Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.6 No. 9 (2025): September 2025 Issue
https://doi.org/10.51168/sjhrafrica.v6i9.2068
Original Article

Vitamin B12 deficiency presenting with syringomyelia. Case report.

Dr. Sanaullah Mudassir[1], Dr. Ankit Panjwani[2]*
[1]Senior Consultant Neurologist, Department of Neurology, Jay Prabha Medanta Superspeciality Hospital, Patna, Bihar, India.
[2]Consultant Neurologist, Department of Neurology, Government Medical College and Superspeciality Hospital, Nagpur, India.

Abstract:

Background:

Syringomyelia is characterized by the formation of a cyst or cavity (syrinx) within the spinal cord. It is typically associated with Chiari malformation, trauma, or other spinal cord abnormalities. Vitamin B12 deficiency can lead to various neurological and haematological manifestations, including myelopathy, neuropathy, cognitive impairment, and macrocytic anemia. This case highlights a rare or underreported association between vitamin B12 deficiency and syringomyelia.

Case presentation:

A 22-year-old young male, a vegetarian, presented with progressive gait disturbances for the last 4 months. He also had a history of imbalance on standing with closed eyes, with increased gait difficulty in the dark. There was also a history of numbness and paraesthesia of both upper and lower limbs. The neurological examination showed spasticity of both lower limbs, hyperreflexia, and gait ataxia with normal bulk and power, and increased deep tendon reflexes and absent bilateral ankle jerks. The plantar were flexors on both sides. Romberg's test was positive. Blood investigation revealed pancytopenia (haemoglobin-4.6, total leucocyte count- 3900, Platelet count-62,000) with raised MCV- 118 fL. Serum vitamin B12 was found to be low (78 pg/ml) with elevated homocysteine levels (27 umol/l). MRI revealed prominence of the central canal in the cervical spinal cord at the C2-C3 vertebral level. The nerve conduction study showed inexcitable bilateral sural nerves. He was treated with intramuscular vitamin B12 injections. He was given 1000 µg of methylcobalamin initially daily for a week and then weekly for a month, and once-monthly injections for 6 months. After 9 months, he significantly recovered to his near-normal physical capacity with a normal gait. There was also almost complete regression of the syrinx cavity.

Conclusion:

Syrinx formation secondary to B12 deficiency is rarely reported in the literature. This case highlights the importance of treating the underlying vitamin B12 deficiency before planning for any interventional procedure.

***Keywords**: B12 deficiency, syringomyelia, reversible.*
***Submitted:** July 27, 2025 **Accepted:** August 20, 2025 **Published:** September 30, 2025*

***Corresponding Author:** Ankit Panjwani, MD, DM*
***Email:** ankitpanjwani92@gmail.com*
Consultant Neurologist, Department of Neurology, Government Medical College and Superspeciality Hospital, Nagpur, India.

Background:

Syringomyelia is characterized by the formation of a cyst or cavity (syrinx) within the spinal cord. It is typically associated with Chiari malformation, trauma, or other spinal cord abnormalities. Vitamin B12 deficiency can lead to various neurological and haematological manifestations, including myelopathy, neuropathy, cognitive impairment, and macrocytic anemia[1]. This case highlights a rare or underreported association between vitamin B12 deficiency and syringomyelia.

Syringomyelia leads to progressive neurological deficits, including pain, weakness, spastic-ataxic gait, and sensory loss. The most widely recognized postulate for syrinx formation suggests that the pulsatile pressure is conveyed to the central canal via the Virchow-Robbins gaps, resulting in a syrinx2. Vitamin B12, also known as cobalamin, is a water-soluble vitamin essential for the proper functioning of the nervous system. Around half of the senior population may be deficient in vitamin B12, with this prevalence being higher among Indians and especially among vegetarians3.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.6 No. 9 (2025): September 2025 Issue
https://doi.org/10.51168/sjhrafrica.v6i9.2068
Original Article

Deficiency in B12 can lead to a variety of neurological symptoms, such as peripheral neuropathy, myelopathy, and cognitive disturbances.

