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Visual Snow Syndrome in a Medical Professional: A Case Report and Comprehensive Literature Review

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Abstract

Visual Snow Syndrome (VSS) is a rare, chronic neurological condition characterized by a persistent, dynamic "TV-static" like disturbance across the entire visual field. We present the case of a 32-year-old male medical resident who experienced a sudden onset of VSS following a period of high academic stress and a viral infection. Despite the severity of symptoms, including palinopsia, nyctalopia, and tinnitus, neuro-ophthalmologic examinations—including optical coherence tomography angiography (OCT-A) and automated perimetry—revealed no structural abnormalities beyond a bilateral posterior vitreous detachment (PVD). This case highlights the clinical challenge of VSS, where structural integrity contrasts with severe sensory dysfunction. We review the current literature on pathophysiology, emphasizing cortical hyperexcitability and thalamocortical dysrhythmia, and discuss management strategies, including the patient's subjective improvement with escitalopram, a response rarely documented in broader cohorts.

Keywords

Visual Snow Syndrome, Palinopsia, Cortical Hyperexcitability, Neuro-Ophthalmology, Thalamocortical Dysrhythmia

1. Introduction

Visual Snow Syndrome (VSS) is a neurological disorder characterized by continuous visual disturbance, described by patients as the perception of countless tiny, dynamic dots covering the entire visual field, similar to the "static" of a poorly tuned analog television. Although first described in 1995, its formal recognition has grown recently, and it has been included in the International Classification of

Headache Disorders (ICHD-3) as a complication of migraine [1] [2] (See **Figure 1**).

Figure 1. Description of the perception of the VNS.

2. Case Presentation

A 32-year-old male patient reported palinopsia, excessive entoptic phenomena, night blindness, and transient photophobia, with a duration of more than 12 months. The onset was sudden following severe academic stress and a prior viral infection. He denied migraine, head trauma, or hallucinogen use, with a personal history of anxiety and bilateral tinnitus, and a family history of migraine. The ophthalmological examination was structurally normal, with a visual acuity of 20/20, and no abnormalities of the anterior segment, fundus, or optic nerve that would explain the symptoms. Additional studies included optical coherence tomography (OCT) and ocular ultrasound, confirming bilateral posterior vitreous detachment without additional pathological findings, visual fields demonstrating a normal profile in the right eye, while the left eye showed localized sensitivity fluctuations, which is within the normal range for this test. He received escitalopram, with partial subjective improvement in the intensity of static, along with the use of color filters, mental distraction techniques and the discontinuation of exacerbating factors such as caffeine, following the previous failure of flunarizine treatments.

3. Theoretical Framework

3.1. Epidemiology

VSS typically manifests in young adults between 19 and 29 years of age, without a clear sex predominance. The most common comorbidities—often linked to increased symptom severity—include migraine (50% - 72%) and tinnitus (62% - 75%). Anxiety and depressive disorders are also reported in up to 35% of cases [1]-[3].

3.2. Pathophysiology

The etiology of visual snow syndrome is idiopathic; however, current evidence suggests a neurofunctional origin. The main hypotheses are: 1) Cortical hyperex-

citability: Specifically in the secondary visual cortex and the right lingual gyrus (Brodmann area 19), where PET studies have detected hypermetabolism. 2) Thalamocortical dysrhythmia: A dysfunction in the filtering of sensory stimuli that allows the conscious perception of normally ignored visual noise. 3) Salience network dysfunction: Alterations in connectivity that prevent adequate habituation to visual stimuli [2] [4]-[6].

3.3. ICHD-3 Diagnostic Criteria

1) Criterion A: Continuous, dynamic visual snow present throughout the visual field for 3 months or more. 2) Criterion B: Presence of ≥2 of the following: palinopsia, photophobia, nyctalopia (impaired night vision), enhanced entoptic phenomena. 3) Criterion C: Symptoms must not be consistent with a typical migraine aura. 4) Criterion D: Symptoms are not better explained by another disorder [1] [2] [4].

3.4. Differential Diagnosis

It is imperative to perform neuro-ophthalmological examinations that turn out normal (visual acuity, fundus examination, visual fields, electroretinogram and brain magnetic resonance imaging) to rule out retinal pathologies, tumors or Hallucinogen Persistent Perception Disorder (HPPD) [1] [3] [7].

