Psychosomatics 2014:55:179–185 Published by Elsevier Inc. on behalf of The Academy of Psychosomatic Medicine.

Case Reports

The Neuropsychiatry of Scurvy

Amanda Daschbach Wright, M.D., Elizabeth Stevens, D.O., Maysun Ali, D.O., David W. Carroll, D.N.P., Thomas M. Brown, M.D.

Introduction

Scurvy is an ancient, persistent disease. Since the 1500s, when transoceanic travel created predictable opportunities to watch scurvy evolve, clinicians have understandably focused on the bleeding and intense joint pain that often preceded death. But scurvy affects more than just collagen. Lassitude, psychomotor retardation, and irritability have for centuries been recognized as sentinel expressions of this vitamin deficiency. Lind[1] described the earliest stage of scurvy as characterized primarily by a "lazy, inactive disposition" and an ill "countenance." A century later, Shapter[2] reported that the onset of scurvy was heralded by "feelings of weakness," of listlessness and "a disinclination to exertion" and that "the patient is nervous." Fouron and Chicoine found irritability to be the only universal finding in 38 patients with scurvy and that more patients had psychomotor retardation than had abnormal bleeding. Other symptoms can include weight loss, myalgias and arthralgias, "corkscrew" or coiled hairs, swollen, purple gums, and friable nails with periungual bleeding. As the condition worsens, teeth can become loose and avulsed, wounds heal poorly, and spontaneous hemorrhages and edema of the lower extremities can occur, leading to femoral neuropathy with joint hemorrhages and effusions.[3] More recently, extrapyramidal symptoms due to scurvy have been reported. But despite their early and frequent appearance, the central signs and symptoms of scurvy are sometimes overlooked. The authors present a case of scurvy in which the neuropsychiatric symptoms, including parkinsonism, loomed large both in suggesting the diagnosis and in tracking recovery.

Case

Mr. M, was a 50-year-old Hispanic man with a history of chronic paranoid schizophrenia, hepatitis C, alcohol dependence, and tobacco dependence, presented with weakness, gait instability, and somnolence. Mr. M's mother stated that in the 2 days leading to his presentation, he seemed very thirsty and drank nine to ten 16-ounce bottles of water and at least six 12-ounce beers, per day. Previously he was able to walk without assistance and per his mother's report had been stable on 37.5 mg of fluphenazine decanoate every 2 weeks for management of psychiatric symptoms. His last injection was 17 days before his critical presentation for weakness.

Mr. M's calculated admission serum sodium level was 92–94 mmol/L (calculated using serum osmolality and osmolar gap), with a normal range of 133–145 mmol/L. Over the preceding 4 years, his serum sodium had ranged from 129 to 134 mmol/L. Serum osmolality on admission was low at 221 mOsm/L (normal range: 278–305 mOsm/L). Urine osmolality was normal at 538 mOsm/kg (normal range: 50–1200 mOsm/kg). Urine sodium and chloride levels were below the lower limit of normal. He also had a community-acquired pneumonia. He was admitted to the medical intensive care unit and sodium

Received August 23, 2013; revised October 7, 2013; accepted October 8, 2013. From University of Texas Health Science Center at San Antonio, San Antonio, TX; Audie L. Murphy Memorial Veterans Administration Center, San Antonio, TX. Send correspondence and reprint requests to Thomas M. Brown, M.D., Audie L. Murphy Memorial Veterans Administration Center, 7400 Merton Minter Boulevard, San Antonio, TX 78229; e-mail: thomas.brown5@va.gov, drthomasmbrown@yahoo.com

Published by Elsevier Inc. on behalf of The Academy of Psychosomatic Medicine.

replacement was started using standard replacement protocols. A CT study of the head that was performed on admission had normal findings. An MRI of the brain, with and without contrast, on hospital day (HD) 9 was also read as normal. On HD10, Mr. M was transferred from the medical intensive care unit to the medical floor. His sodium was now normal at 133 mmol/L. The etiology of his hyponatremia was thought to be multifactorial with contributions from alcoholism, polydipsia, and medication side effects, including a possible fluphenazine-induced syndrome of inappropriate antidiuretic hormone release.