There are very few documented instances of syringomyelia in vitamin B12-deficient patients4-7. In one of the previous reported cases in the literature, there was an association with Chiari malformation as well, and in another case, there was a syrinx-like clinical presentation with radiological features of subacute combined degeneration7. Although the link between B12 deficiency and neurological conditions is well-documented, the causal association between B12 deficiency and syringomyelia remains not well understood. Vitamin B12 plays a critical role in myelin synthesis and nerve function. Deficiency can result in demyelination, which may exacerbate or contribute to spinal cord damage in patients with syringomyelia. The mechanisms of these interactions likely involve both direct neurotoxic effects on the spinal cord as well as potential disruption of vascular supply to the spinal cord8. In one of the reported cases, the authors postulated that B12 deficiency may cause an asymptomatic syrinx cavity. This speculation was further supported by the notion that the lesions that are formed in medulla spinalis due to SCD usually disappear after six or eight weeks, similar to the disappearance of the syrinx cavity on MRI after eight weeks in their case. The postulate of syrinx formation due to vitamin B12 deficiency in our case is supported by the remission of the radiologic abnorm alities and clinical complaints following B12 therapy.

Case presentation:

A 22-year-old young male, a vegetarian, presented with progressive gait disturbances for the last 4 months. He also had a history of imbalance on standing with closed eyes, with increased gait difficulty in the dark. There was also a history of numbness and paraesthesia of both upper and lower limbs, initially starting from the tips of fingers and toes. He also had slippage of footwear while walking, without awareness. There was no history of neck pain, electric shock-like sensation going down his spine on neck bending, bladder or bowel dysfunction. There was no history of diabetes, chronic diarrhoea, or alcohol addiction. On general physical examination, he was having pallor suggestive of anemia. The neurological examination showed spasticity of both lower limbs, hyperreflexia, and gait ataxia with normal bulk and power, increased deep tendon reflexes, and absent bilateral ankle jerks. The plantar were flexors on both sides. Romberg's test was positive. Blood investigation revealed pancytopenia (haemoglobin-4.6, total leucocyte count- 3900, Platelet count-62,000) with raised MCV- 118 fL. Serum vitamin B12 was found to be low (78 pg/ml) with elevated homocysteine levels (27 umol/l). Other routine blood investigations, such as blood sugar, renal function tests, lipid, liver function tests, folate level, and serum electrolytes, were normal. The thyroid function tests were also normal. MRI revealed prominence of the central canal in the cervical
spinal cord at the C2-C3 vertebral level (figures 1 and 2).

Syringomyelia is characterized by the formation of a cyst or cavity (syrinx) within the spinal cord. It is typically associated with Chiari malformation, trauma, or other spinal cord abnormalities. Vitamin B12 deficiency can lead to various neurological and haematological manifestations, including myelopathy, neuropathy, cognitive impairment, and macrocytic anemia. This case highlights a rare or underreported association between vitamin B12 deficiency and syringomyelia.