3.5. Therapeutic Strategy

The use of colored filter lenses (especially blue, yellow, or FL-41 filters) helps reduce the intensity of snow and photophobia in a high percentage of patients. Cognitive Behavioral Therapy (CBT) is emerging as a promising option to improve quality of life and decrease the emotional burden of the disorder. [1] [3] [8]. Regarding pharmacotherapy, lamotrigine is the drug with the best results (partial efficacy in 61.5% of patients), followed by benzodiazepines with a reported partial relief rate of 71.4%. SSRI antidepressants are usually ineffective or even worsen symptoms [3] [4] [8].

4. Discussion

The patient strictly meets the ICHD-3 diagnostic criteria for visual snow syndrome, presenting both criterion A (continuous visual static across the entire visual field) and criterion B, including palinopsia, excessive entoptic phenomena, photophobia, and night blindness [4].

The symptomatology is consistent with that described in the literature, particularly floaters, afterimages, and photophobia, although the absence of migraine is noteworthy, which is interesting given the very high prevalence of migraine in patients with visual snow syndrome (between 50% and 72% according to various studies). However, there is a strong familiar genetic predisposition (mother and sisters with migraine), suggesting a hereditary predisposition to cortical hyperexcitability [1] [2].

The onset of VSS in this patient began suddenly in December 2024, immediately following a viral flu episode and a period of severe academic stress related to medical residency. The primary symptom is a binocular, pan-field "TV static" composed of dynamic white dots that persist continuously, even with eyes closed, for over 12 months.

The literature indicates that, although 40% of patients have had symptoms for as long as they can remember, a sudden onset occurs in approximately 26% of cases, often without an apparent cause or following events such as migraine or stress. The patient reports a chronic course without remission for more than a year, a typical pattern of VSS. Furthermore, caffeine and sleep deprivation act as aggravating factors in this patient, which is consistent with reports where stress and caffeine worsen the perception of snow [2] [3] [4] (See **Figure 2**).

Neuro-ophthalmological studies were structurally normal, including visual fields and optical coherence tomography (OCT), confirming a bilateral posterior vitreous detachment, insufficient to explain the visual snow, thus supporting a central origin. Distinctive findings include pulse-dependent modulation of visual static and subjective improvement with escitalopram, elements that are rarely reported and add value to this case, reinforcing the need for a precise clinical diagnosis and a multidisciplinary approach. However, the mechanism of the use

