CASE REPORT

A Proposed Variant of Visual Snow Syndrome

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ABSTRACT

The visual snow syndrome (VSS) is a relatively new clinical entity for the neuro-optometrist, and others (e.g., neurologist), with its wide array of visual (e.g., palinopsia) and non-visual (e.g., tinnitus) symptoms, especially the primary perception of visual snow (VS).

In this case series, we present two unusual patients with a new tentative variant of VSS, neuro-optometric diagnosis: VSS without VS. These two novel cases are discussed with regard to their visual symptoms and related clinical visual signs, as well as possible neuro-optometric therapeutic interventions (e.g., chromatic tints).

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INTRODUCTION

The neuro-perceptual phenomena of "visual snow" (VS) and its more global visual snow syndrome (VSS) have been of continuing interest to both the medical and optometric communities over the past 30 years.[1] However, their etiologies remain an enigma, with no clear and irrefutable underlying neurological or physiological mechanism(s) identified to date.[1] This is due in part to the wide array of visual and non-visual symptoms reported, as well as the complexity of the visual pathways.[2] Furthermore, visual snow can appear in isolation: VS alone is reported to be present in 3.7% of the UK population, whereas it is reported to be present in 2.2% of this population for the full syndrome.[3]

To be diagnosed with VSS, one must have: visual snow as the primary visual symptom for at least 3 months; then 2 or more of the following secondary visual symptoms, namely photosensitivity, enhanced entoptic imagery, difficulty with night vision ("nyctalopia"), and palinopsia. In addition, for the syndrome, patients typically report one or more of the following visual and non-visual symptoms: photopsia, phonophobia, tinnitus, balance problems, hyperacusis, cutaneous allodynia, tremor, and migraine.[1,4]

In the present paper, we present clinical data and speculate that there might be an additional, new, and rare variety: VSS without VS. Two cases will be presented that have been carefully examined by us clinically over the past year with this presumptive diagnosis.

METHODS

This was a retrospective, records-based study of all patients (n=61) reporting visual symptoms common to VSS over the past 2.5 years in the first author's practice. All patients with a history of VS/VSS over the past 2.5 years were reviewed (n=61). This yielded 2 patients (~3 %). They were examined by the first author in his private neuro-optometric

clinical practice in the past year. Ages were 31 and 37 yrs. There was one male and one female. Each received a comprehensive vision examination that included 3 areas: refractive, binocular, and ocular health status. The ocular health examination included external examination of the eye and adnexa including pupillary examination, slit lamp examination, and dilated fundus examination. In both cases the ocular health examination was negative. Corrected monocular and binocular visual acuities at both distance and near were 20/20. Patients had no history of ocular or neurological disease. Only one had a history of multiple concussions. Each completed the Visual Snow Syndrome Symptom Survey (VSSSS) questionnaire[4] based on previously established criteria. See Table 1.

Table 1: VSS questionnaire findings in the 2 patients

Patient	Case One	Case Two
Age (years)	31	37
Date of Examination	7/23/2024	8/27/2024
Number of Concussions	3	0
Medications	Topamax	Nexium
Systemic Health	Anxiety, migraines	Stress, anxiety, panic attacks, ocular migraines
Visual Snow	***no***	***no***
Palinopsia	**yes**	**yes**
Photosensitivity	**yes**	**yes**
Entoptic Phenomenon	no	no
Nyctalopia	no	**yes**
Photopsia	**yes**	no
Tinnitus	**yes**	**yes**
Balance Problems	**yes**	**yes**
Tremor	**yes**	**yes**
Phonophobia	no	no
Cutaneous Allodynia	no	no
Hyperacusis	no	no

Case 1

This was a 31-year-old woman who underwent a neuro-optometric evaluation in July 2024. She reported severe migraines which were diagnosed by her neurologist, photosensitivity, eye fatigue, and poor distance perception/judgement especially when driving. She had strabismus surgery for constant esotropia in 2003. After a boating incident in 2023, where a storm caused her to be "thrown around" in the boat, she immediately noticed a "trail of light" when shifting gaze, and a halo-like appearance around objects. We questioned her extensively regarding this symptom, and came to the conclusion that she had palinopsia. This has persisted to the time of the evaluation. She denied seeing any visual static, visual snow, or fuzzy appearance in her vision. The brain MRI after this incident was negative, and her neurologist believed that she may have suffered a concussion, although she did not hit her head. Additionally, she had a previous history of two other medically diagnosed concussions. She did not have any history of hallucinogen usage, long COVID symptoms, or post anesthesia complications. Additionally, there was no history of dysautonomia (including postural orthostatic tachycardia syndrome) or Ehlers Danlos syndrome, or any other connective tissue disorder.

