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Case Report

Myeloneuropathy after Uncontrolled Rapid Weight Loss: A Case Report

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Abstract

As a result of the escalating global epidemic of obesity, conservative and surgical interventions, and the incidence of secondary neurological complications involving the central and peripheral nervous systems, have increased. This case report presents a patient diagnosed with myelopathy and polyneuropathy after an uncontrolled rapid weight loss with a very low-calorie diet program. Our aim in presenting this case report is to identify the patients with acute polyneuropathy or myelopathy following rapid weight loss and to recognize the contributing factors.

Keywords: Obesity; Weight loss; Rapid; Myeloneuropathy; Polyneuropathy; Myelopathy

Introduction

Industrialization of the food system has paralleled obesity prevalence over the past several decades. According to World Health Organization (WHO) data from 2014, approximately 13% of the global population is obese, with over 1.9 billion adults aged 18 and up being overweight [1]. Obesity is frequently measured using Body Mass Index (BMI) values, with a BMI of 30 kg/m^2 or higher indicating obesity, a BMI of 40 kg/m^2 indicating morbid obesity, and a BMI between 25 and 29.9 kg/m^2 indicating overweight [2]. Obesity is linked to a higher prevalence of morbidity and a shorter life expectancy. To overcome the adverse effects of obesity on both health and body image, patients use many methods to lose weight, including lifestyle changes, dietitian follow-up, exercises, and psychotherapy. Significant weight loss with conservative approaches or bariatric surgeries is associated with obesity-related complications [3]. Neurological complications have been reported in 5%-16% of bariatric surgery patients. Among neurological complications, 54% to 62% are peripheral neuropathy, while 2% are myelopathy [4]. This case report presents a patient diagnosed with myelopathy and polyneuropathy after uncontrolled rapid weight loss with a very low-calorie diet.

Case Presentation

A 36-year-old woman was admitted to the neurology clinic for numbness and weakness in the lower extremities. The patient, who was learned to have a medical history of morbid obesity (BMI: 51.2 kg/m^2), had lost 45 kg in the last four months. She had started a self very low-calorie diet program four months ago without the supervision of any health authority. In the second month of her diet program, she complained of numbness in both lower limbs with tingling and dysesthesia, and when she entered the fourth month, she developed weakness in both lower extremities. She needed support to walk. No recent fever episodes or infection was noted. Upon admission, her examination showed normal mental functions, intact cranial nerves, muscle weakness (distal 3/5 proximal 4/5 in Medical Research Council (MRC) grade) in lower extremities with hyporeflexia, sensory loss in a stocking distribution, and impaired proprioception. The patient's imaging findings observed a contrast-enhancing hyperintense lesion between C3 and T1 on cervical Magnetic Resonance Imaging (MRI). A lumbar puncture showed normal pressure, with protein 42 mg/dl and glucose 46 mg/dl. Cell count and gram staining revealed no cells or bacteria. No oligoclonal bands were detected in the cerebrospinal fluid. Lumbar MRI findings were normal. Her serum folic acid level was 1.88 µg/ml (range 3.1-17.5), and her vitamin B12 level was 389 ng/L (range 191-663). The laboratory tests of the patient are summarized in Table 1. Contrast-enhanced brain and whole spinal MRIs were performed. While a few parenchymal millimetric hyperdense focus on T2-weighted images in brain MRI (Figure 1), focal mild hyperintensities in contrast-enhanced cervical vertebral MRI (Figure 2), and suspicious mild hyperintensities in contrast-enhanced thoracic vertebral MRI were observed (Figure 3). Contrast-enhanced lumbar vertebral MRI findings were normal. Electrodiagnostic studies revealed subacute severe distal symmetrical axonal sensorimotor polyneuropathy. EMG findings are summarized in Tables 2 and 3. The clinical picture of patient was suggestive of myelopathy with peripheral neuropathy. The patient, diagnosed with myeloneuropathy, was put on intravenous methylprednisolone 1 g daily for five days. Vitamin D and folic acid replacement was performed. In addition, combined vitamin B supplements were administered for other B group vitamins whose levels could not be measured but which were thought to be accompanied by deficiency. After her medical treatment was completed, she was admitted to our clinic for rehabilitation. On admission, a neurological examination of her muscle strength was evaluated bilateral hip flexors were 4/5, knee extensors 4/5, and ankle dorsiflexors 3/5 in both lower extremities as MRC grade. There was no motor deficit in the upper extremities. Deep tendon reflexes were decreased in the lower extremities and no pathologic reflexes were noted. Fine touch sensation was decreased in a stocking distribution to the level of her lower extremities. Positional and vibratory sensations were found to be impaired. The patient was

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Table 1: Laboratory test results.

