

Subacute combined degeneration due to vitamin B12 deficiency in a chronic alcoholic presenting as paraparesis

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ABSTRACT

Chronic alcohol abuse can damage the central and peripheral nervous systems through direct neurotoxicity and by causing nutritional deficiencies. Vitamin B12 (cobalamin) and thiamine deficiencies are well recognised in people with alcohol use disorder and may result in reversible myelopathy or polyneuropathy. We report a 42-year-old man with long-standing heavy alcohol consumption who presented with progressive numbness and weakness of the lower limbs. Examination revealed symmetric lower-limb weakness, sensory loss and areflexia without cranial-nerve or upper-limb involvement. Laboratory tests showed macrocytosis, mildly elevated transaminases and gamma-glutamyl transpeptidase, but normal serum creatinine and electrolytes. Ultrasonography revealed hepatomegaly with grade I fatty change. Magnetic resonance imaging (MRI) of the brain was unremarkable, whereas sagittal T2-weighted images of the spine demonstrated longitudinally extensive hyperintense signal abnormalities in the posterior columns without cord expansion. The clinical presentation and imaging findings were consistent with subacute combined degeneration of the spinal cord due to vitamin B12 deficiency in the setting of chronic alcoholism. The patient improved with parenteral vitamin B12 and thiamine replacement along with gabapentin for neuropathic pain. This case highlights the importance of recognising nutritional myelopathy in chronic alcoholics because early diagnosis and vitamin replacement can prevent permanent neurological deficits.

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INTRODUCTION

Excessive alcohol consumption can injure multiple organ systems. Neurological complications include Wernicke–Korsakoff syndrome, cerebellar degeneration, peripheral neuropathy and myelopathy. Alcoholic neuropathy, one of the most common sequelae of alcohol misuse, typically presents with distal paraesthesias, burning pain and gait ataxia. The pathophysiology of alcoholic neuropathy is multifactorial; direct toxicity of ethanol and its metabolites causes axonal degeneration and demyelination, while nutritional deficiencies—particularly of B-group vitamins—impair neuronal energy metabolism. Chronic alcohol use also predisposes to vitamin B12 (cobalamin) deficiency by reducing dietary intake, impairing absorption and increasing urinary losses. Vitamin B12 deficiency can cause subacute combined degeneration (SCD) of the spinal cord, a potentially reversible demyelination of the posterior and lateral columns that manifests as progressive sensory and motor deficits in the lower limbs. MRI of the spinal cord in SCD typically shows longitudinally extensive T2-hyperintense lesions confined to the dorsal columns with an “inverted V” appearance on axial images.

Hepatic dysfunction and portosystemic shunting in advanced liver disease may cause hepatic myelopathy, characterised by spastic paraparesis with minimal sensory loss. Distinguishing hepatic myelopathy from nutritional

myelopathy is crucial because the latter is reversible with vitamin replacement. We describe a chronic alcoholic who developed progressive paraparesis due to vitamin B12 deficiency. The case emphasises the need for early recognition of nutritional myelopathy in general medical practice and reviews the relevant literature.

CASE PRESENTATION

History and examination

A 42-year-old man from south India presented with progressive weakness and numbness in both lower limbs. He was previously well until six months prior to admission, when he noted paraesthesias in the left foot that gradually progressed to involve both legs and thighs. Over the subsequent weeks he developed difficulty rising from a squatting position and an unsteady gait. One week before admission he experienced aching pain and stiffness in both lower limbs and required support to walk. There was no history of trauma, back pain, bowel or bladder dysfunction, fever, weight loss, unsteadiness of the upper limbs or visual symptoms. He complained of a sore throat for ten days.

The patient drank approximately 360 mL of spirits daily for 17 years and often skipped meals. He had no known diabetes mellitus or hypertension and denied smoking or

