ICE CREAM ONLY DIET: PRESENTING WITH SPASTIC PARALYSIS

Yong Mi Shin, DO[1], Kathryn Flynn, OMS-IV[2], Mary Clare Parrott, DO[1]
1. Department of Internal Medicine, Jefferson Health Northeast, Philadelphia, PA
2. Department of Internal Medicine, Philadelphia College of Osteopathic Medicine, Philadelphia, PA

Introduction

Subacute combined degeneration (SCD) is demyelination of the dorsal spinal cord that presents with debilitating neurological symptoms involving spasticity and paresthesia of extremities. It most commonly presents with Vitamin B12 deficiency, which exhibits macrocytic, megaloblastic anemia commonly caused by nutritional deficiency, or pernicious anemia. This is a case of a 71-year-old male who ate only ice cream for six months due to symptoms of dysgeusia. Patient was found with severe Vitamin B12 (cobalamin) deficiency and radiographic evidence of SCD that caused severe spasticity and progressing paresthesia of his extremities.

Case Report

A 71-year-old Caucasian male presented with progressive paresthesia and spasticity of his upper and lower extremities that kept him mostly bed bound in the last 2-3 months. Due to symptoms of dysgeusia, patient reported consuming mainly ice cream for 6 months.

Neurological exam was significant for spasticity in all extremities with limited motor and muscle strength exam due to discomfort. Patient also had extension contracture of his bilateral wrists and flexion contracture of bilateral knees. He had no cranial nerve deficits and grossly intact sensation, but there was inability to elicit reflexes.

Labwork was notable for pancytopenia with marked macrocytic anemia (Hgb of 6.9 g/uL, MCV 109.9 fL), mild leukopenia and mild thrombocytopenia. Cobalamin was profoundly low at 60 pg/mL (reference >150 pg/mL) while folic acid was elevated. Anti-parietal cell antibodies were negative, but positive for anti-intrinsic factor antibody suggesting pernicious anemia. MRI of brain & spine showed signs of demyelination, consistent with diagnosis of SCD (Fig 1).

Patient was given blood transfusion and was started on daily cyanocobalamin 1g IM injections and oral multivitamin replacement. Patient started to regain his functions and mobility during his 10 day hospitalization on daily replacement therapy. 1 month after patient transitioned to weekly IM injection upon discharge, pancytopenia fully resolved and patient was able to walk with a cane with minimal spasticity of his lower extremities. At 7 months, follow up MRI of brain & cervical spine showed decreased T2 weighted signal within the dorsal column (Fig 2) which reflected his clinical improvement.

Magnetic Resonance Imaging

Figure 1. MRI Brain and Cervical spine without contrast showing sagittal T2 hyperintense signal in the T2 weighted image (Fig 1a) of the dorsal column without a signal noted in T1 weighted image (Fig 1b).

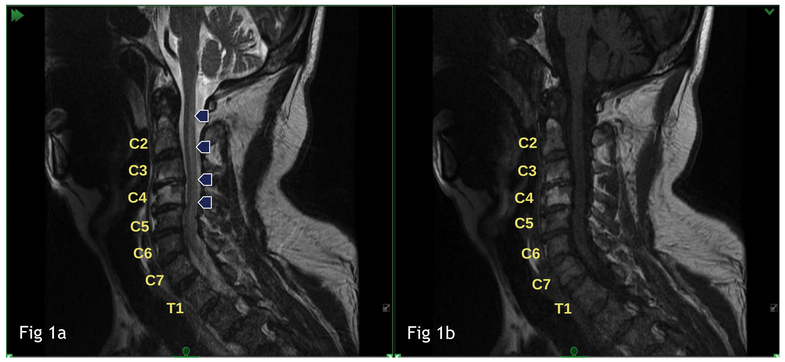
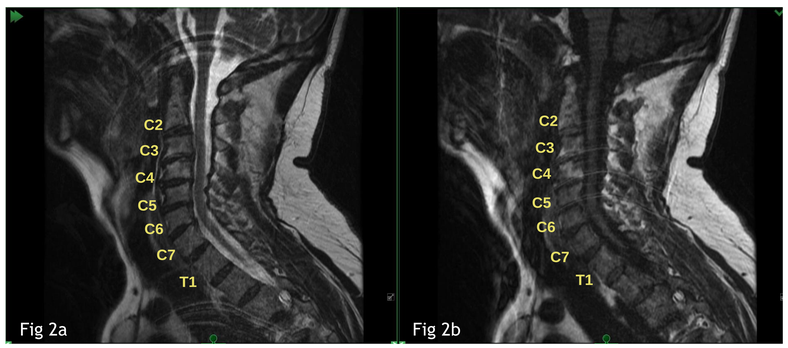


Figure 2. MRI Brain and Cervical spine without contrast showing decreased sagittal T2 hyperintense signal in comparison to Figure 1a after 7 months of oral cobalamin supplement daily and weekly cobalamin IM therapy.



Discussion

Cobalamin is an essential element for many biochemical processes in our body. Its deficiency in chronic state can lead to demyelination and axonal degeneration of our spinal cord, or subacute combined degeneration. In this case, our patient's malnutrition state from his ice cream diet contributed to his cobalamin deficiency. However, with such profoundly low cobalamin level, pernicious anemia could not be ruled out.

There has been growing prevalence of positive antibodies against anti-intrinsic factor or anti-parietal cell factor in the elderly population. Late age discovery can suggest acquired autoimmune process from chronic inflammatory destruction against gastric mucosa that impacts proper function of intrinsic factor, which usually facilitates cobalamin absorption in our GI system. One particular cause, atrophic gastritis, is associated with other elemental deficiencies and requires close monitoring for its known gastric adenocarcinoma risk.

Our patient's debilitating neurological symptoms were consistent with his MRI findings of spinal cord demyelination. Aggressive cobalamin replacement therapy caused reversal of his neurologic dysfunction and improvement in his subsequent MRI, which further confirmed our diagnosis of SCD.

Conclusion

A 71-year-old male presented with marked macrocytic anemia and debilitating neurological symptoms suggesting severe cobalamin deficiency. Patient was found to have pernicious anemia with positive anti-intrinsic factor antibody and MRI images confirming the diagnosis of SCD. Early diagnosis and replacement therapy can greatly improve and prevent hematological and neurological dysfunction in the more highly prevalent elderly population.

References

1. Zhang et al, 2018, Three-point sign in subacute combined degeneration of the spinal cord, Medicine, 97(31):e11620
2. Andrès et al, 2020, The Conditions and Diseases that Cause Vitamin B12 Deficiency in Elderly Patients: from Metabolism to Diseases. Quart J Geriat Med, 01(01):8–16.
3. Zik, 2019, Late Life Vitamin B12 Deficiency, Clin Geriatr Med, 35(3):319–325.
4. Kulnigg-Dabsch, 2016, Autoimmune gastritis, Wien Med Wochenschr, 166(13):424–430.
5. Wong, C.W. (2017). Vitamin B12 Deficiency in the Elderly. p159-166 . Watson, R.R, Nutrition and Functional Foods for Healthy Aging, Cambridge, MA, Academic Press.