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Case Report

Semaglutide Associated with Central Serous Chorioretinopathy: A Case Report

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Abstract: This clinical case examines central retinal serous chorioretinopathy in a patient undergoing treatment with semaglutide. The subject of the case is a middle-aged male who developed this retinal condition following the administration of 14 mg of oral semaglutide. He reported a variety of vision-related disturbances that significantly impacted his daily activities. Fortunately, the visual impairments were completely resolved upon discontinuation of the medication. This case underscores the critical importance of vigilant ophthalmological monitoring for patients receiving semaglutide. It is imperative to monitor for any potential adverse effects on vision, thereby facilitating the prompt recognition and management of any changes or complications. By prioritizing regular eye examinations, healthcare providers can protect patients' visual health and promptly address any issues that may arise during their treatment.

Keywords: Obesity; Semaglutide; Central Serous Chorioretinopathy.

1. Introduction

Adult obesity is a nutritional disturbance involving excess fatty tissue, indicated by a body mass index (BMI) of 30 kg/m² or higher, while a BMI of 25 to 29.9 means overweight [1]. In Brazil, the National Health Research (PNS, 2020) demonstrates more than half of the adult population, a total of 60,3%, have been diagnosed as overweight, with a prevalence of 62,6% in the female population as opposed to 57,5% in the male population [2]. Meanwhile, in the global scenario, the prevalence of obesity or overweight population has increased by 27,5% from 1980 to 2013 in the adult population.[3] Obesity is associated with an increase in annual health costs, has negative impacts on education and school absence, and has a long-term impact on earning and social relations, witnessed as obese women have more significant difficulties in job scenarios and productivity.

The first treatment for obesity is modifying life habits, primarily related to nutritional orientations to lower calorie consumption. However, pharmacologic therapy may be used when these tactics fail to reduce weight, depending on adequate physical activity frequency and calorie control. As of 2016, obesity treatment in Brazil involved using one of three approved medications: Sibutramine, Orlistat, and Liraglutide.[4]

Numerous clinical trials have provided compelling evidence for the effectiveness of semaglutide as a treatment for obesity. A notable example is the STEP 1 trial, which was a rigorously designed, randomized, double-blind, multicenter, placebo-controlled study. This trial enrolled 1,961 adults who were classified as obese (with a Body Mass Index, or BMI, of 30 kg/m² or higher) or overweight (BMI of 27 kg/m² or above) and who also had at least one weight-related medical condition. Importantly, none of the participants had diabetes. The primary aim of the STEP 1 trial was to assess and compare the impact of administering semaglutide at a dosage of 2.4 mg against a placebo, particularly in conjunction with a structured lifestyle intervention program. The results were striking, as participants who received semaglutide 2.4 mg experienced an average weight loss of 16.9%. In contrast, those receiving the placebo only lost an average of 2.4%. Moreover, individuals treated with semaglutide were significantly more likely to achieve notable milestones in weight reduction, with higher percentages of participants reaching weight loss thresholds of 5%, 10%, 15%, and even 20%. This study underscores the potential of semaglutide as a powerful tool in the management of obesity, particularly for individuals struggling with related health issues [5].

In 2017, semaglutide was approved for glycemic control in type 2 diabetes with the commercial name Ozempic®. In 2021, semaglutide was approved for long-term obesity treatment by the FDA and commercialized as Wegovy®. Its usage was authorized in Brazil in 2023.[6] Semaglutide is a glucagon-like peptide-1 receptor agonist.[7] Semaglutide treatment significantly reduced fat and lean mass due to improved efficiency in skeletal muscle oxidative phosphorylation.[8] This article aims to describe the use of semaglutide for treating obesity and an undescribed adverse event. Reviewing the literature indexed in Pumed, Lilacs, and Embase published up to May 2025, we did not find any article that refers to the association between central retinal sclerosis and the use of semaglutide. Therefore, this is the first case report that we are aware of.