The department of psychiatry was consulted on HD10 to assist with medication recommendations given his psychiatric history and treatment with fluphenazine decanoate. A review of his record revealed that he had parkinsonism and abnormal tongue movements documented since 2004. He had consistently declined treatment for these and was maintained on injectable antipsychotics because of compliance issues and suboptimal treatment while on atypical antipsychotics. On initial psychiatric examination, He had parkinsonism with resting tremor, flat affect, hypophonia, and markedly reduced psychomotor activity. He also had flaccidity, clonus, and a grasp reflex. He had no abnormal nystagmus or other evidence of dystaxia. He had no palatal, gum, or periungual bleeding. He had no corkscrew hairs. He denied joint pain. Although Mr. M had a history of auditory hallucinations and ideas of reference with apathy and social withdrawal, his current mental status examination revealed the belief that his parents were impostors and that he was completely well.

Given his history of reduced oral intake, alcohol dependence, a 2-pack/day history of smoking for many years, and a recent catabolic state requiring treatment with furosemide, nutritional factors contributing to his weakness were considered. Concern about his metabolic state and the possibility of a refeeding syndrome, given his alcohol use, led to a cautious introduction of enteral feeds on HD2 with vitamin supplementation. A serum alcohol level was not obtained. A urine drug screen obtained on admission had negative results. Liver function tests repeatedly showed normal findings. The vitamin C level was tested on HD10. On HD17, Mr. M's vitamin C test returned showing a level of 0.4 mg/dL, which was at the lower limit of the reference range (reference range: 0.4–2.0 mg/dL). Serum levels within the reference range were obtained for zinc (98 mg/mL; reference range: 56–134), selenium (233 mg/mL; reference range: 100–340), and vitamin E (6.9 mg/mL; reference range: 4.6–17.8). Serum iron levels on HD10 were within the reference range at 53.0 mg/dL (reference range: 45–182), as was an iron saturation at 26.4% (reference range: 20%–55%). Ferritin was measured on HD41 and was within the range at 205 ng/mL (reference range: 10–322). In 2007, serum ceruloplasmin level was within the range at 35 mg/dL (reference range: 18–36). Serum manganese level was not tested, but Mr. M had neither occupational exposure nor parenteral nutrition to suggest this might be a risk. To assess the degree to which his psychomotor retardation was due to parkinsonism, a single dose of 25 mg of diphenhydramine was given intravenously. There was a distinct, though modest, improvement with resolution of both hypophonia and resting tremor but no increase in overall psychomotor activity.

Given his multiple risk factors for vitamin C deficiency, he was given 200 mg of vitamin C with 4 mg of zinc intravenously daily. Following 2 days of treatment, his parkinsonism completely resolved with improved phonation and facial expression, no resting tremor or cogwheeling and no oral dyskinesias were observed (Abnormal Involuntary Movement Scale testing score 0 on HD19). His energy level also markedly improved. Now able to participate effectively in an interview, he reported ongoing psychotic symptoms and an oral antipsychotic was resumed. The levels of zinc, selenium, vitamin E, and a retest for vitamin C were all within the reference range. On HD24, Mr. M remained on a schedule of oral antipsychotic and both psychosis and extrapyramidal symptoms (EPS) were absent.

Discussion

Prevalence of and Risk Factors for Hypovitaminosis C

Scurvy is often regarded as rare, yet its precursor, hypovitaminosis C, is not. In a study of micronutrient consumption among European adults, Roman Viñas et al.[4] reported that 10% to 20% had deficient intake of vitamin C and 11% had deficient intake of zinc. Certain populations are at higher risk for abnormally low serum vitamin C levels. Smokers, for example, have roughly double the daily vitamin C requirement