** (0) **Foscarnet** (0), **Ganciclovir** (0), **Valacyclovir** (0), **Cidofovir** (0)
	For previous cases of Zoster Sine Herpete, which intravitreal treatments were required?	**Foscarnet** (0), **Ganciclovir** (0)
	In the setting of Zoster Sine Herpete, were you able to discontinue antiviral treatment?	**Yes** (8/11; 72 %), **No** (3/11; 27 %)
	In the setting of Zoster Sine Herpete, have you altered treatment if there were posterior segment complications? If yes, please explain. (Free text)	N/A; Not applicable; N/A; Longer duration of treatment, No posterior segment complications
	If there is any other information you would like to share regarding your case (s), please feel free to share below. (Free text)	Very rare, but real entity

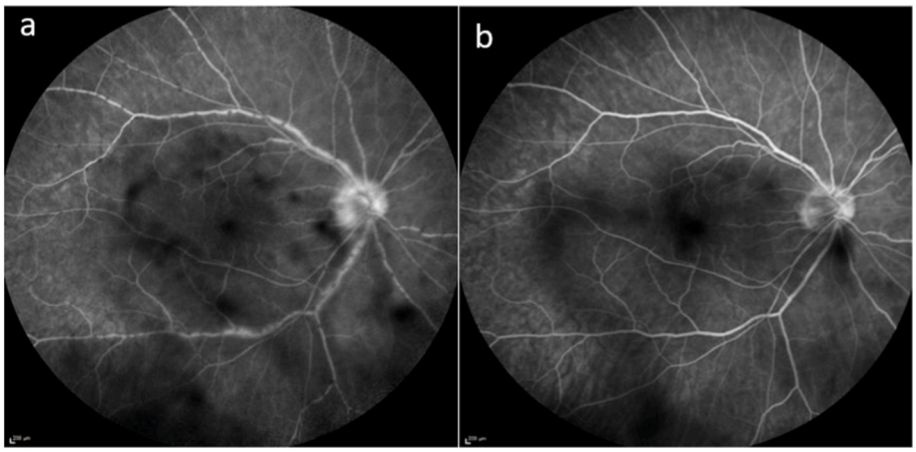

Fig. 1. Fluorescein angiography of the right eye. (a) Retinal phlebitis and optic nerve inflammation three months after initial presentation and before starting valacyclovir. (b) Phlebitis and inflammation dramatically improved one week after starting valacyclovir.

dose and valacyclovir dose was decreased to 1000mg daily, the patient experienced a flare of right eye uveitis with recurrence of anterior chamber inflammation and elevated IOP. Topical steroids and 1000mg of valacyclovir three times daily were restarted, and her uveitis resolved. Twenty-three months after beginning oral antiviral therapy, the patient's right eye visual acuity has improved to 20/30 + 2 without recurrent phlebitis and uveitis, postherpetic neuralgia, and with well controlled IOP at 14 mmHg. However, she remains on oral valacyclovir

Table 2
Review of literature: Retinal phlebitis as zoster sine herpete.

	Presented Case	Case 1[13]	Case 2[13]	Case 3[14]	Case 4[14]	Case 5[14]	Case 6[15]
Age	54 years	20 years	33 years	29 years	37 years	67 years	11 years
Gender	Female	Female	Male	Female	Female	Female	Male
Visual acuity	20/60–2 OD	20/200 OS	20/25 OS	20/60 OD	20/60 OU	CF OD 20/50 OS	20/20 OD
Intraocular pressure (mmHg)	30 OD 15 OS	15 OD 33 OS	15 OD 21 OS	N/A	N/A	N/A	N/A
Other clinical findings	Conjunctival redness, cell in anterior chamber	Ciliary vasodilation, corneal edema, corneal precipitates, cell in anterior chamber, and vitreous opacity	Conjunctival redness, ciliary vasodilation, corneal edema, corneal precipitates, cell in anterior chamber	Unilateral vitritis, retinal ischemia, CME, papillitis	Papillitis, macular edema	Bilateral diffuse posterior pole edema, papillitis, optic neuropathy	NVD, NVE, severe vitritis, keratitis with KPs, anterior uveitis, PSC
V1 pain	Present	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned
ZSH diagnosis method	PCR screening of aqueous sample	Serological screening (VZV IgM and IgG) and PCR screening of aqueous sample	Serological screening (VZV IgM and IgG) and PCR screening of aqueous sample	PCR screening of aqueous sample	PCR screening of aqueous sample	PCR screening of aqueous sample	PCR screening of vitreous sample
Periphlebitis diagnosis method	Fluorescein angiography	Fundus exam and fluorescein angiography	Fundus exam and fluorescein angiography	Fundus exam	Fluorescein angiography	Fluorescein angiography	Fluorescein angiography
Testing results	PCR: positive for VZV DNA	IgM: 84 (normal, negative) IgG: 2816 (normal) PCR: positive for VZV DNA	IgM: not detected IgG: 704 (normal) PCR: positive for VZV DNA	PCR: positive for VZV DNA	PCR: positive for VZV DNA	PCR: positive for VZV DNA	PCR: positive for VZV DNA
Treatment	Oral valacyclovir, difluprednate, dorzolamide/timolol, and topical prednisolone acetate	Oral acyclovir and acetazolamide, topical steroids and mydriatics, oral prednisolone	Oral acyclovir and prednisolone	Oral valacyclovir and prednisone	Oral valacyclovir and prednisone	Oral valacyclovir, oral steroids, and cyclosporine A	Oral acyclovir, topical steroids, and topical NSAIDs
Iris atrophy	Presented 3 months after initial onset	Presented 2 months after initial onset	Presented 3 weeks after initial onset	Not mentioned	Not mentioned	Not mentioned	Presented 12 months after initial onset
Outcome	Resolution of inflammation and periphlebitis, but unable to discontinue valacyclovir or topical steroids Visual acuity of 20/25–2 OD	Resolution of phlebitis and vitreous opacities Visual acuity of 20/20 OS	Resolution of inflammation and periphlebitis	Resolution of inflammation with continued steroid and antiviral use at 12 months follow-up Visual acuity of 20/40 OD	Resolution of inflammation with continued steroid and antiviral use at 6 months follow-up	Resolution of inflammation with continued steroid and antiviral use at 15 months follow-up	Infrequent recurrences of anterior uveitis, controlled with antiviral courses Visual acuity of 20/200 OD