2. Case Report

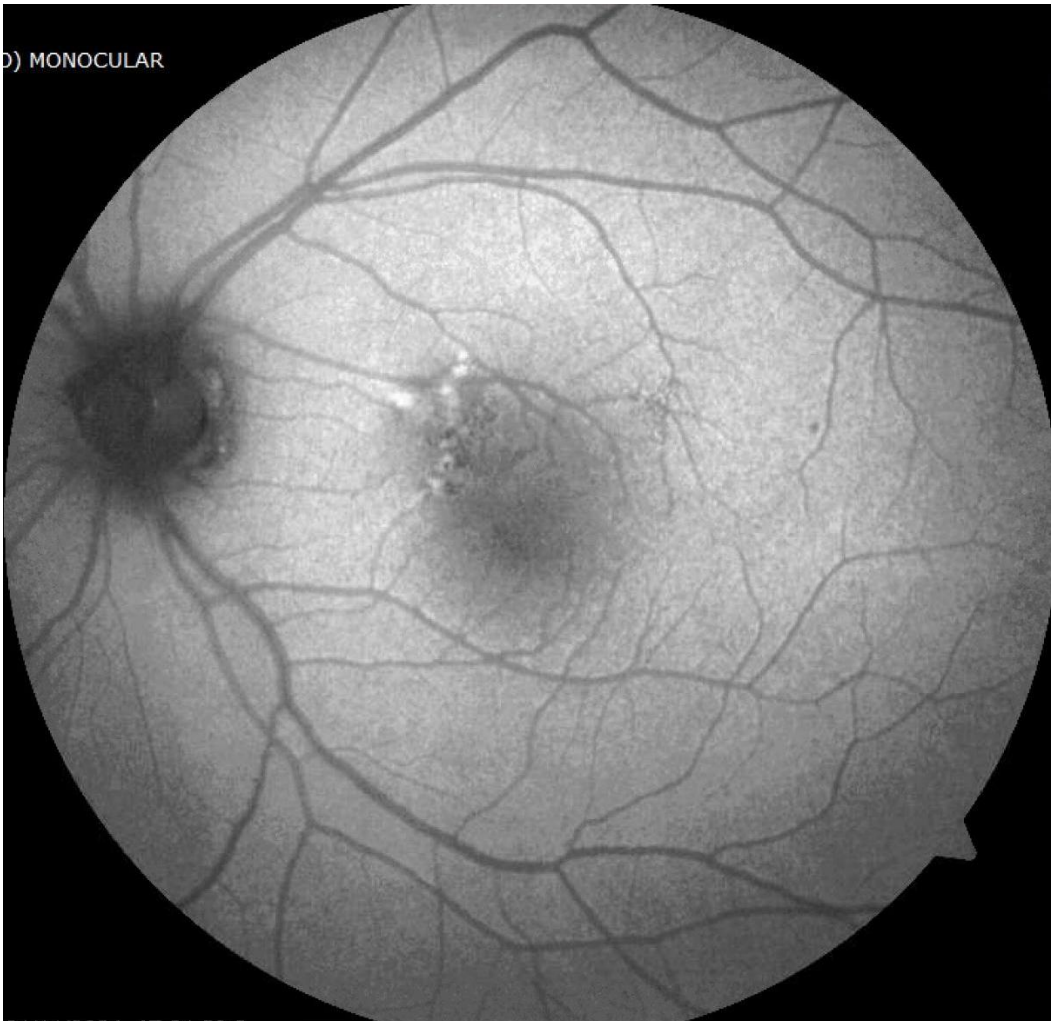
A 47-year-old man diagnosed with obesity (body mass index = 31) was prescribed 3 mg of daily semaglutide (Rybelsus®) in January 2024. After a month of treatment, he lost 2 kg (reduction from 96 to 94 kg) and did not present any side effects other than constipation. At this time, he was instructed to increase the semaglutide dose to 7 mg daily. After six months of maintenance, his weight reached 86 kg. The label recommendation for semaglutide is to increase from 7 mg to 14 mg after 30 days if adequate glycemic control is not achieved. As this is not the clinical indication for the patient in question, we chose to continue at a lower dose for a longer period before increasing to the full dose.

As he did not experience any side effects, the goal was to reduce his body mass index to 25 by losing another 8 kg, and the dose of semaglutide was readjusted to 14 mg on alternate days. After two more months, he achieved a weight loss of 3 kg, reaching 83 kilograms. Always maintaining good tolerance, the 14 mg dose of semaglutide began to be used daily. After two weeks, he started to experience floaters, blurred vision, and aura light. He was advised to undergo ophthalmological examinations, which identified serous retinal detachment and choroidal thickening in the peri-macular region, characterizing central serous chorioretinopathy (CSC) (Figure 1 and 2). The optical coherence tomography (OCT) and angiography did not demonstrate ischemic changes in the optic nerve. Semaglutide was discontinued, and a watchful waiting approach was proposed without interventions to observe clinical progress. After two weeks, the patient no longer presented any ocular symptoms.

The patient is sedentary, has no insomnia, and does not work night shifts. He had no previous history of diabetes. His plasma glucose, cholesterol, and triglycerides were regular until treatment discontinuation. He had a history of cholecystectomy 8 years ago and continued to use finasteride 5 mg and rosuvastatin daily, which he had been taking for over 10 years. He does not have any cardiovascular disease, is not a smoker, and does not use illegal drugs. He was not under a stress regime other than his daily work, he had not

used corticosteroids in the last year, and his basal cortisol was normal. Polysomnography was performed and did not reveal sleep apnea. Serology for inflammatory processes was normal. The Naranjo scale was applied to assess the probability that the symptoms were an adverse event of semaglutide [9]. We obtained a score of 5, which suggests that the cause of CSC was probably caused using semaglutide.