We diagnosed intermittent alternating esotropia, oculomotor dysfunction, and "visual snow syndrome without snow". Recommendations included spectacles with an FL 41 chromatic tint to alleviate the photosensitivity and a vision therapy program focused on improving eye coordination, eye tracking, and accommodative abilities. To date, she has not commenced with the vision therapy program. Spectacles with a 25% FL 41 tint were prescribed. Unfortunately, she was lost to follow-up, so we were unable to assess the long effect of the tint in reducing symptoms. Table 2 summarizes the neuro-optometric evaluation in our office.

Case Two

This was a 37-year-old man who underwent a neuro-optometric evaluation in 2024. He had a longstanding history of strabismus with multiple corrective eye surgeries in childhood. He currently experiences intermittent diplopia, extreme dizziness, nausea, eye strain, and inadequate distance perception/judgement.

Table 2: Case 1, Neuro-Optometric Evaluation

Test	Result	Normal Range	Interpretation
Best Corrected Visual Acuity	RE/LE: 20/20	20/20	Normal
Refractive Status	RE -0.25- 0.25 x 120 LE: Plano	N/A	RE: Slight Compound Myopic Astigmatism LE: Emmetropic
Distance Phoria	12 pd IAET	0-2 exophoria	Intermittent Alternating Esotropia
Near Phoria	12-14 pd IAET	0-6 exophoria	Intermittent Alternating Esotropia
Nearpoint of Convergence	8 inches	1-3 inches	Fusional Instability
Near Convergence Range	18 pd	18-24 pd	Normal
Near Convergence Recovery	16 pd	7-15pd	Normal
Near Divergence Range	1 pd	16-24pd	Basic Esotropia
Near Divergence Recovery	0 pd	10-16pd	Basic Esotropia
Distance Convergence Range	18 pd	14-24pd	Normal
Distance Convergence Recovery	14 pd	6-12pd	Normal
Distance Divergence Range	1 pd	5-10pd	Basic Esotropia
Distance Divergence Recovery	0 pd	2-6pd	Basic Esotropia
Stereopsis (global)	Less than 500″	250″	Intact, but Reduced
Stereopsis (local)	70″	20″	Reduced Stereopsis
Pursuit Eye Movements	+2	+3 or greater	Oculomotor Dysfunction
Saccadic Eye Movements	+2	+3 or greater	Oculomotor Dysfunction
Developmental Eye Movement Test (DEM)	Speed: 36th %ile Errors: none (77th %ile)	38th %ile or better 38th %ile or better	Borderline Normal
Simultaneous Visual Memory: Tachistoscope	87th %ile	38th %ile or better	Normal
Sequential Visual Memory	69th %ile	38th %ile or better	Normal

pd=prism diopters cpm=cycles per minute RE=right eye LE=left eye IAET= intermittent alternating esotropia
Highlighted areas indicate abnormality

In March 2024, his visual symptoms worsened when he lost his habitual spectacles. Despite receiving multiple new prescriptions from other eye doctors since then, he has not fully adapted to any of them. He also reported mild ocular migraines, visual motion sensitivity, trailing of images when visually tracking, photosensitivity, tinnitus, balance problems, and intermittent tremors, as well as brain fog, although he denies seeing visual static or visual snow. We questioned him extensively regarding the trailing images he reported. He said that he saw multiple images when reading especially on screens. He also reported persistent afterimages at times, also more prevalent when looking at computer screens. These symptoms helped clarify the diagnosis of palinopsia. The diagnosis of ocular migraines was confirmed by his neurologist in April 2024. A recent brain MRI revealed a benign pineal cyst and cervical spine arthritis, with the former possibly contributing to some of his symptoms. He did not have any history of hallucinogen usage, long COVID symptoms, or post anesthesia complications. Additionally, there was no history of dysautonomia (including postural orthostatic tachycardia syndrome), Ehlers Danlos syndrome, or any other connective tissue disorder.