1000 mg twice daily, 2 % dorzolamide hydrochloride/0.5 % timolol maleate ophthalmic solution twice daily, and prednisolone acetate 1 % once daily in the right eye. Her therapeutic response to this treatment regimen and ongoing need for antiviral therapy with valacyclovir further supports the diagnosis of ZSH as the cause of her uveitis.

3.2. Literature review

After reviewing the OVID Medline, Scopus, and PubMed databases, the Scopus database yielded the most results of ZSH associated with phlebitis, arteritis, vasculitis, or acute retinal necrosis. A broad search algorithm was utilized to ensure that no relevant articles were inadvertently excluded. However, many of the articles found with the keywords 'phlebitis', 'arteritis', and 'vasculitis' did not refer to retinal vasculitis, but rather inflammation in the central nervous system or elsewhere. The articles referring to non-ocular inflammation were excluded. The combination of keywords 'zoster sine herpete' and 'phlebitis' yielded only a single search result; this article presented two cases of zoster sine herpete with retinal periphlebitis.[13] A summary of the two cases and our featured case is included (Table 2). Further web searches yielded two additional articles that presented four additional cases of retinal vasculitis in the setting of VZV infection and are also included in Table 2.[15]

3.3. Survey

Of the 76 ophthalmologists in the ZEDS group, 28 (37 %) submitted responses to the survey.

Diagnosis of ZSH. The results of this survey further emphasize the rare incidence of ZSH, even amongst cornea and uveitis specialists. Of the 28 respondents, only about a third (11/28, 39.3 %) had ever diagnosed ZSH, and the majority of these physicians (9/11, 81.8 %) had diagnosed fewer than 5 cases of ZSH in their careers. Two had diagnosed 5–9 cases, with no respondents reporting more than 9 cases of diagnosed ZSH. Five physicians had diagnosed ZSH over the course of 1–10 years, two over 10–20 years, and four over 21–30 years.

Physical exam findings were the most reported method of diagnosis (70 %), but clinical efficacy based on empiric antiviral treatment (50 %), VZV PCR screening (50 %), and serologic evidence (30 %) were also reported methods.

Clinical presentation of ZSH. Keratitis and undifferentiated uveitis were the most commonly reported findings (81.8 %). Neurotrophic corneas (54.5 %), iris atrophy (54.5 %), and glaucoma (36.4 %) were also reported. Only one respondent (9.1 %) noted posterior segment complications. Of the 11 respondents who had diagnosed ZSH, three had obtained fluorescein angiography, and only two respondents (18.2 %) had diagnosed retinal phlebitis in the setting of zoster sine herpete. Of note, we determined that these two respondents were alluding to the same patient.

Treatment of ZSH. All 11 respondents reported managing ZSH through use of oral antivirals. Nine (81.8 %) also used topical steroids and 1 (9.1 %) used immunomodulatory therapy, but not steroids. No use of either intravenous or intravitreal antiviral medications was reported. Valacyclovir was the most used oral antiviral (90 %), followed by acyclovir (40 %), famciclovir (20 %), and valganciclovir (10 %). A quarter of respondents (3/11, 27.3 %) reported that they were unable to discontinue antiviral treatment.

