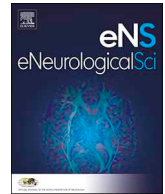




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Letter to the Editor

## Reversible neurogenic dysphagia: A rare presentation of vitamin B<sub>12</sub> deficiency



Dear Editor,

Pernicious anemia is an autoimmune disorder that refers to vitamin B<sub>12</sub> deficiency caused by autoantibodies that target intrinsic factor and/or gastric parietal cells leading to impaired absorption of vitamin B<sub>12</sub> [1]. The clinical spectrum of vitamin B<sub>12</sub> deficiency may range from an asymptomatic state to demyelination of white matter in the brain and spinal column, which can present as dementia, peripheral neuropathy, ataxia, and rarely, bulbar dysfunction [2]. Dysphagia is an uncommon manifestation of vitamin B<sub>12</sub> deficiency that is potentially reversible if diagnosed and treated within the first six months.

### 1. Case report

A 66-year-old male presented to the hospital with dyspnea and productive cough. Review of systems disclosed dysphagia to solids and an unintentional 30-pound weight loss over the past year. In addition, family members described 18 months of cognitive decline, decreased concentration and difficulty with executive functioning.

On exam, he was tachycardic, hypoxic, cachectic, and anxious. There were bibasilar rales. There was poor recall, lack of orientation to time, visual hallucinations, and disinhibition. Neurological exam revealed normal gait, normal deep tendon reflexes, and no sensory deficits. No deficits in vibratory sensation or proprioception were noted. The Montreal Cognitive Assessment (MoCA) score was 13/30 and Saint Louis University Mental Status (SLUMS) exam score was 15/50, indicating dementia. The white blood cell count was normal and there was mild macrocytic anemia (Hgb 12.4, MCV 99). Chest x-ray demonstrated multiple infiltrates and *Strep pneumoniae* urine antigen was positive.

The rapid cognitive decline, solids dysphagia, and weight loss prompted an evaluation for reversible causes of dementia. The serum B<sub>12</sub> level was 63 pg/mL (normal > 200 pg/mL), serum homocysteine 50.7 μmol/L (normal < 15 μmol/L), and methylmalonic acid (MMA) 14,908 μg/dL (normal < 4.7 μg/dL). A barium swallow demonstrated esophageal and pharyngeal dysmotility with delayed esophageal emptying and micro-aspiration. An esophagogastroduodenoscopy revealed atrophic gastritis of the gastric fundus and body (Fig. 1). Anti-intrinsic factor antibodies were positive. The presumptive diagnosis was aspiration pneumonia caused by neurogenic dysphagia as a sequela of severe vitamin B<sub>12</sub> deficiency resulting from pernicious anemia. He received intramuscular B<sub>12</sub> with significant improvement in both anemia and dysphagia, with no further hospitalizations for aspiration over one year of follow-up.

### 2. Discussion

The neurologic clinical sequelae of vitamin B<sub>12</sub> deficiency include dementia, deficits in the posterior and lateral columns of the spinal

cord, peripheral neuropathy, psychosis, ataxia, and, less commonly, bulbar symptoms like dysphagia [2]. Neurogenic dysphagia from B<sub>12</sub> deficiency has only rarely been reported [1].

In normal physiology, vitamin B<sub>12</sub> is involved in DNA synthesis, erythropoiesis, and maintenance of the myelin sheath [2]. Deficiency of vitamin B<sub>12</sub> typically develops due to malabsorption rather than dietary deficiency, and pernicious anemia is by far the most common cause across all populations, including elderly patients [2–4]. Neurologic symptoms manifest late in the course of B<sub>12</sub> deficiency and usually occur after the development of anemia [5]. Patients with functional deficiency of cobalamin and low-normal serum levels can be identified by elevated serum homocysteine and MMA levels [1]. However, only about one-third of patients with neuropsychiatric symptoms of B<sub>12</sub> deficiency have hematological abnormalities [6]. Therefore, clinicians must not rely on abnormal red blood cell indices as an initial screen for B<sub>12</sub> deficiency.

Neurologic lesions associated with B<sub>12</sub> deficiency are progressive, ranging from demyelination to axonal degeneration ultimately leading to neuronal death, and are not always reversible with B<sub>12</sub> supplementation [2]. Studies in human and animal models have shown that cobalamin deficiency perturbs cytokine expression, which is believed to play a role in the pathogenesis of white matter myelinolytic lesions [7]. Elevated levels of tumor necrosis factor-α and decreased levels of epidermal growth factor (EGF) have been isolated from the cerebrospinal fluid of patients with vitamin B<sub>12</sub> deficiency [7]. Neurological deficits can reverse, especially if treatment is initiated within 6 months of initial presentation, and improvement in neurologic symptoms can be expected within one week to three months after supplementation is initiated [2,5,8]. Prompt supplementation with vitamin B<sub>12</sub> has been reported to reverse neurological symptoms, however, the severity and duration of symptoms before treatment influence the degree of recovery [5]. Following three months of B<sub>12</sub> supplementation, there was complete reversal of neurogenic dysphagia in our patient. However, neurological deficits present for greater than one year are often associated with either no improvement or only partial relief of symptoms [5]. In our patient, who presented with 18 months of cognitive decline, one year follow-up showed no cognitive improvement despite continued B<sub>12</sub> supplementation. Therefore, early recognition of a compatible clinical syndrome, thorough testing, and intramuscular treatment for vitamin B<sub>12</sub> deficiency is critical to preserve and restore neurological function.

### Conflict of interest

None.

### Funding

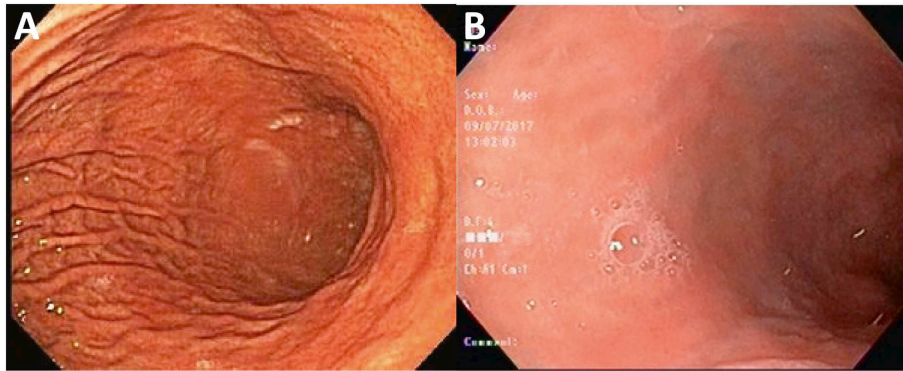
None.

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**Fig. 1.** Atrophic gastric mucosa from pernicious anemia.  
 A) Example of normal gastric mucosa on EGD.  
 B) Atrophic gastric mucosa on EGD from this patient.

### Authorship

All authors had access to the data and played a role in writing this manuscript.

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