Parameters	Results	Reference values
Hgb (g/dL)	9.3	11.7-15.5
PLT (10^3/µL)	470	150-450
WBC (10^3/µL)	11.08	3.57-11.01
HCT(%)	31.2	34.35
MCV (fL)	85.5	73.54
ESR (mm/h)	16	0-20
CRP (mg/L)	8.08	0-9
Serum iron (µg/L)	336	330-1930
TIBC (µg/dl)	335	125-345
Ferritin (µg/L)	19.5.5	15-150
HbA1c (%)	5.4	45081
Magnesium (mg/dl)	2.33	1.6-2.6
Ca (mg/dl)*	8.47	8.6-10.2
BUN (mg/dl)	21.9	16-43
Crea (mg/dl)	0.45	0.5-0.9
AST (U/L)	51.3	0-32
ALT (U/L)	182	0-33
ALP (U/L)	125	40-150
CPK (U/L)	22	10-171
Total protein (g/dL)	5.43	6.4-8.3
Albumin (g/dL)	3.44	3.5-5.0
Prealbumin (mg/dL)	14.6	20-40
25(OH)Vit-D3 (ng/mL)	<3.0	>30
PTH (pg/mL)	17.9	32478
Vit-B12 (ng/mL)	389	126-505
TSH (uIU/mL)	2.1	0.27-4.2
Folic acid (ng/mL)	1.88	3.1-17.5

Hgb: Hemoglobin; PLT: Platelet; WBC: Whight Blood Cell; HCT: Hematocrit; MVC: Mean Corpuscular Volume; ESR: Erythrocyte Sedimentation Rate; CRP: C-Reactive Protein; TIBC: Total Iron Binding Capacity; Crea: Creatinin; BUN: Blood Ure Nitrogen; Ca: Calcium; AST: Aspartat Transaminase; ALT: Alanin Transaminase; ALP: Alkalin Phosphotase; CPK: Creatine Phosphokinase; Vit: Vitamin; PTH: Parathormone; TSH: Thyroid Stimulating Hormone; *Corrected serum calcium

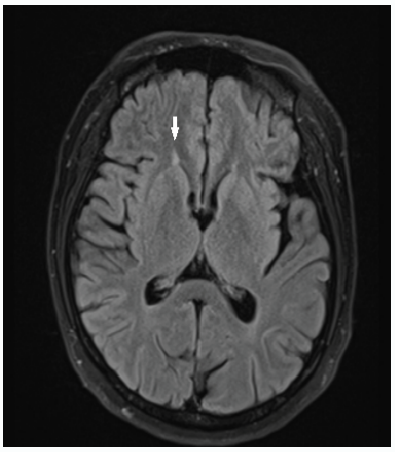

Figure 1: Brain MR imaging shows parenchymal millimetric hyperintense lesion (arrow), in fluid-attenuated inversion recovery (FLAIR) images. MR: Magnetic Resonance.

ambulating with a wheelchair. Pregabalin 75 mg twice a day was started for neuropathic pain. The rehabilitation program arranged galvanic current and electrical stimulation for the lower extremities followed by range of motion, stretching and strengthening exercises, ambulation training, and balance/coordination exercises. In the follow-up, it was observed that there was an improvement in positional and vibratory sensation and progress in muscle power, balance, ambulation, and daily life activity. After a six-week rehabilitation program, she began to ambulate with a pair of walking canes.

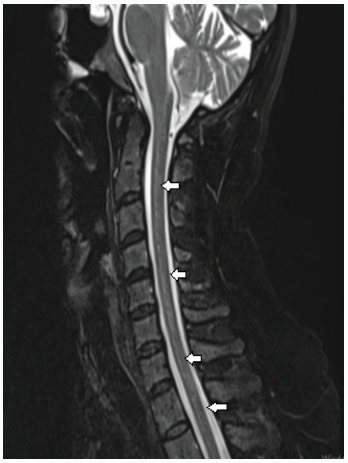

Figure 2: Contrast-enhanced sagittal T2-weighted cervical vertebral MR imaging shows focal mild hyperintensities in cervical spinal cord (white arrows). MR: Magnetic Resonance.