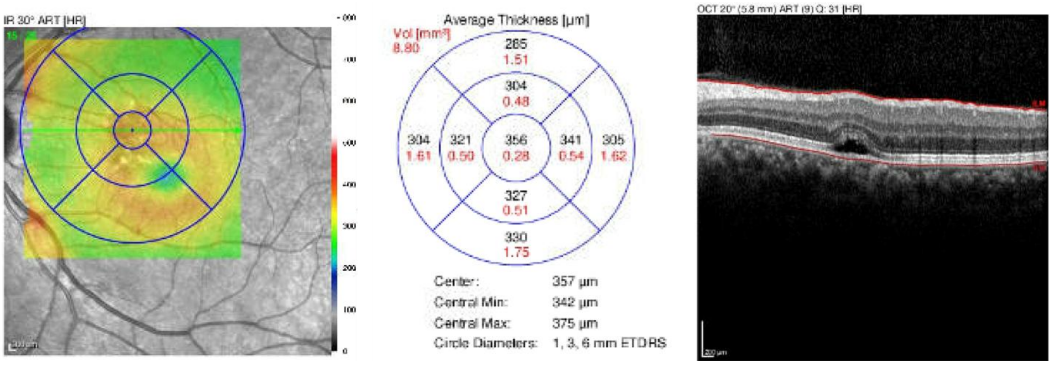
Figure 1. Retinography shows discrete blurring of vessels central to the macula in the left eye. The red-free image confirms central serous chorioretinopathy. The exam is in the patient's possession and archived in their electronic medical record.



3. Discussion

A comprehensive meta-analysis examining the effects of semaglutide revealed significant secondary outcomes related to safety concerns. Specifically, the analysis highlighted that individuals treated with semaglutide faced a 1.59-fold increase in the risk of encountering gastrointestinal adverse events. Furthermore, patients in the semaglutide group were found to be twice as likely to discontinue treatment due to these adverse effects. The occurrence of serious adverse events was also notable, with those receiving semaglutide experiencing a 1.6-fold greater likelihood of such events compared to other treatments. Most of these serious events were linked to gastrointestinal and hepatobiliary disorders, with particular attention drawn to cases of acute pancreatitis and cholelithiasis. These findings underscore the need for careful monitoring of patients on semaglutide to manage and mitigate potential risks effectively [10].

Figure 2. Optical Coherence Tomography (OCT) showing findings compatible with a small retinal detachment in the left eye. The report confirms central serous chorioretinopathy. The exam is in the patient's possession and archived in their electronic medical record.



The American Academy of Ophthalmology and the North American Neuro-Ophthalmology Society advise on the side effects of semaglutide for the eyes. They alert to the risk of nonarteritic anterior ischemic optic neuropathy (NAION) in patient-prescribed semaglutide.[11] We are aware of a single clinical case of a 72-year-old man who presented bilateral scotomas associated with posterior vitreous detachment that were reversible upon discontinuation of oral semaglutide [12] We are unaware of previous reports of the association of oral semaglutide use with CSC.

