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Brachial Plexopathy Following Rapid Weight Loss from Tirzepatide: A Case Report

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Background: Weight reduction and metabolic changes have been associated with the development of compressive peripheral neuropathies.

Case Report: We present the case of a right-handed 60-year-old man who had a medical history of hypertension and hypercholesterolemia and developed neuromuscular symptoms after being treated with tirzepatide. During a 6-month course of tirzepatide use, the patient lost 60 pounds. However, the treatment was complicated by right-sided brachial plexopathy symptoms, including scapular pain, paresthesia, and progressive triceps weakness. Electromyography and nerve conduction studies confirmed denervation potentials at the right C5–C7 roots. Imaging studies were unremarkable. Symptoms persisted despite conservative interventions but improved gradually after the tirzepatide regimen was ended. At 2 months after the cessation, the patient's pain decreased from a 5/10 score to a score of one-2/10, and his strength improved. The follow-up EMG/NCV demonstrated the resolution of the denervation-caused changes.

Conclusion: This case highlights a possible association between the rapid weight loss caused by GLP-1 RAs and the development of brachial plexopathy. In this patient's case, the latter condition improved after his medication was discontinued.

Key words: Tirzepatide, brachial plexopathy, case report, neuropathy, weight loss

BACKGROUND

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are medications that lower serum glucose levels and promote weight loss. For this reason, GLP-1 Ras are commonly used to treat both type 2 diabetes mellitus and obesity by. These medications do the latter by slowing gastric emptying and increasing satiety (1). Tirzepatide is a newer agent in this class, using dual GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) receptor agonist activity (2). This dual incretin mechanism has been shown to demonstrate greater efficacy for glycemic control and weight reduction than do traditional GLP-1 RAs, prompting rapid increase in the use of tirzepatide since its FDA approval for type 2 diabetes and obesity in 2022 (3).

The most frequently FDA-listed adverse effects associated with GLP-1 RAs and GIPs are gastrointestinal, including nausea, diarrhea, and vomiting, followed by general systemic symptoms (fatigue, injection site) and immune reactions (hypersensitivity) (2). Although gastrointestinal adverse events predominate, recent studies have highlighted other emerging risks associated with the medications, such as acute pancreatitis, hypotension, and neurological adverse events (NAEs) (4). Reported NAEs of GLP-1 RAs are infrequent and mild, with the most commonly reported NAEs being dizziness and headache (2). A pharmacovigilance analysis of the FDA Adverse Event Reporting System (FAERS) from 2005-2024 found that 11.58% of all adverse events

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associated with GLP-1 RAs were neurological, with allodynia showing the highest reporting odds ratio (ROR 22.55, 95% CI: 21.71-30.08) (4).

Meanwhile, the brachial plexus is a complex of nerves composed of the C5-T1 nerve roots, which course through the anterior and middle scalene muscles before traveling beneath the clavicle and over the first rib. Following this, the brachial plexus enters the axilla, where it is surrounded by muscle and adipose tissue, which provides cushioning and protection from mechanical stress (5). Brachial plexopathy is a neurological condition in the ipsilateral upper extremity that results from damage to the brachial plexus, a network of nerves that innervate the shoulder, arm, and hand (5). Etiologies of this condition are classically divided into traumatic (e.g., traction or compression injuries) or nontraumatic (e.g., inflammatory, infectious, or radiation-induced) causes, leading to symptoms such as pain, weakness, sensory deficits, and functional impairment (6). To date, isolated drug-induced or weight-loss-associated brachial plexopathy has not been well described in the medical literature. Few, if any, reports have described neuropathy or denervation in association with the GLP-1 RA class. A recently published retrospective case-controlled study from 2005 to 2024 identified an increased likelihood that patients who used GLP-1 RAs would develop diabetic lumbosacral radioplexus neuropathy (DLRPN) and common fibular neuropathy (CFN), for reasons likely related to metabolic changes and weight loss (7).

Here, we report the case of a patient who developed brachial plexopathy with electromyography-confirmed denervation after undergoing tirzepatide therapy for weight loss. This case adds to the limited literature exploring neurological complications associated with GLP-1 RAs. The patient provided informed consent to be included in this case report.