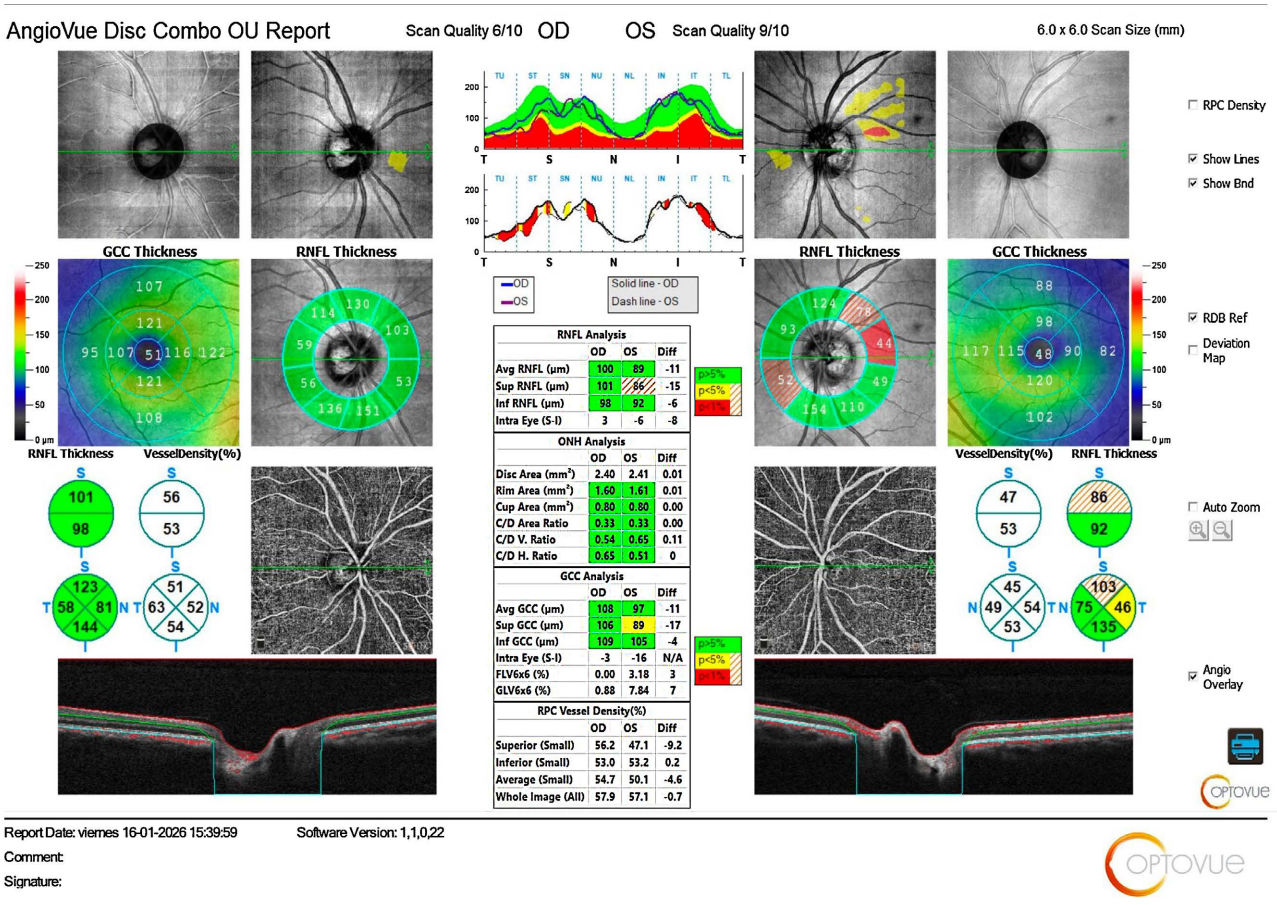


Figure 2. Optic disc OCT—thickness of the nerve fiber layers is 100 µm in the right eye (OD) and 89 µm in the left eye (OS).

of escitalopram remains speculative [1] [7] (See **Figure 3**).

The patient's visual acuity is 20/20 bilaterally, visual fields within normal limits in the right eye (VFI 99%) and minimal sensitivity fluctuations in the left eye (VFI 98%), consistent with the literature reporting normal visual fields in the vast majority of patients. Optical coherence tomography (OCT) and angio-OCT confirmed a bilateral posterior vitreous detachment (PVD), which explains the floaters and acute photopsias. However, the persistent "visual snow" and palinopsia cannot be attributed to this structural condition, reinforcing that VSS is a central visual processing disorder, possibly due to hyperexcitability of the visual cortex. Brain computed tomography (CT) scans showed structurally normal results, supporting the diagnosis of VSS. While Magnetic Resonance Imaging (MRI) is the preferred modality to rule out subtle demyelinating or ischemic pathology, it was not performed in this instance [4] [7].

The diagnostic workup for this case was designed to meet ICHD-3 criteria by confirming the structural integrity of the visual system while ruling out organic mimics. Tests performed included Best-Corrected Visual Acuity (BCVA), which was 20/20 bilaterally, automated perimetry (Humphrey 24-2 SITA Faster), spectral-domain OCT of the optic nerve and macula, OCT Angiography (OCT-A),