4. Discussion

Herpes zoster ophthalmicus is a manifestation of the herpes zoster virus that can be diagnosed by the presence of the characteristic dermatomal rash, keratitis, anterior uveitis and decreased corneal sensation.[4,5,9,16] However, ocular ZSH presents a unique challenge to ophthalmologists as diagnosis relies on clinical findings that may be less specific, such as acute V1 dermatomal pain, features of the uveitis, response to treatment, and ultimately laboratory testing, thereby potentially delaying proper diagnosis and treatment.[17] Furthermore, acute anterior uveitis is suggestive of multiple etiologies, further hindering the path towards diagnosis. Uveitic glaucoma is suggestive of viral uveitis, but not specific. For instance, Shahidatul-Adha and colleagues noted that uveitic glaucoma was the most common ocular complication of ocular TB occurring in 29.4 % of patients in their series of 34 patients treated at Hospital Universiti Sains Malaysia.[12] In a study of 1099 eyes with uveitis conducted in Japan, while 30 % of cases with active herpetic anterior uveitis had accompanying elevation of IOP, 20 % of patients with active HLA-B27 anterior uveitis also had increased IOP with one case related to peripheral anterior synechia and no cases secondary to steroid use.[18] Amongst ophthalmologists involved in the Zoster Eye Disease Study, many have very limited experience with ZSH as only a minority have ever diagnosed this condition and none have diagnosed more than nine cases during their careers.[19]

Despite the lack of formal guidelines on ZSH, diagnosis and treatment methods were similar across respondents. Most survey respondents based their diagnosis on clinical findings confirmed by tissue testing (either by PCR screening or serologic evidence). Similar clinical manifestations were reported, including keratitis, uveitis, uveitic glaucoma, neurotrophic corneas, iris atrophy, and postherpetic neuralgia. As with HZO, oral antivirals were the treatment of choice across all respondents and most (72.7 %) were able to discontinue antiviral treatment. However, a significant minority (27.3 %) of respondents reported that some of their cases required ongoing antiviral therapy. Given the variability in disease amongst such cases, there remains no standard guideline for systemic antiviral therapy. Instead, antiviral therapy is chosen on a case-by-case basis.

The low reported rate of posterior segment complications may be because the respondents consider ARN to be a distinct entity and do not classify it as ZSH. Furthermore, the ZEDS group is composed of cornea, rather than retina, specialists, who may be more focused on anterior segment complications of ZSH. Indeed, no respondents outside of our institution reported having diagnosed retinal phlebitis in the setting of ZSH. This data correlates with prior literature, which found only six prior cases of retinal vasculitis in the setting of confirmed VZV infection.[13–15] These cases shared many similarities with our presented case, including clinical presentation, namely decreased visual acuity, increased IOP, iris atrophy, and intraocular inflammation. Prior cases also relied on PCR analysis of aqueous humor as the primary method of diagnosis, with serology proving to be more unreliable. Prolonged treatment with oral antivirals, primarily acyclovir or valacyclovir, was also similar between cases, with follow-up period ranging between six and twenty-three months.

These findings related to the diagnosis, treatment, and complications of ZSH provide useful insight into a rare ophthalmic condition. However, we understand the limitations of a survey study. All respondents were cornea specialists in the Zoster Eye Disease Study, whose experiences with ZSH may not be representative of all ophthalmologists. Furthermore, only 11 respondents had experience with ZSH, constituting a relatively small and limited sample size. With respect to posterior segment complications, most respondents who had treated ZSH did not report obtained additional imaging such as fluorescein angiography, thus lowering their ability to diagnose posterior segment complications of ZSH. Finally, with all surveys, there is also the risk of sampling bias associated with non-respondents and recall bias, all of which could be avoided with a prospective study of ZSH cases.

In summary, ocular ZSH is a rare condition that is challenging to diagnose, and therefore a high degree of clinical suspicion is required. Unlike many uveitic entities, which respond well to corticosteroids regardless of their etiology, patients with ZSH may not stabilize until antiviral medication is given. Furthermore, as our case and survey suggest, prolonged antiviral treatment may be necessary. Clinicians caring for uveitis patients should consider ZSH particularly in patients with decreased corneal sensation, uveitis, iris atrophy, and uveitic

glaucoma despite the absence of a characteristic zoster rash and corneal inflammation. Posterior segment complications, while rare, may occur and retinal angiography may be useful in the management of such patients.

Patient Consent

Written consent to publish this case has not been obtained. This report does not contain any personal identifying information.

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Authorship

All authors attest that they meet the current ICJME criteria for Authorship.

Declaration of competing interest

The authors have no conflict of interest.

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