Our evaluation revealed intermittent, alternating, V-pattern esotropia with dissociated vertical deviation, inferior oblique overaction, and visual suppression. These findings are consistent with infantile esotropia and contribute to his inadequate distance perception, eye coordination difficulties, and diplopia. Additionally, he exhibited signs of "visual snow syndrome without snow", with symptoms including light sensitivity, palinopsia without trailing, night vision problems, and tinnitus. He also had an oculomotor dysfunction affecting his ability to track and scan visual stimuli, leading to

Table 3: Case 2, Neuro-Optometric Evaluation

Test	Result	Normal Range	Interpretation
Best Corrected Visual Acuity	RE/LE: 20/20	20/20	Normal
Refractive Status	RE: +5.75 -0.75 x 135; 2 pd BD LE: +5.25 sph ADD: +0.75	N/A	RE: Compound Hyperopic Astigmatism; Presbyopia LE: Simple Hyperopia; Presbyopia
Prescription Dispensed	RE: +5.50 -1.00 x 130; 2 pd BD LE: +5.25 -0.50 x 070 ADD: +1.25	N/A	RE/LE: Compound Hyperopic Astigmatism and Presbyopia
Distance Phoria	9 pd IAET with IO OA	0-2 exophoria	Intermittent Alternating Esotropia with Inferior Oblique Over-Action and Dissociated Vertical Deviation
Near Phoria	About 14 pd CAET with +4 RIO OA and +2 LIO OA	0-6 exophoria	Constant Alternating Esotropia with Inferior Oblique Over-Action and Dissociated Vertical Deviation
Nearpoint of Convergence	6 inches RE turns in	1-3 inches	Esotropia
Near Convergence Range	12 pd, RE suppression	18-24 pd	Infantile Esotropia
Near Convergence Recovery	RE suppression	7-15pd	Infantile Esotropia
Near Divergence Range	4 pd, RE suppression	16-24pd	Infantile Esotropia
Near Divergence Recovery	RE suppression	10-16pd	Infantile Esotropia
Distance Convergence Range	18 pd	14-24pd	Normal
Distance Convergence Recovery	12 pd	6-12pd	Normal
Distance Divergence Range	14-16 pd	5-10pd	Normal
Distance Divergence Recovery	12 pd	2-6pd	Normal
Distance RE Supra-vergence Range	14 pd	3 pd	Inferior Oblique Over-Action
Distance RE Supra-vergence Recovery	10 pd	1-2 pd	Inferior Oblique Over-Action
Distance RE Infra-vergence	RE Suppression	3 pd	Infantile Esotropia
Worth 4 dot (distance)	Shallow RE Suppression	Flat Fusion	Infantile Esotropia
Worth 4 dot (intermediate)	Shallow RE Suppression	Flat Fusion	Infantile Esotropia
Worth 4 dot (near)	Deep, intermittent RE suppression	Flat Fusion	Infantile Esotropia
Stereopsis (global)	0″	250″	Absent Stereopsis
Stereopsis (local)	0″	20″	Absent Stereopsis
Distance Fusional Facility	Suppression	≥15 cpm	Infantile Esotropia
Vergence Facility (near)	Suppression	≥15 cpm	Infantile Esotropia
Pursuit Eye Movements	+3	+3 or greater	Normal
Saccadic Eye Movements	+2	+3 or greater	Oculomotor Dysfunction
Test of Silent Word Reading Fluency	53rd %ile	38th %ile or better	Oculomotor Dysfunction
Developmental Eye Movement Test (DEM)	Speed: 1st %ile Errors: none (77th %ile)	38th %ile or better	Oculomotor Dysfunction
RightEye Reading Eye Movement Test 10th grade text	4th grade level	38th %ile or better	Oculomotor Dysfunction
Simultaneous Visual Memory: Tachistoscope	13th %ile	10th or greater grade level	Simultaneous Visual Memory Deficit
Sequential Visual Memory	14th %ile	38th %ile or better	Sequential Visual Memory Deficit

pd=prism diopters cpm=cycles per minute RE=right eye LE=left eye IAET= intermittent alternating esotropia BD=base down IO OA=inferior oblique overaction RIO OA=right inferior oblique overaction LIO OIA=left inferior oblique overaction Highlighted areas indicate abnormality