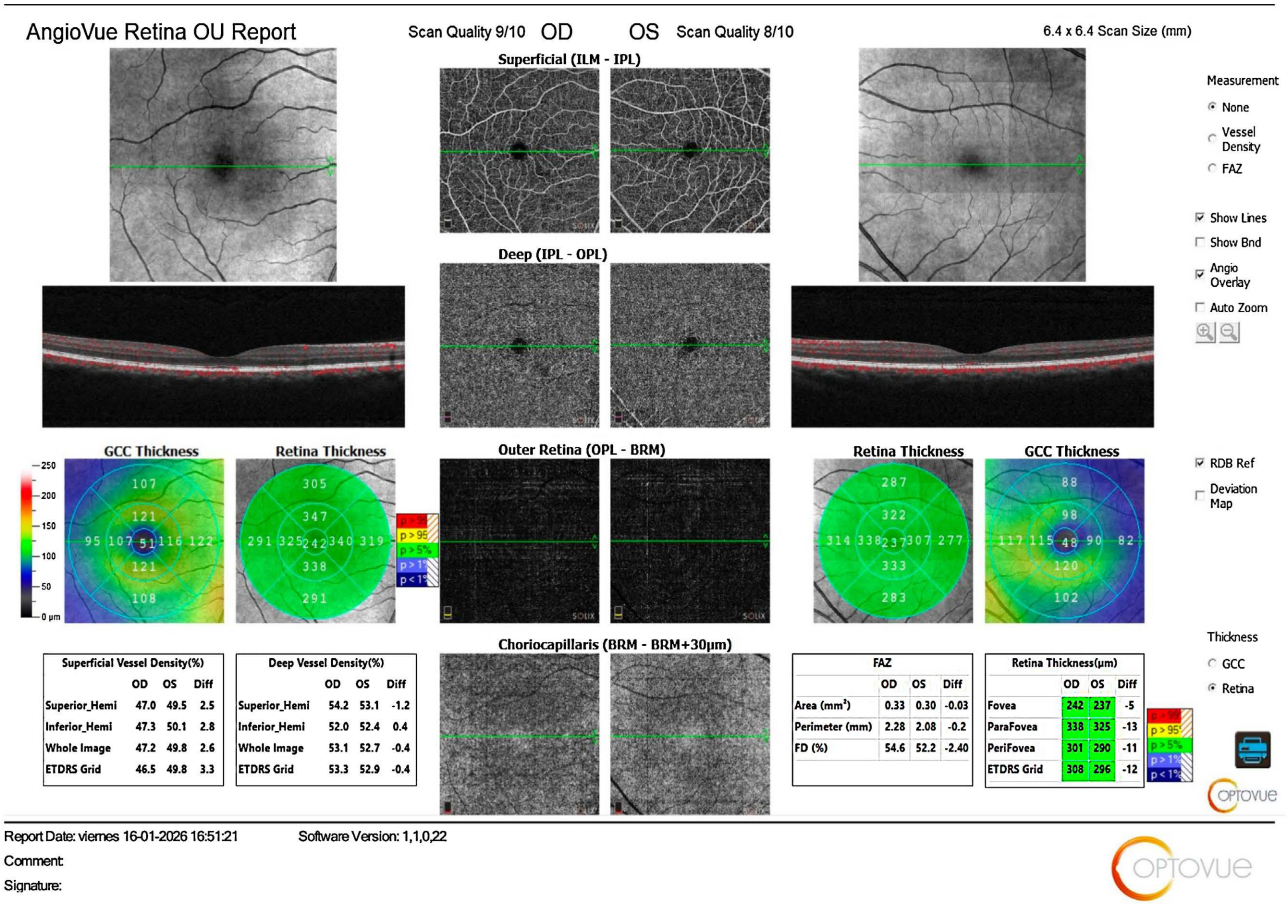


Figure 3. Macular OCT—foveal thicknesses are 242 µm (OD) and 237 µm (OS), Foveal Avascular Zone (FAZ) area (0.33 mm^2 in OD and 0.30 mm^2 in OS).

and a simple brain CT. Notably, Brain MRI and Electroretinography (ERG) were not obtained, as they were not explicitly indicated after initial structural tests showed complete retinal and vascular integrity and no focal neurologic deficits. A simple brain CT was selected as the primary neuroimaging modality to rapidly exclude gross lesions, space-occupying masses, or evidence of intracranial hypertension following the patient's sudden onset of symptoms during a high-stress period. Furthermore, CT was chosen over MRI for economic reasons, as MRI is extremely expensive [1] [3] (See **Figure 4**).