CASE PRESENTATION

The patient is a 60-year-old right-handed man with a past medical history of hypertension and hypercholesterolemia/lipidemia. He was taking 100 mg of losartan and 40 mg of rosuvastatin daily. The patient's height was 5 feet 11 inches, and his weight was 225 pounds. Due to an elevated hemoglobin A1c of 6.2 and a fasting blood sugar of 130, the patient was started on a weekly regimen of 2.5 mg of tirzepatide by injection. Before starting tirzepatide, the patient had a waist circumference of 150 cm and a neck circumference of 49 cm at the Chassaignac tubercle.

Two weeks after the initiation of tirzepatide, the patient developed heartburn and thus began taking famotidine. Despite treatment, his symptoms did not improve sufficiently, and he developed epigastric abdominal pain. Within 4 weeks, he lost 20 pounds.

Due to the progression of epigastric pain, an H. pylori test was performed, which was positive. After the patient completed 2 weeks of quadruple therapy, his symptoms improved, and a repeat H. pylori test was negative. After 2 months on tirzepatide, the patient had lost 30 pounds. By the fourth month of therapy, his weight was 180 pounds, his waist circumference was 120 cm, and his neck circumference was 39 cm.

In the fifth month of therapy, the patient awoke one night with neck pain, right scapular pain, numbness, and tingling in the right arm. He began home exercises and used over-the-counter acetaminophen and ibuprofen. The following day, his symptoms worsened, and he developed weakness in the right triceps and shoulder movement.

The patient then presented to the emergency room, where a cervical spine MRI was performed and reported as unremarkable. He was prescribed a Medrol Dosepak (tapered dosing over 7 days), physical therapy, and 300 mg of gabapentin to be taken 3 times daily. His symptoms were only slightly responsive.

Over the next 2 weeks, while undergoing chiropractic care and acupuncture, the patient developed further weakness in his right triceps. The first electromyography and nerve conduction velocity (EMG/NCV) study of the patient demonstrated denervation potentials at right C5-C6, C7, and C8, with the greatest involvement at C5-6 and C7.

He was maintained on medications, transcutaneous electrical nerve stimulation (TENS), interferential current (IFC) therapy, a shoulder strap for posture maintenance, and home exercises. Despite this treatment, he continued to experience pain rated 5/10, which he described as a deep burning in the mid-lower back and right scapular region radiating into the right arm. Weakness persisted, particularly during overhead activities. He was unable to push more than 10 pounds above shoulder level with his right arm.

At the patient's sixth month on tirzepatide, his weight was 165 pounds, his waist circumference was 115 cm, and his neck circumference was 32 cm. At this point, his tirzepatide regimen was discontinued, and weight monitoring was continued.

Within 4 weeks of tirzepatide cessation, the patient's

symptoms gradually improved. Weakness lessened, and pain decreased from 5/10 to one-2/10 daily. Oral medications were tapered off, and numbness and weakness improved.

Two months after the discontinuation of tirzepatide, the patient's symptoms had improved dramatically. A repeat EMG/NCV study showed resolution of denervation potentials.

DISCUSSION

This case highlights a rare complication associated with rapid weight loss following treatment with tirzepatide. SInce GLP-1 RAs rise in prominence, their metabolic effects have been widely studied; however, emerging data suggest potential neuromuscular sequelae associated with the rapid weight loss caused by these medications (7). Like DLRPN and CFN, the pathophysiology likely results from the rapid loss of subcutaneous fat, which normally forms a cushion around peripheral nerves. The quick reduction of this fat thereby increases compression or traction forces. In this case, the decrease in the patient's neck circumference likely led to a marked reduction in adipose tissue and muscle around the brachial plexus, exposing these nerves to mechanical stress from ordinary movements such as posture (e.g., sleep) or movement (e.g., exercise). The loss of this mechanical cushioning may have made the plexus more vulnerable to compression or traction injuries, potentially leading to demyelination, axonal involvement, or ischemia. However, the neurological-symptom improvement following the discontinuation of the tirzepatide despite the substantial weight loss suggests that tirzepatide alone may have exerted physiological changes rather than weight loss alone.