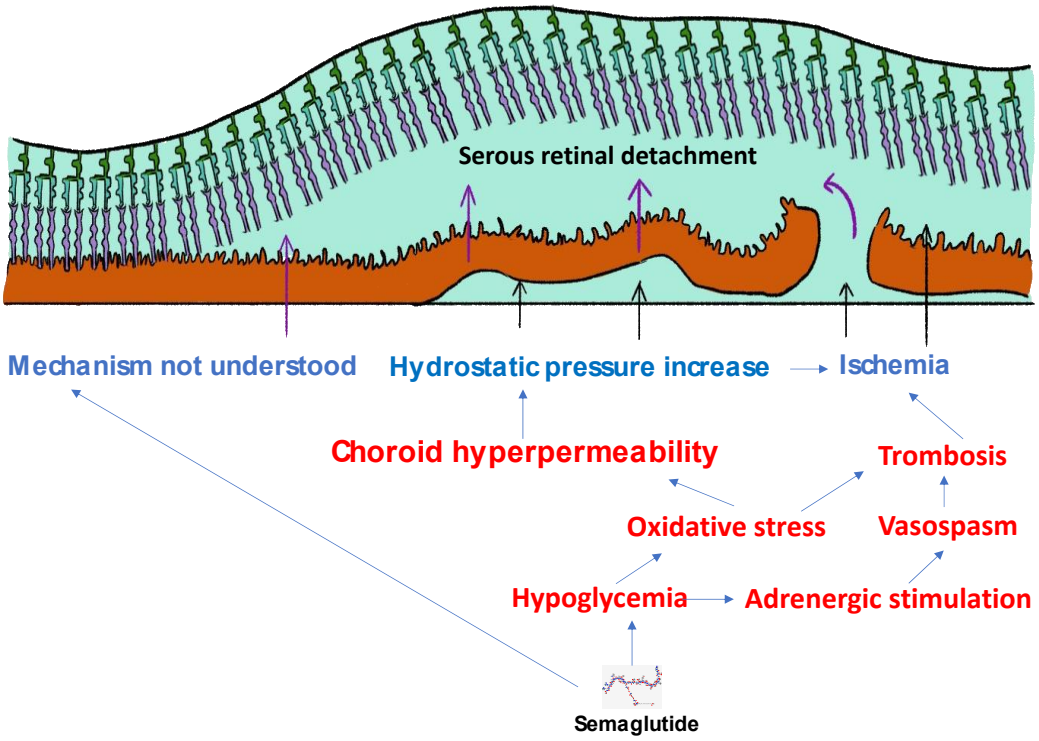
CSC has no widely accepted pathophysiology identification. However, there have been hypotheses explaining the alterations in the outer blood-retinal barrier. Patients with CSC have been shown to have thicker sclera, demonstrating a narrow passage of the scleral channel and provoking venous congestion. Other studies suggest a possible association with systemic inflammatory markers, which can provoke the production of reactive oxidative species that damage the retinal pigment epithelium (RPE), while activated platelets contribute to ischemia. Corticosteroids have also been strongly associated with such developments, dysregulating the dynamics in the choroid, provoking choroidal vessel permeability, and damaging the RPE barrier by stopping ion transportation [13].

There is no clear evidence of the mechanisms by which semaglutide may cause CSC. Semaglutide is generally associated with an improvement in diabetic retinopathy. However, adverse events in the retina may occur with the use of semaglutide.[14] Two primary hypotheses explore the potential effects of semaglutide on retinopathy: 1. Rapid Blood Sugar Control: One hypothesis posits that the quick and significant improvements in blood sugar levels brought about by semaglutide may temporarily exacerbate diabetic retinopathy, a phenomenon often referred to as "early worsening." This occurs when blood sugar levels are stabilized too rapidly, resulting in transient alterations within the retina. These changes can aggravate existing symptoms, although they typically stabilize and improve over time as the body adapts to the new blood sugar levels; 2. Cardiovascular Effects: The second hypothesis centers around the cardiovascular benefits associated with semaglutide. Although semaglutide is associated with cardiovascular improvements, the intrinsic microvascular changes associated with semaglutide effects may lead to fluid leakage. In addition to these factors, we can explore the potential for retinal damage from ischemia induced by semaglutide [15]. It is possible that the underlying pathophysiological mechanisms might parallel the retinal injuries seen following intense physical exertion [16-18]. This connection suggests that just as the body experiences stress during exhaustive exercise, leading to reduced blood flow and subsequent damage to the retina, similar processes could unfold in response to this medication (Figure 3).

Semaglutide has the potential to induce retinal detachment, which may occur through ischemic processes that mirror those seen in nonarteritic anterior ischemic optic neuropathy (NAION). Moreover, this drug might lead to heightened choroid hyperpermeability and other mechanisms that are not yet fully understood, contributing to the

complexity of its effects on ocular health. These hypotheses emphasize the dualistic effects of semaglutide on diabetic retinopathy, highlighting both the immediate challenges and the promising long-term benefits associated with its use. It will be important for the academic community to develop follow-ups with patients undergoing ophthalmological monitoring while they are using semaglutide to further clarify the relationship between the risks of retinal damage.

Figure 3. Possible pathophysiological mechanism of central serous chorioretinopathy associated with semaglutide.



4. Conclusion

Semaglutide is an important treatment tool for obesity. However, as with any medication, side effects are always possible. The academic community must monitor every new medicine and report any adverse events. For example, the alert to the risk of nonarteritic anterior ischemic optic neuropathy (NAION) in patient-prescribed semaglutide should be increased for other adverse events. Not only because of the description of this single clinical case, but because of other eye problems already reported, monitoring eyes closely for possible side effects could help to prevent damage from semaglutide.

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Research Ethics Committee Approval: Patient consent was obtained for the description and publication of this case report. This study was conducted in accordance with the Helsinki Declaration and was approved by the Ethics Committee of the São José do Rio Preto Medical School approved this report under the number CAAE: 86575425.5.0000.5415.

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Conflicts of Interest: None.

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