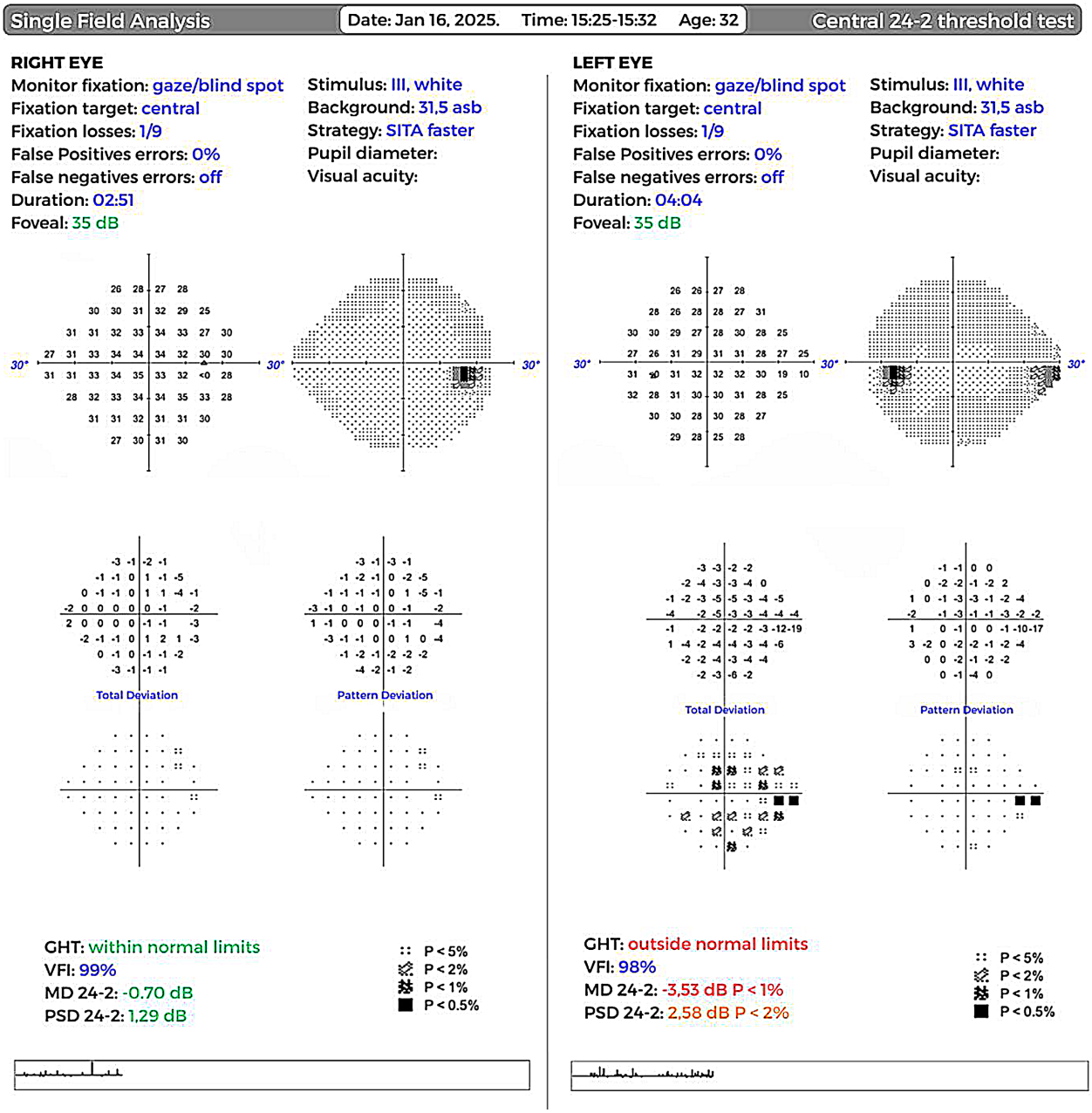


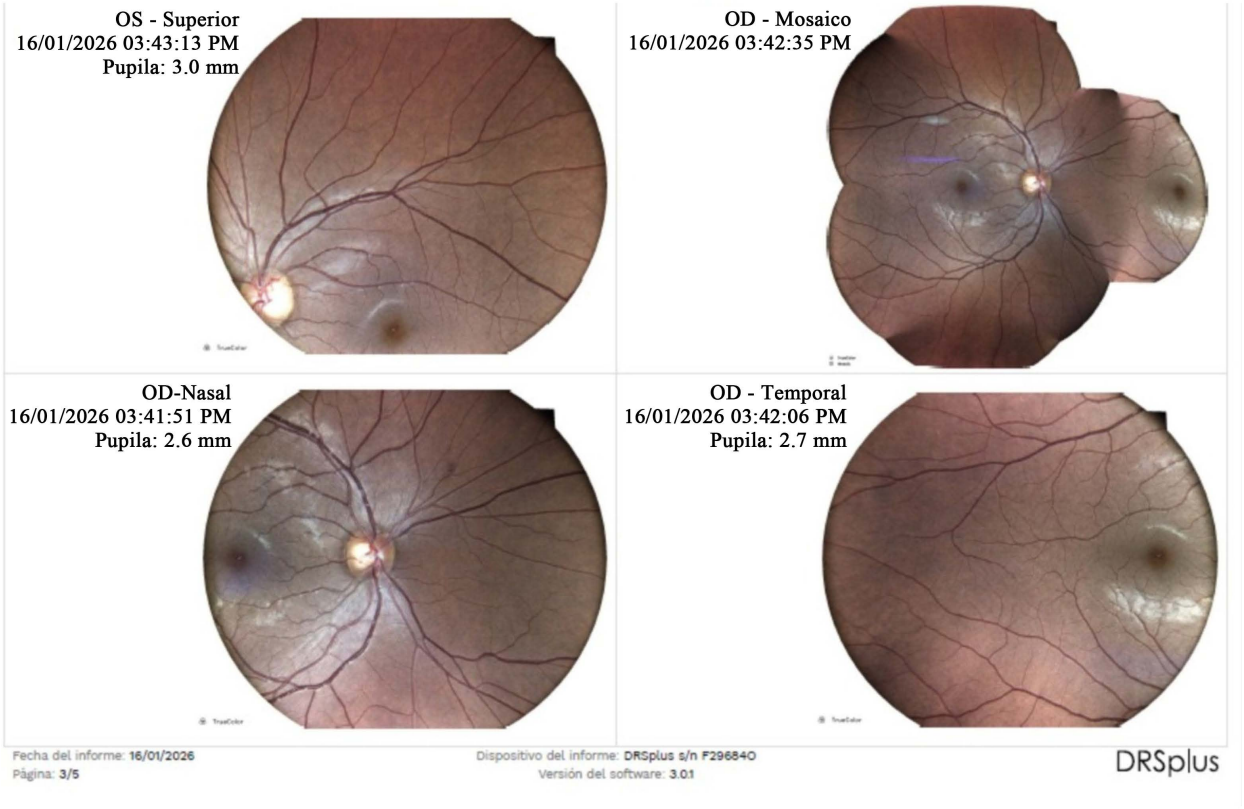
Figure 4. Automated static perimetry (OS). The analysis reveals a VFI of 98% and an MD of −3.53 dB (P < 1%). The localized reduction in sensitivity is classified as a functional artifact due to the absence of corresponding anatomical lesions in the OCT and Brain CT. This reflects subjective interference from visual snow during the examination.

