Case Report

Subacute Combined Degeneration of the Spinal Cord Diagnosed by Clinical and Morphological Evaluation in a Resource-Limited Setting: A Case Report

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ABSTRACT

Subacute combined degeneration (SCD) is a potentially reversible but progressive myelopathy caused by vitamin B_{12} deficiency. Biochemical markers and MRI are often unavailable or unaffordable in resource-limited settings, making timely clinical diagnosis dependent on careful clinical evaluation and morphological haematological assessment. A 42-year-old female petty trader presented with recurrent severe anaemia, progressive lower limb weakness, and colicky abdominal pain. Examination showed pallor, angular cheilosis, glossitis, bilateral lower limb hypotonia, paraparesis with power of 2/5, hyporeflexia, impaired joint position and vibration sensations, and stocking-distribution sensory loss. Although the automated blood count showed a deceptively normocytic mean corpuscular volume (79.6 fL), peripheral blood film and bone marrow aspiration demonstrated dimorphic erythropoiesis, oval macrocytes, microcytes, hypersegmented neutrophils, giant metamyelocytes, and megaloblastic erythropoiesis, consistent with combined vitamin B_{12} and iron deficiency. MRI and serum vitamin B_{12} assay were unaffordable. Parenteral vitamin B_{12}, folic acid, iron supplementation, and neurorehabilitation resulted in rapid haematological recovery. At one-year follow-up, she remained asymptomatic and ambulant. An integrated clinico-morphological approach can facilitate early recognition and successful management of SCD where confirmatory investigations are unavailable.

Keywords: Bone marrow aspiration; Megaloblastic anaemia; Peripheral blood film; Subacute combined degeneration; Vitamin B_{12} deficiency.

INTRODUCTION

Subacute combined degeneration (SCD) of the spinal cord is a rare but potentially reversible neurological complication of vitamin B_{12} (cobalamin) deficiency.[1,2] It is characterised by demyelination of the dorsal and lateral columns of the spinal cord, causing impaired proprioception, vibration sense, corticospinal tract dysfunction, and peripheral neuropathy. Vitamin B_{12} is an essential cofactor in DNA synthesis and myelin maintenance; hence, prolonged deficiency causes both megaloblastic haematopoiesis and neurological dysfunction.[1,3,4] It is a multisystem disorder and early features include fatigue, glossitis, angular cheilosis, weight loss, and megaloblastic anaemia. Neurological symptoms may progress from paraesthesia and gait instability to weakness, impaired joint position and vibration sense, spastic paraparesis, sphincter dysfunction, cognitive impairment, and irreversible disability if treatment is delayed.[1,2,5]

Diagnosis is usually supported by serum vitamin B_{12}, methylmalonic acid (MMA), homocysteine assays, and spinal MRI, which typically shows symmetrical posterior column T2 hyperintensities ("inverted V" sign).[3,6] However, serum vitamin B_{12} may be normal despite significant deficiency, and MRI is normal in many patients, with reported sensitivities of approximately 15% to just over 50%.[1,6–10] Therefore, neither is mandatory when the clinical presentation is strongly suggestive.

In low- and middle-income countries (LMIC), including Nigeria, MRI and specialised biochemical tests are often unavailable or unaffordable, so diagnosis relies on clinical assessment supported by haematological investigations. Similarly, reports from Nigeria and other African countries demonstrated that SCD was diagnosed and treated using clinical and morphological evidence when advanced investigations were unavailable.[11,12]

An additional diagnostic challenge is coexisting iron deficiency, which may mask the macrocytosis of megaloblastic anaemia on full blood count (FBC) and

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produce a normal or reduced mean corpuscular volume (MCV), potentially delaying diagnosis.[13,14] In such cases, peripheral blood film (PBF) and bone marrow aspiration (BMA) examination will still reveal macro-ovalocytes, hypersegmented neutrophils, megaloblasts and giant metamyelocytes despite apparently normal red cell indices.[15,16]

We report a 42-year-old woman with SCD secondary to vitamin B_{12} deficiency, diagnosed by integrating characteristic neurological findings with PBF and bone marrow morphology in the absence of MRI and biochemical confirmation. The case highlights the value of a clinico-morphological approach in resource-limited settings.