The duration and rate of a given patient's weight loss have both been associated with the development of CFN. More specifically, weight loss that exceeds 11.023 pounds per month and proceeds consistently for several months is the strongest risk factor for developing CFN (8). However, both the mechanical changes and the metabolic changes induced by rapid weight loss have been implicated. For example, rapid weight loss can decrease a patient's quantities of vitamins B1, B6, B12, and E as well as folate, which are vital nutrients for healthy nerve function (9). Although tirzepatide does not have a defined, clear role in neurotoxicity, the medication's primary mechanism for weight loss through appetite/satiety control can lead to caloric restriction, further contributing to the risk of nutritional deficiency and subsequent metabolic neuropathies. Thus, discontinuation of tirzepatide in this case may have alleviated any metabolic stressors that allowed for neurological recovery without soft tissue cushioning. Other mechanistic data for this potential connection are still preliminary. GLP-1 receptors have been identified throughout the peripheral nervous system, including sensory/autonomic fibers, dorsal root ganglia, and Schwann cells (10). Preliminary studies have supported this neuroprotective effect in which GLP-1 RA signaling modulates several intracellular cascades that involve axonal regeneration, inflammatory modulation, and neurotrophic factors (11). As a result, this phenomenon raises the possibility that rapid changes in GLP-1 RA levels can lead to downstream effects that affect perineural processes or immune signaling.

If the patient continues to have persistent symptoms, other diagnoses, such as hereditary neuropathy with liability to pressure palsies (HNPP), hereditary neurologic amyotrophy (HNA), or Parsonage-Turner syndrome can be considered, since these conditions can be unmasked with rapid weight loss (10). However, only 11%-20% of HNPP patients present with brachial plexopathy, making said presentation unusual (11). Alternative etiologies across the spectrum of immune-mediated radiculoplexus neuropathies, such as PTS and diabetic amyotrophy, should be considered, especially in patients with diabetes (11). However, the patient did not exhibit the classic stepwise progression of neuropathic pain or the patchy/asymmetric pattern of PTS. Diabetic amyotrophy likewise presents with asymmetric pain and weakness but is typically located in the proximal lower extremities, accompanied by systemic symptoms, rapid weight loss, or changes in hemoglobin A1c (11). However, this patient's symptoms are limited to the upper extremities without any systemic symptoms. Notably, there was a report of diabetic amyotrophy symptom improvement after caudal epidural steroid injection, which raises the question of whether a cervical epidural steroid injection could provide an analogous benefit in the upper extremity (12). A subset of these radiculoplexus neuropathies may have microvasculitis or immune-mediated features and may be responsive to immunotherapy (13). Other causes, such as elevated inflammatory markers, prodromal viral illnesses, and radiation plexopathies, were absent in this case. These mechanisms highlight the complexity of differentiating among mechanical, metabolic, and immune-mediated etiologies.

Given the association, albeit rare, between rapid weight loss and the development of peripheral neuropathies, we predict that the prevalence of these neuropathies will see an increase in proportion to the rapid rise of GLP-1 RA usage. To the best of our knowledge, this case is the first one to detail a brachial plexopathy secondary to pharmacologically induced weight loss caused by tirzepatide. Clinicians prescribing GLP-1 RAs should encourage patients to lose weight gradually and counsel them on the risks of rapid weight loss and the potential neuropathic complications. Physiatrists and neurologists should consider patients' weight loss histories if consulted on or evaluating acute neuropathic symptoms, especially for patients who take a GLP-1 RA.

Notably, the causality between tirzepatide and brachial plexopathy cannot be established definitively in a single case report. Further pharmacovigilance and large double-blind randomized controlled trials are warranted to determine the incidence and potential mechanisms of neuropathies associated with GLP-1 RAs.

CONCLUSIONS

This case illustrates the need for the careful monitoring of patients on GLP-1 RAs such as tirzepatide, particularly in the context of rapid weight loss. While these agents demonstrate significant metabolic and anthropometric benefits, they may predispose susceptible individuals to neuromuscular complications, potentially through mechanical, metabolic, or drug-related phenomena. Clinicians should remain vigilant for newly onset neuropathic pain or weakness in patients undergoing therapy and consider discontinuation of GLP-1 RAs if symptoms progress.

Author Contributions:

JW and FE contributed to the conceptualization of this project. All authors contributed to writing and editing the manuscript and to the data analysis and figure preparation.

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