A distinctive finding in this case is the pulse-synchronous modulation of the visual static, where the patient perceives the intensity of the "snow" fluctuating in coordination with his arterial pulse during episodes of tachycardia. This phenomenon is not widely documented in standard reviews and suggests a state of high autonomic arousal and increased attentional salience, possibly as a byproduct of thalamocortical dysrhythmia. This case generates the hypothesis that the failure of central filtering mechanisms in VSS allows for the conscious awareness of internal physiological noise (vascular pulsations), similar to the mechanism proposed for tinnitus, the auditory equivalent of visual snow. This framing suggests that cortical hyperexcitability may be exacerbated by cardiovascular triggers when the brain's "noise-canceling" systems are disorganized [1] [6] [7].

Furthermore, the subjective improvement in static intensity with escitalopram use is noteworthy. This is significant because the literature generally shows that antidepressants are often ineffective or even worsen symptoms in many patients (only 6.7% report improvement). The patient reported a significant subjective improvement after treatment, scoring 8 out of 10 on the severity scale in the intensity of the static following the implementation of combined medical and lifestyle interventions. This perceived improvement marked a transition from a state of high anxiety and functional disability to one of increased tranquility and habituation [2]-[4].

Regarding pharmacological management, the patient was prescribed escitalopram at a dose of 10 mg daily from January to October 2024 (10 months) for comorbid anxiety. A subjective improvement in the perceived intensity of the visual static (scoring 8/10 on the severity scale) was noted after the first month of treatment. However, the patient experienced several adverse effects, including restless sleep, vivid nightmares, and a notable decrease in libido. Prior to the Selective Serotonin Reuptake Inhibitors (SSRI) trial, treatment with flunarizine had been attempted under neurological supervision; it was discontinued as it provided no symptomatic relief and resulted in significant weight gain. Simultaneously with the medication, the patient implemented a multifaceted approach involving the cessation of caffeine, the initiation of regular physical exercise (walking and gym), and the use of photochromic lenses. The patient described these lenses as a 'brain distraction' technique, as they modulate luminosity by darkening in bright environments and remaining clear in low light.

The differential diagnosis was strengthened by screening for red flags such as acute unilateral deficits, focal neurologic signs, and head trauma, all of which were negative. Hallucinogen Persistent Perception Disorder (HPPD) was explicitly excluded through a thorough history in which the patient confirmed he has never used recreational drugs, LSD, or psilocybin. Retinal and optic nerve disorders were excluded by the 20/20 BCVA, normal global RNFL thickness (100 μm OD and 89 μm OS), and normal foveal avascular zone (FAZ) areas (0.33/0.30 mm^2) on OCT-A. These normal findings, combined with the lack of pigmentary changes, allowed for the exclusion of rod-cone dystrophy and other peripheral retinal pathologies that can manifest with visual snow-like symptoms (See **Figures 5**-7).