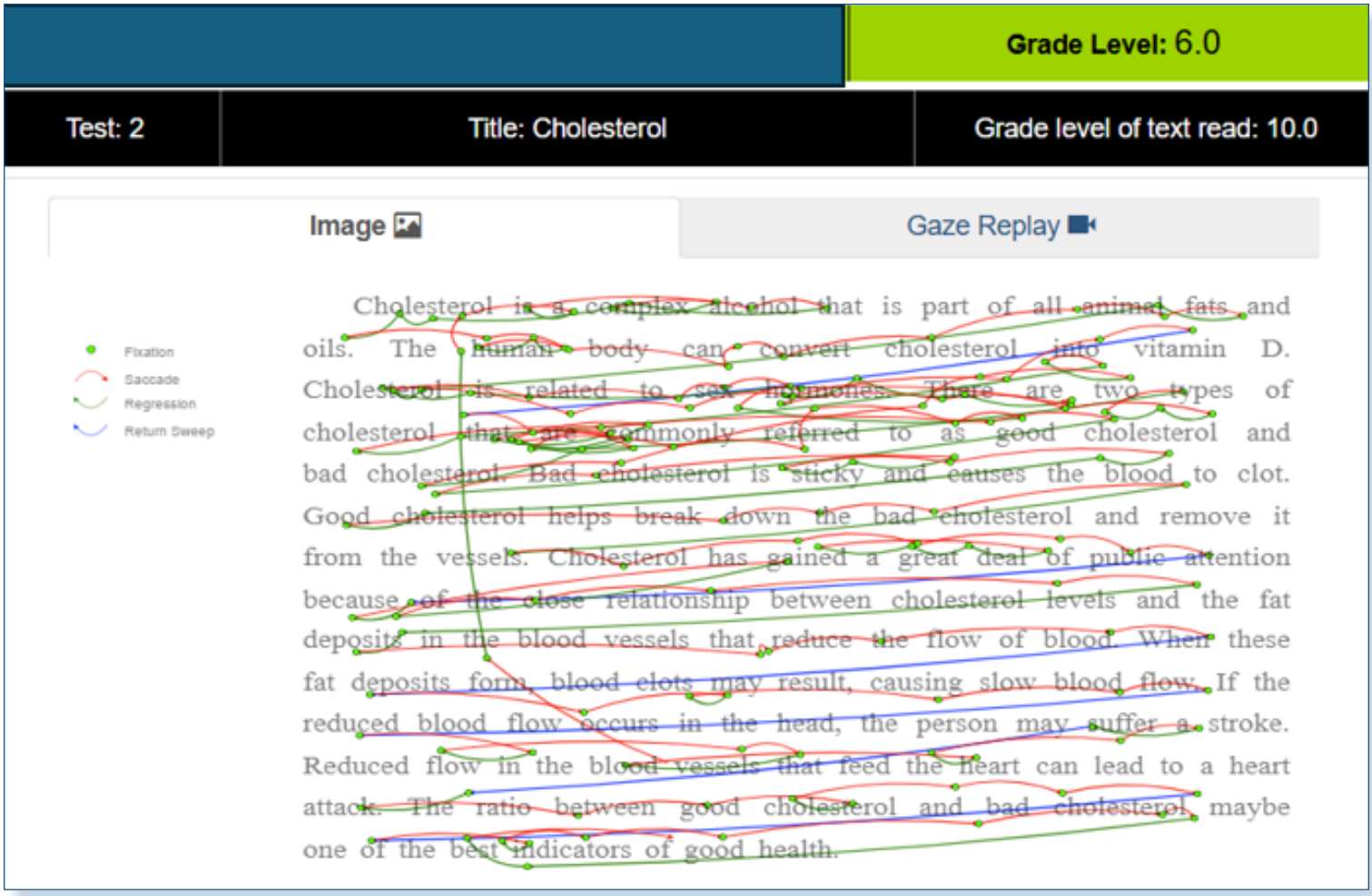

Figure 1. RightEye reading eye movement recording for Case 1

loss of place while reading and difficulty with visual tasks such as driving. Table 3 summarizes his neuro-optometric evaluation.

There were two recommendations made to the patient.

1. Spectacle Correction: The patient was dispensed a pair of single-vision distance spectacles. They were the same power and prism as his most comfortable habitual spectacles. He uses progressive spectacles for reading and computer use. Numerous chromatic tints were trialed (e.g., blue, BPI Omega, and BPI FL-41), but none were preferred by the patient.
2. Vision Therapy: A 6-week trial program (12 sessions) of in-office vision therapy was recommended to improve his eye coordination, visual tracking, and visual-vestibular integration with goals to reduce diplopia, improve visual comfort, enhance reading efficiency, and reduce visual motion sensitivity. Additional therapy sessions would likely be required depending on his progress. To date, he has not commenced the vision therapy program.

RESULTS

The results for the two cases are summarized in Tables 1-4 and Figure 1. There were several interesting and important findings.

First, neither of the patients reported VS. This was carefully and repeatedly queried in the case history resulting in definitive responses of 'no'.