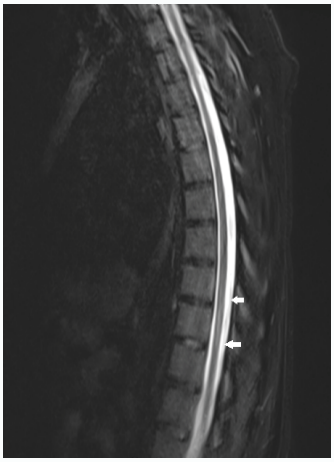

Figure 3: Contrast-enhanced sagittal T2-weighted thoracic vertebral MR imaging shows focal mild hyperintensities in thoracic spinal cord (white arrows). MR: Magnetic Resonance

Discussion

Various modalities can achieve weight loss, including conservative modalities or bariatric surgeries. Dietary interventions may result in significant weight loss, but the greatest obstacle is the seemingly inevitable weight gain in the following years [5]. Due to the limited efficacy of available conservative treatments, bariatric surgical procedures have been utilized more frequently [6]. Numerous neurological complications of bariatric surgical procedures are well-described in the literature including chronic or subacute symmetrical polyneuropathy, acute severe polyneuropathy, burning feet syndrome, myotonic syndrome, posterolateral myelopathy, and Wernicke-Korsakoff Syndrome (WKS) [7,8]. While reviewing the literature, it was noticed that most of the reported neurological complications developed in morbidly obese patients after bariatric surgical procedures [3,8-11]. Apart from bariatric surgery, a few patients developed WKS due to various diseases such as anorexia nervosa and inflammatory bowel diseases [12]. Neurological complications that develop after rapid weight loss due to a low-calorie diet have not been found in the literature. Therefore, to our knowledge, this case is a first in the literature.

Table 2: Nerve conduction study.

Nerve stimulation	Velocity (m/s)	Latency (ms)	Amplitude (mV)
MOTOR			
Right Median Nerve (APB)			
Wrist		2.86	8.2
Elbow	50	7.45	7.6
Right Ulnar Nerve (ADM)			
Wrist		2.08	7.9
Below elbow	59	5.83	8.4
Above elbow	54	7.5	8.6
Right Peroneal Nerve (EDB)			
Ankle		3.54	0.5
Fibulae head	39	12.29	0.3
Knee	39	14.58	0.3
Left Peroneal Nerve (EDB)			
Ankle		3.59	0.6
Fibulae head	39	13.39	0.4
Knee	40	15.42	0.4
Right Tibial Nerve (AH)			
Ankle		5.16	1.1
Popliteal fossa	40	15.47	0.8
Left tibial nerve (AH)			
Ankle		5.05	1.1
Popliteal fossa	40	15.16	1.1
SENSORY			
Right median nerve (Digit II)	40.4	2.97	20.3
Right ulnar nerve (Digit V)	44.9	2.45	34
Right sural nerve	No Response		
Left sural nerve	No Response		

APB: Abductor Pollicis Brevis; ADM: Adductor Digiti Minimi; EDB: Extensor Digitorum Brevis; AH: Abductor Hallucis

Polyneuropathies are the most typical peripheral nervous system disorders, with a prevalence rate of 5% to 8%. There are also polyradiculoneuropathies with additional proximal involvement and asymmetric forms such as mononeuritis multiplex. Based on the type of nerve fiber involved, the primary focus may be on the sensory, motor, or autonomic symptoms. Time course is an important parameter, ranging from acute to subacute to chronic. Cerebrospinal fluid analysis is required in the case of a suspected inflammatory etiology. Clinical examination of the type of distribution and severity, electrophysiological tests (axonal, demyelinating), and laboratory tests for diabetes mellitus, vitamin deficiency, alcohol abuse, and autoantibodies can identify the affected systems. Diabetes mellitus is the most common cause of polyneuropathy worldwide. Chemotherapy, alcoholism, autoimmune diseases, bariatric surgeries, anorexia nervosa, vitamin deficiencies genetic mutations are other causes [13]. Peripheral neuropathy is the most common neurological complication, affecting up to 16% of bariatric surgical patients [7]. Nutritional deficiencies may play a crucial role in the pathogenesis of disease. Folate deficiency has been associated with painful peripheral polyneuropathies. Copper deficiency has been linked to painful peripheral neuropathy as well as radiculoplexopathy-associated myelopathy [2]. Myelopathy is another potentially disabling neurological complication associated with bariatric surgery. Myelopathy and peripheral neuropathy may coexist, resulting in a condition known as myeloneuropathy [3]. Juhasz-Pocsine et al. [14] observed that myelopathy has been reported in patients with low serum B12 or copper levels. A deficiency in vitamin E (tocopherol) has also been linked to treatable myelopathy. According to Koffman et al. [4], pyridoxine deficiency is another cause of myelopathy. Despite their obesity, obese individuals may have subclinical or overt nutritional deficiencies. Massive and rapid weight loss may also be associated with deficiencies in essential vitamins and minerals [3]. Our patient lost 45 kg in a few months with a very low-calorie diet program, just like patients who underwent bariatric surgery. In this case, estimated risk factors associated with myeloneuropathy are as follows: rapid weight loss, low serum folate, calcium, and vitamin D, low serum albumin and protein, inadequate vitamins and micronutrients supplementation, B1, B6, vitamin E, and copper levels could not be measured because it is not available in our laboratory. However, multivitamin replacement, including these vitamins, was performed considering the possible deficiencies [15].