CASE PRESENTATION

A 42-year-old petty trader was referred from a private hospital with a one-year history of recurrent severe anaemia, four weeks of progressive inability to stand or walk, and four days of colicky abdominal pain with loose mucoid bloody stools.

One year earlier, she received two units of blood for severe anaemia of uncertain cause during evaluation for epigastric pain, with persistent weakness thereafter. There was no history of gastrointestinal or abnormal uterine bleeding.

Four weeks before presentation, she developed progressive bilateral lower limb weakness with severe leg pain, paraesthesia, numbness, cramps, and inability to bear weight. There was no preceding trauma. She subsequently developed colicky abdominal pain with bloody mucoid diarrhoea, four days prior to presentation and reported gradual weight loss, but denied fever, cough, night sweats, back pain, or sphincteric disturbance.

She had no significant past medical history and denied alcohol, smoking, or drug (mis)use.

Physical examination

She was chronically ill-looking, markedly pale, and moderately dehydrated, with a respiratory rate of 26 breaths/min, pulse of 110 beats/min, and blood pressure of 100/60 mmHg. Temperature was normal. The abdomen was generally tender, worse at the epigastrium, without organomegaly. Neurological examination showed bilateral lower limb hypotonia, power 2/5, and hyporeflexia; upper limb tone, power and reflexes were normal.

Initial investigations and management

The initial differential diagnoses included chronic severe anaemia of uncertain aetiology, acute dysentery, peptic ulcer disease, and lumbar spondylosis with possible compressive myelopathy.

Initial investigations demonstrated severe anaemia (haemoglobin 5.9 g/dL; haematocrit 18%) and severe hypocalcaemia (serum calcium 1.2 mmol/L). Screening for HIV, hepatitis B virus, and hepatitis C virus was negative.

She received supportive care, correction of hypovolaemia and hypocalcaemia, empirical antimicrobial therapy, proton pump inhibition, and three units of packed red cells.

By day three, her gastrointestinal symptoms had resolved, but she developed worsening lower back pain radiating to both lower limbs with muscle spasms and fasciculations. Angular cheilosis and a beefy red tongue suggestive of glossitis were also noted.

Pre-transfusion full blood count showed bicytopenia with haemoglobin 5.9 g/dL, white cell count 3.2×10^9/L (neutrophils 1.0, lymphocytes 2.1, mixed cells 0.1×10^9/L), platelets 152×10^9/L, and an apparently normocytic MCV (79.6 fL).

PBF showed a dimorphic red cell population comprising oval macrocytes admixed with microcytes together with hypersegmented neutrophils.

The reticulocyte count was reduced at 0.2%.

Prompted by the severe anaemia, neutropaenia, and a markedly reduced reticulocyte count, a bone marrow aspiration was done which showed a hypercellular marrow with megaloblastic erythropoiesis, giant metamyelocytes, giant band forms, and micro-normoblasts, consistent with combined megaloblastic and iron deficiency anaemia.

Imaging demonstrated a calcified uterine fibroid and degenerative lumbosacral vertebral changes. Stool microscopy and culture were negative.

Based on the clinical and morphological findings, severe megaloblastic anaemia with iron deficiency and suspected vitamin B_{12} deficiency-associated myelopathy was diagnosed. Treatment comprised intramuscular vitamin B_{12} (1000 µg on alternate days for six doses, then weekly), folic acid (5 mg daily), ferrous sulphate (200 mg three times daily), and analgesia. Orthopaedic and gynaecological consultations advised conservative management.

Neurological evaluation and clinical course

Within ten days of haematinic therapy, glossitis, angular cheilosis, paraesthesia, and generalised weakness improved markedly. However, she developed poor sleep and transient irrational speech while persistent lower limb spasms remained. Repeat testing showed improving haematological indices (haemoglobin 7.9 g/dL), normal calcium (2.33 mmol/L), and essentially normal renal and liver function. Neurological review showed improved lower limb power (3/5), restored deep tendon reflexes, bilateral flexor plantar responses, impaired joint position sense, and symmetrical glove-and-stocking sensory loss, supporting SCD secondary to vitamin B_{12} deficiency.