Second, both patients reported palinopsia and photosensitivity, which are two of the four secondary visual symptoms necessary for the diagnosis of VSS. In both cases, the palinopsia was the most prominent and disturbing visual symptom. In addition, tinnitus, tremor, migraine, and balance problems were reported by each. Other tertiary symptoms reported were photopsia and night vision

problems, as well as anxiety which is common to this population.[5]

Third, there was a wide range, and high frequency, of oculomotor and binocular visions problems diagnosed in the clinical examinations, which mirrored their visual symptoms. This is consistent with earlier reports in the VS/VSS population.[6] And, interestingly, both had a history of strabismus, a motor abnormality.

Fourth, in the objective, reading eye movement records of case one, there were two abnormalities noted (Table 4 and Figure 1). Reading speed was reduced by approximately 20% as compared to normative data, which was reflected in reading of the grade level 10 paragraph at the grade 6 level. This significant reduction in reading speed is consistent with earlier findings.[6] Second, there was a large difference in the gaze disparity value as compared to normative findings. Presumably, this reflected presence of alternating fixation during the testing, as the patient was an intermittent, alternating esotropia.

Table 4: RightEye reading results for Case 1

Reading Metrics	My Eyes	Grade Average
Correct Comprehension Answer (%)	90	70
Reading Rate (wpm)	178	224
Fixation # (per 100 words)	101	101
Average Fixation Duration (ms)	231	260
Regression # (per 100 words)	21	19
Regression/Fixation Ratio (%)	20	19
Gaze Disparity (mm)	19	4

DISCUSSION

VSS without VS appears to be a new, unusual, and important neuro-perceptual, neuro-motor variant of VSS. We have searched the literature carefully and have not found any mention of such a diagnostic group having this specific constellation of visual and non-visual symptoms. However, as a tentative, new diagnosis, it needs to be confirmed by others in the field, especially regarding its prevalence, as well as treatment options and their success.

Both patients had the following symptoms: palinopsia, photosensitivity, tinnitus, balance problems, and migraine. Since migraine has been found in only 9.8% of those reporting palinopsia,[7] this newly-proposed diagnosis does not appear to simply be a migraine variant. Furthermore, since neither had any related ocular or neurological diseases, these too can be ruled out as possible contributory factors. Lastly, and most importantly, neither patient reported VS after careful, repeated query, hence the proposed new diagnosis.

What can the neuro-optometrist offer to these and related patients (i.e., visual snow syndrome)? There are a wide range of potentially helpful options. First, since many of these patients manifest anxiety,[7] simple reassurance that the condition is almost always benign immediately sets their mind at ease. In addition, for some with residual anxiety, referral to a mental health specialist (e.g., psychologist or psychiatrist) for coping strategies or medications, respectively, might be fruitful. Second, there are specific chromatic spectacle tints that may alleviate/reduce some of their visual symptoms, such as photosensitivity and palinopsia.[8] Third, saccadic tracking training has been found to reduce the perception of palinopsia. presumably by re-establishing a more normal saccadic suppression mechanism/threshold6. Fourth, since many (~60%) have concurrent oculomotor dysfunctions, such as vergence and accommodative insufficiency, a basic regiment of oculomotor-based vision therapy would be beneficial.[6] Lastly, some report balance problems, so referral to a neurologist and vestibular specialist would be in order.

The areas of VSS and VS have had a resurgence of interest over the past decade or so.[1] This is important, as they represent baffling neurological conditions that are problematic to both the patient and doctor. While therapeutic interventions have provided symptomatic relief for many of these patients in general,[1] the precise underlying neurological, physiological, and perceptual pathways remain equivocal and highly speculative, thus leading to confusion and frustration in these literatures, hence an enigma. Further work is needed to understand this most intriguing area of both clinical and vision science, which includes the rarity of VSS without VS.

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Dr. Tannen received the A.M. Skeffington award for excellence in Optometric Writing from the College of Optometrists in Vision Development in 2016, the Ludlam Education Award by the Neuro-Optometric Rehabilitation Association in 2014, and the Scientific Achievement Award by the New Jersey Society of Optometric Physicians in 2002. He is a Fellow in the American Academy of Optometry, and the College of Optometrists in Vision Development (COVD), Past President of COVD, and an Associate Clinical Professor Emeritus of the SUNY/State College of Optometry in New York. Dr. Tannen lectures internationally and has co-authored over 60 journal articles, as well as a clinical textbook on Eye Movements.