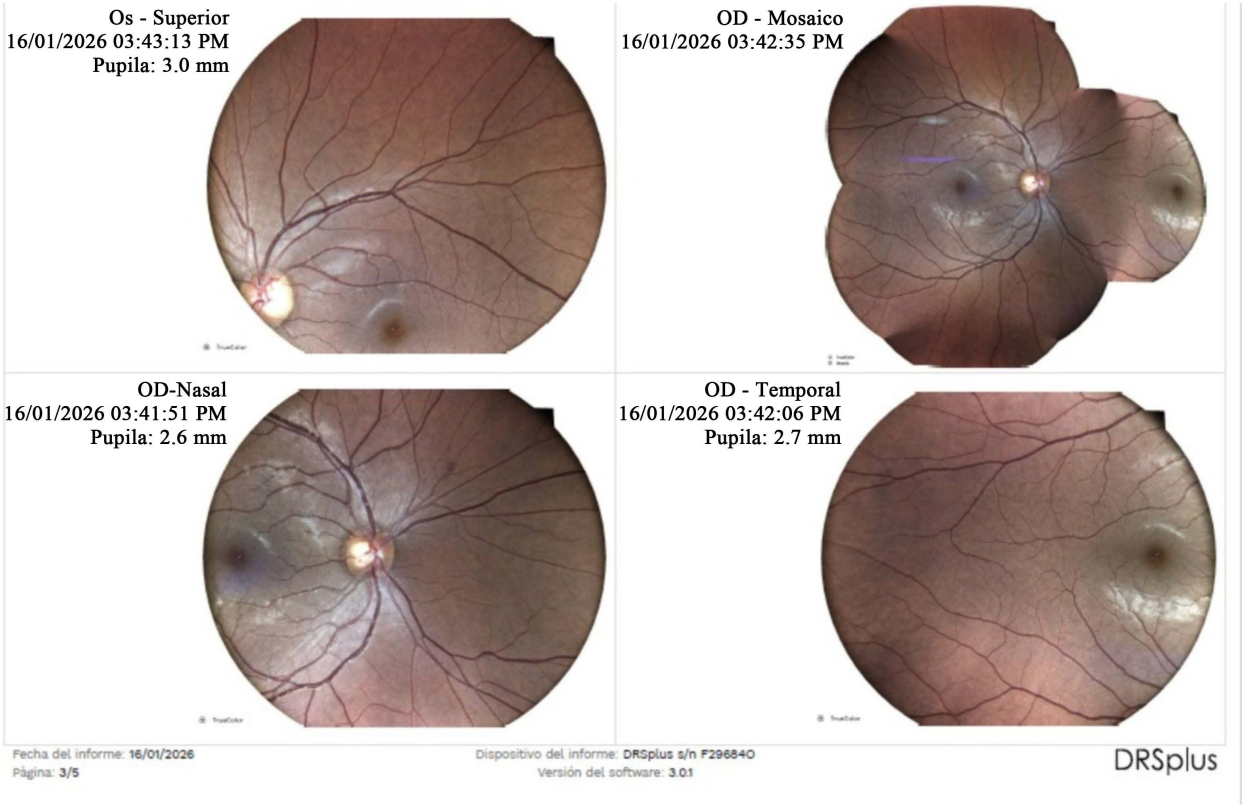


Figure 5. True-color confocal fundus photography (DRSplus) shows a structurally normal retina in both eyes, with sharp optic disc margins, normal foveal reflex, intact vasculature, and no lesions, pigmentary changes, or exudates.