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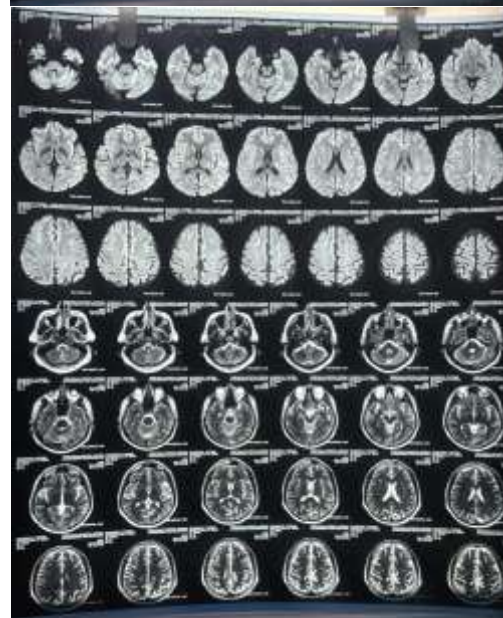
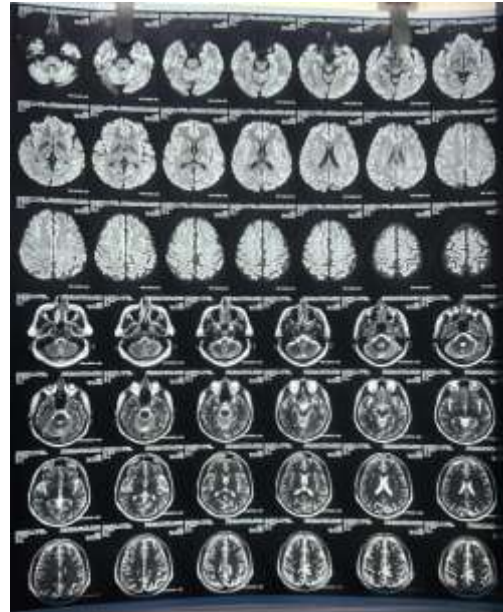
illicit drug use. There was no family history of neuropathy. He was not taking medications known to cause neuropathy. Physical examination showed an alert, oriented man with a blood pressure of 150/80 mm Hg, pulse rate of 71 beats per minute and oxygen saturation of 98 % on room air. He had no pallor, icterus or lymphadenopathy. Cardiovascular and respiratory examinations were normal. The abdomen was soft; the liver was palpable 2 cm below the costal margin. Neurologically, cranial-nerve functions were intact. Tone in both upper and lower limbs was normal, but power in the proximal muscles of the lower limbs was graded as 4/5 compared with 5/5 in the upper limbs. Deep tendon reflexes were absent in the lower limbs and reduced in the upper limbs. Plantar responses were withdrawal bilaterally. Sensory examination revealed decreased pin-prick, touch and temperature sensations in a stocking distribution over both lower limbs; vibration and proprioception were relatively preserved. There was no cerebellar sign. Examination of the spine did not reveal tenderness or deformity.

Investigations

Routine blood tests showed a haemoglobin level of 13.7 g/dL, packed cell volume 42.4 %, total leukocyte count $9.79 \times 10^9/L$ and platelet count $351 \times 10^9/L$. The mean corpuscular volume was increased (100.6 fL), and the mean corpuscular haemoglobin was 32.5 pg, consistent with macrocytosis. Erythrocyte sedimentation rate was 31 mm/h. Serum urea (27 mg/dL) and creatinine (0.62 mg/dL) were within normal limits. Serum sodium (138.1 mmol/L), potassium (4.1 mmol/L) and chloride (101.7 mmol/L) were normal. Liver function tests showed mild transaminitis: aspartate aminotransferase 112.7 IU/L, alanine aminotransferase 72.4 IU/L and γ -glutamyl transpeptidase 156.1 IU/L. Ultrasonography of the abdomen demonstrated hepatomegaly with grade I fatty change and an umbilical hernia. Urine examination was normal, with no albumin or sugar and only 1–2 pus cells.

Thyroid function tests showed a normal free T3 (2.35 pg/mL) and free T4 (1.08 ng/dL) with a mildly elevated thyroid-stimulating hormone (6.27 μ IU/mL). Tests for C-reactive protein (0.7 mg/L) and creatine phosphokinase (97.7 IU/L) were unremarkable. Serum vitamin B12 and folate levels were not available at presentation but were presumed to be low based on the macrocytosis and chronic alcoholism. Nerve conduction studies were planned but deferred. Chest radiograph was normal.

Imaging findings





MRI of the brain with axial fluid-attenuated inversion recovery (FLAIR) and T2-weighted sequences was normal, without evidence of demyelination or ischemia. However, sagittal T2-weighted MRI of the spine revealed a longitudinally extensive hyperintense signal in the posterior columns of the thoracic cord (white arrows in Fig. 1), extending over several vertebral segments without cord expansion. The conus medullaris appeared normal. Axial images (not shown) demonstrated bilateral posterior column hyperintensity, compatible with the “inverted V” sign described in subacute combined degeneration. There was no evidence of compressive disc herniation, ligamentous hypertrophy or vertebral collapse. These imaging findings, in conjunction with the clinical picture, supported the diagnosis of nutritional myelopathy due to vitamin B12 deficiency.

Management and outcome

The patient was admitted for evaluation of paraparesis. He was counselled about alcohol cessation. Empirical therapy with parenteral vitamins was initiated: intramuscular vitamin B12 (hydroxocobalamin) 2 mL once daily, intravenous thiamine 100 mg once daily and oral gabapentin 300 mg at night for neuropathic pain. Vital signs were monitored regularly. Within one week he reported reduced paraesthesias and mild improvement in strength. Follow-up over the subsequent weeks (after discharge) was not available for this report. Nevertheless, early treatment with vitamin supplementation is known to halt or reverse neurological deficits in SCD.