The treatment of vitamin and micronutrient deficiencies frequently results in an amelioration of clinical symptoms and clinical signs. Approximately 50% of patients with polyneuropathy suffer from pain. Gabapentin, pregabalin, duloxetine, and tricyclic antidepressants are the first choice of pharmacological treatments for neuropathic pain. We used pregabalin for neuropathic pain in this patient. In neuropathies, physiotherapy is guided by symptoms and functional deficits. It includes exercises that enhance balance, coordination, and proprioception to improve standing and walking stability. In addition, it is instrumental in regaining muscle strength when accompanied by muscle weakness [13]. Our patient progressed in the area of muscle strength, balance, and ambulation with physiotherapy.

Differential diagnosis includes Wernicke's Korsakoff syndrome (WKS), neuropsychiatric disorders, CNS demyelination, and Guillain-Barre syndrome. WKS are the most widely recognized neurological complication of thiamine (vitamin B1) deficiency. Confusion, ataxia and nystagmus, amnesia, confabulation, apathy, and lack of insight are among the symptoms that can be seen [3]. Our patient had no of these symptoms. Brain MRI scenes also do not support the WKS.

Table 3: Needle electroneuromyography findings.

	Spontaneous					MUAP			Recruitment
Muscle	IA	Fib	PSW	Fasc	H.F	Amp.	Dur.	PPP	Pattern
Right Tibialis Anterior	N	3+	3+	None	None	N	N	1+	N
Right Gast. (Medial head)	N	3+	3+	None	None	N	N	1+	Reduced
Left Tibialis Anterior	N	3+	3+	None	None	N	N	1+	N
Left Gast. (Medial head)	N	2+	2+	None	None	1+	1+	1+	Reduced
Right Vastus Medialis	N	3+	3+	None	None	1+	1+	1+	Reduced
Left Vastus Medialis	N	3+	3+	None	None	N	N	N	N
Right Gluteus Medius	N	None	None	None	None	N	N	N	N
Left Gluteus Medius	N	None	None	None	None	N	N	N	N
Right Abductor Pollicis Brevis	N	None	None	None	None	N	N	N	N
Right Deltoid	N	None	None	None	None	N	N	N	N
Left Cervical paraspinals	N	None	None	None	None	N	N	N	N

MUAP: Motor Unity Action Potencial; İA: İnitial Activity; Fib: Fibrilation; PSW: Positive Sharp Wave; Fasc: Fasciculation; HF: High Frequency; Amp: Amplitude; Dur: Duration; PPP: Poliphasia; Gast.: Gastrocnemius; N: Normal

Neuropsychiatric disorders are common in obese people, including eating disorders such as anorexia nervosa and bulimia. Addictive behaviors like gambling, compulsive shopping, and mood disorders like depression and acute mania. The patient's depressive mood was observed during her hospitalization, and she requested a psychiatry consultation in this case report. Some patients reported CNS resembling Multiple Sclerosis (MS) after bariatric surgery [11]. Our patient did not have clinical and imaging findings compatible with MS. Guillain Barre syndrome was also excluded from our patient with clinical, electrophysiological, and cerebrospinal fluid findings.

Result

Nutritional deficiencies that develop after rapid, uncontrolled weight loss cause many neurological problems, such as polyneuropathies and myelopathies. With this case report, we aimed to increase awareness of potential neurological findings following rapid weight loss with self-low-calorie diet programs. To avoid many complications, patients should undergo a nutritional and psychological evaluation before any weight reduction treatment, including both conservative and surgical procedures.

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