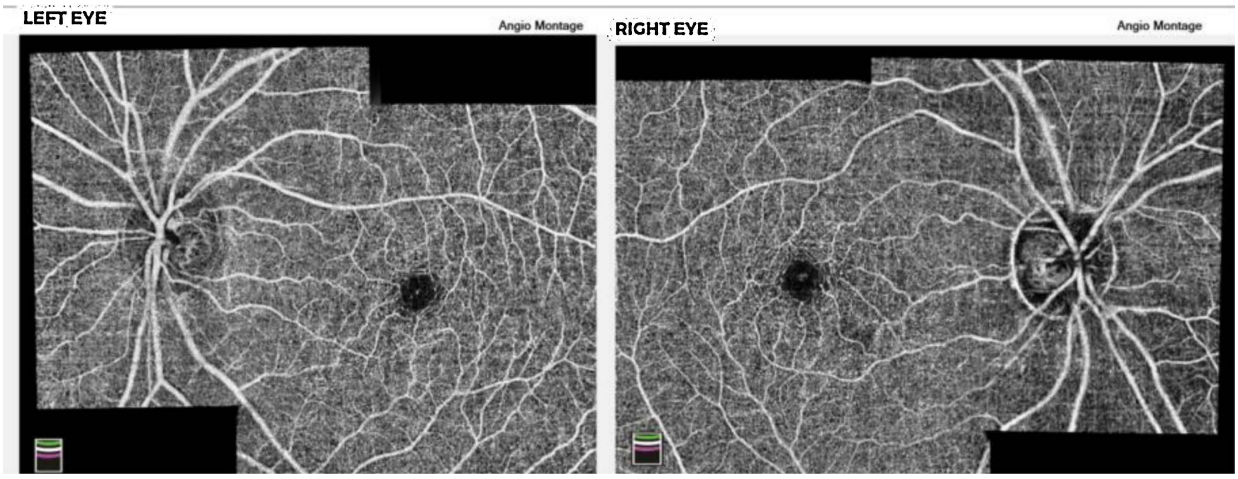


Figure 6. Angio-OCT reveals a vascular architecture without areas of non perfusion or telangiectasias.

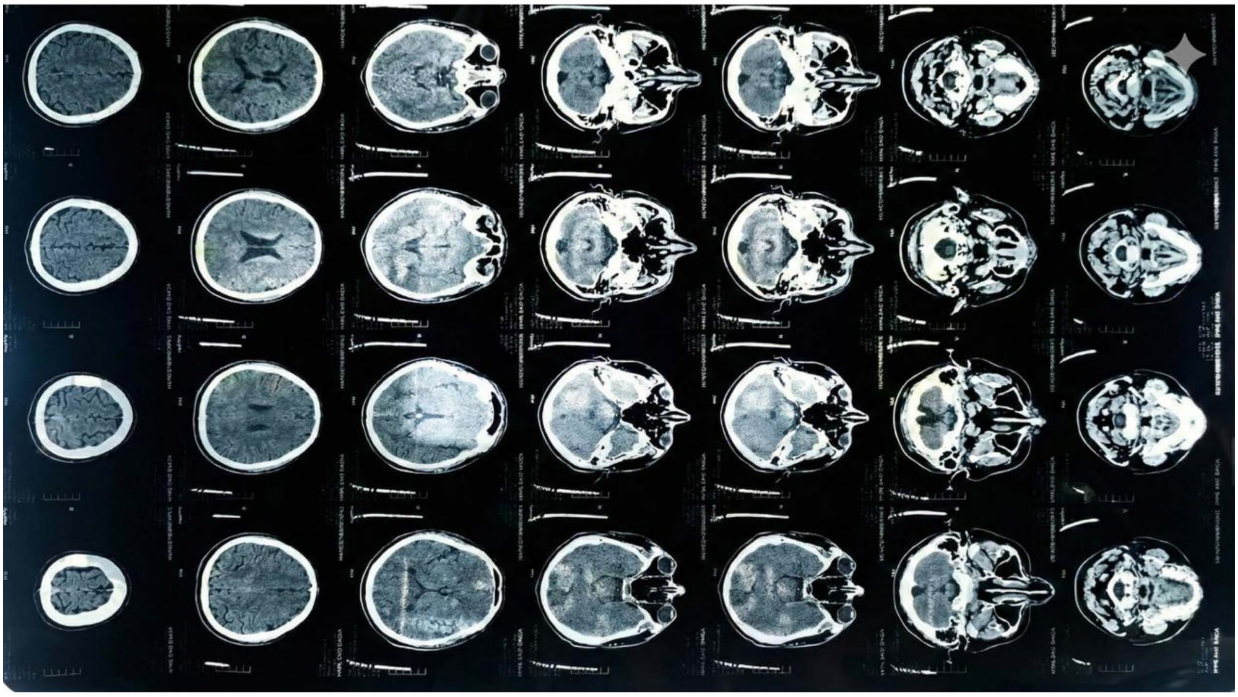


Figure 7. The computed tomography scan of the skull performed on the patient shows structurally normal results.

5. Conclusion

VSS represents a clinical continuum where structural normalcy masks profound functional disability. Clinicians must distinguish between structural causes of photopsias, such as PVD, and the global processing dysfunction of VSS. Early clinical diagnosis is vital to reduce patient anxiety and prevent unnecessary invasive testing.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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