Serum vitamin B_{12} assay and spinal MRI could not be performed because of financial constraints. However, given the characteristic neurological findings, marrow morphology, and favourable early response, treatment continued. Gabapentin, baclofen, piracetam, and intensive physiotherapy were added.

Within two weeks, delirium, muscle spasms, and neuropathic pain resolved completely, and she could walk with human assistance. Haemoglobin was 9.8 g/dL with normal neutrophil counts. She was discharged with vitamin B_{12}, folic acid, iron, gabapentin, baclofen, and piracetam.

At clinic follow up, she was walking with a stick, with complete resolution of pain. Haemoglobin was 10.3 g/dL with stable blood counts. At one-year telephone follow-up, she was asymptomatic, walked un-aided, and had resumed her occupation. She had defaulted specialist follow-up because she felt well, but adhered to maintenance medications and agreed to resume clinic attendance after counselling.

DISCUSSION

The diagnostic challenges in this case reflect routine practice across many LMIC, where MRI, serum vitamin B_{12} assays, MMA, and homocysteine testing are unavailable or unaffordable for many patients. Consequently, clinicians often rely on careful neurological examination together with PBF and BMA morphology to establish a working diagnosis and initiate potentially life-saving therapy.

A Nigerian paediatric patient with SCD was successfully diagnosed clinically and responded well to parenteral vitamin B_{12}, although relapse followed default from follow-up, emphasising the importance of lifelong monitoring.[12] Likewise, Ekabe et al. described successful diagnosis of vitamin B_{12} deficiency neuropathy based primarily on clinical findings and peripheral smear morphology.[11] Together with our case, these reports reinforce the importance of sound clinical judgement in resource-poor settings.

Another strength of this report is the clinico-morphological correlation. Unlike reports relying predominantly on biochemical confirmation or MRI, [5,6] our patient exhibited striking neurological symptoms and classical haematological morphology. This created a compelling diagnostic picture that justified immediate empirical therapy. The rapid neurological and haematological response to vitamin B_{12} replacement further strengthened the diagnosis.

Noteworthy, also, was the transient delirium that developed about ten days after initiation of vitamin B_{12} replacement. Neuropsychiatric manifestations, including delirium, psychosis, mood disorders, and cognitive impairment, are recognised in severe vitamin B_{12} deficiency, although their mechanisms remain incompletely understood.[1,4] Whether this represented the natural evolution of the disease or an uncommon recovery phenomenon cannot be determined but more importantly, symptoms resolved completely with continued vitamin B_{12} therapy and supportive neurological care.

The excellent functional outcome supports evidence that neurological recovery depends largely on early recognition and prompt Vitamin B_{12} replacement; early treatment often achieves substantial recovery, whereas prolonged deficiency may cause permanent disability.[1,2,5,7,16]

Although our patient defaulted from follow-up after symptomatic recovery, she remained well at one year review. Such default is common in chronic disease management in low-resource settings, once symptoms resolve. So continuous counselling on adherence and follow-up is essential.[7,12]

LIMITATIONS

Serum cobalamin, methylmalonic acid, and homocysteine assays and spinal MRI could not be performed because of financial constraints, so alternative causes of myelopathy could not be excluded. Nevertheless, the characteristic neurological syndrome, classical PBF and BMA features, and the marked, sustained response to vitamin B_{12} strongly supported the diagnosis.

CONCLUSION

SCD is a potentially reversible but disabling neurological disorder, and biochemical assays and MRI complement but do not replace clinical reasoning as demonstrated by this case. Where these are unavailable, careful neurological evaluation with PBF and BMA morphology can establish a strong working diagnosis, supported by a marked response to vitamin B_{12} replacement. Therefore, clinicians should maintain a high index of suspicion in patients with progressive myelopathy and morphological evidence of megaloblastic anaemia, as prompt empirical treatment may prevent irreversible disability.[1,2,7,11]

RECOMMENDATIONS

In resource limited settings, clinicians should suspect SCD in patients with progressive myelopathy and anaemia, even when the MCV is normal, utilising PBF and BM morphology carefully.

Commence empirical vitamin B_{12} immediately if clinical and morphological findings are highly suggestive of SCD with lifelong follow-up and adherence counselling. And there should be subsidised access to vitamin B_{12} assays and MRI.

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