DISCUSSION

Pathophysiology of nutritional myelopathy in alcohol use disorder

Alcoholic neuropathy and myelopathy arise from a combination of direct neurotoxicity and nutritional deficiency. Chronic alcohol consumption leads to poor dietary intake, malabsorption and impaired storage of essential vitamins. Thiamine (vitamin B1) deficiency causes dry beriberi with a symmetrical ascending polyneuropathy and may also lead to Wernicke–Korsakoff syndrome. Vitamin B12 deficiency leads to defective myelin synthesis and demyelination of the dorsal and lateral columns of the spinal cord, producing subacute combined degeneration. Folate deficiency can produce a similar myelopathy and often coexists with cobalamin deficiency in alcoholics.

Patients with alcoholic neuropathy typically present with pain, paraesthesias and ataxia in the distal lower extremities. Progression of the disease leads to symmetrical ascending motor and sensory deficits. Physical examination shows diminished sensation to vibration, pin-prick and temperature, weakness in ankle and toe movements and absent deep tendon reflexes. In contrast, hepatic myelopathy due to portosystemic shunting manifests as spastic paraparesis with increased reflexes and extensor plantar responses. Our patient had flaccid weakness and areflexia, favouring peripheral neuropathy or nutritional myelopathy rather than hepatic myelopathy.

Imaging and laboratory evaluation

MRI is indispensable for evaluating paraparesis. In subacute combined degeneration, sagittal T2-weighted images show longitudinally extensive hyperintense lesions confined to the dorsal columns without cord enlargement. On axial images, the lesion appears as a bilateral posterior column hyperintensity forming an “inverted V” pattern. These findings help distinguish SCD from multiple sclerosis, transverse myelitis, or compressive myelopathy. Brain MRI may show white-matter hyperintensities but is often normal, as in our patient. Nerve conduction studies can assist in differentiating neuropathy from myelopathy; early alcoholic neuropathy often shows normal or mildly reduced conduction velocities, whereas SCD may have normal peripheral conduction because the pathology is central.

Laboratory tests should include complete blood count, liver and renal function tests, vitamin B12, folate, thiamine and other B-vitamin levels, thyroid function tests and inflammatory markers. Macrocytosis, elevated mean corpuscular volume and mildly elevated liver enzymes may raise suspicion for vitamin B12 deficiency in chronic alcoholics, as seen in our patient. Serum vitamin B12 levels <150 pg/mL confirm deficiency; however, normal levels do not exclude functional deficiency due to impaired cellular uptake or nitrous oxide exposure. Elevated methylmalonic acid and homocysteine levels are more sensitive indicators but were not available in our case.

Differential diagnosis

The differential diagnosis of progressive paraparesis in

adults includes compressive myelopathy (disc herniation, epidural abscess or tumour), inflammatory or demyelinating disorders (multiple sclerosis, neuromyelitis optica, acute transverse myelitis), infectious myelitis (tuberculosis, HIV, HTLV-1), metabolic and nutritional myelopathies (vitamin B12 or folate deficiency, copper deficiency), and peripheral neuropathies such as Guillain–Barré syndrome. Alcoholic neuropathy typically presents with distal symmetrical neuropathy, while thiamine deficiency (dry beriberi) may mimic Guillain–Barré syndrome with rapid progression and areflexia. The absence of back pain, lack of compressive lesion on imaging, macrocytosis and improvement with vitamin supplementation support a nutritional aetiology in our patient.

MANAGEMENT

There is no specific laboratory test to diagnose alcoholic neuropathy; hence, management is empirical and based on history, clinical evaluation and exclusion of other causes. Treatment focuses on cessation of alcohol consumption and replacement of deficient nutrients. Thiamine replacement should precede glucose administration to prevent Wernicke’s encephalopathy in acutely ill patients. Parenteral hydroxocobalamin is preferred for vitamin B12 replacement in symptomatic deficiency, followed by maintenance therapy. Folate supplementation should be provided concurrently, particularly in alcoholics, to prevent masking of cobalamin deficiency. Gabapentin, pregabalin or duloxetine can alleviate neuropathic pain. Physical and occupational therapy help improve functional status.

Prognosis and follow-up

Early diagnosis and treatment of nutritional myelopathy can lead to significant neurological recovery. In reported cases of subacute combined degeneration, patients treated with parenteral cobalamin demonstrate regression of spinal cord lesions on follow-up MRI and clinical improvement. However, prolonged deficiency may result in irreversible cord atrophy and persistent deficits. Abstinence from alcohol and adherence to nutritional supplementation are essential to prevent relapse. Our patient showed early improvement after vitamin therapy, but long-term follow-up data were unavailable.

CONCLUSION

Paraparesis in a chronic alcoholic should raise suspicion for nutritional neuropathy or myelopathy. This case underscores the need for a systematic approach that includes a detailed history, neurological examination and

targeted investigations. Macrocytosis and mild liver enzyme elevations in an alcoholic patient should prompt evaluation of vitamin B12 and folate status. MRI plays a pivotal role in differentiating subacute combined degeneration from other causes of paraparesis and demonstrates characteristic posterior column hyperintensity. Early recognition and treatment with vitamin supplementation can halt disease progression and may lead to complete recovery. Clinicians should maintain a high index of suspicion for nutritional myelopathy in alcohol-dependent patients presenting with lower-limb weakness and sensory loss

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