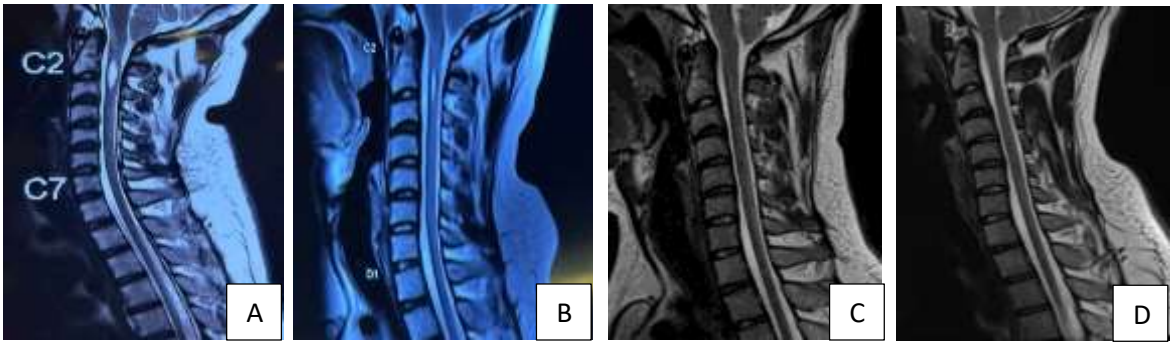

Figure 1. Sagittal T2W images showing hyperintense syrinx cavity in the cervical spinal cord from C2 to C3 level at baseline(a), (b), and longitudinal follow-up MRI after vitamin B12 therapy (c), (d) showing near complete resolution of Syringomyelia in the cervical spinal cord.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.6 No. 9 (2025): September 2025 Issue
https://doi.org/10.51168/sjhrafrica.v6i9.2068
Original Article

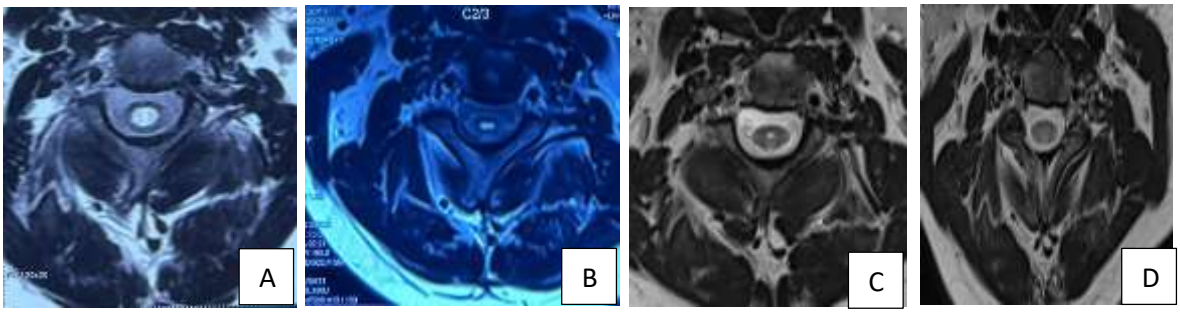

Figure 2. Axial T2W images showing hyperintense syrinx cavity in the cervical spinal cord from C2 to C3 at baseline (a), (b) and longitudinal follow-up MRI after vitamin B12 therapy (c), (d) showing near complete resolution of Syringomyelia in the cervical spinal cord

The nerve conduction study showed inexcitable bilateral sural nerves. The visual-evoked potentials demonstrated normal findings. He was treated with intramuscular vitamin B12 injections. He was given 1000 µg of methylcobalamin initially daily for a week and then weekly for a month, and once-monthly injections for 6 months. The physiotherapy and rehabilitation program was also continued for gait ataxia and spasticity. The patient had marked improvement in clinical and radiological features at follow-up visits. After 9 months, he significantly recovered to his near-normal physical capacity with a normal gait. There was also almost complete regression of the syrinx cavity.

Conclusion:

Syrinx formation secondary to B12 deficiency is rarely reported in the literature. This case highlights the importance of treating the underlying vitamin B12 deficiency before planning for any interventional procedure.

Limitations:

This is a single case report, and further larger studies are needed to establish the causal association between syringomyelia and vitamin B12 deficiency.

Take-away lessons:

Syringomyelia can be a rare presentation of Vitamin B12 deficiency and must be considered as a differential for otherwise unexplained syrinx.

List of abbreviations:

SCD: Subacute combined degeneration

MRI: Magnetic Resonance Imaging

Source of funding:

Self

Conflict of interest:

No conflict of interest

Author contributions:

Drafting of manuscript and data interpretation: Dr. Ankit Panjwani
Data collection and proofreading: Dr. Sanaullah Mudassir

Data availability:

All the available data is shared

Author biography:

Dr. Sanaullah Mudassir is a Senior Consultant Neurologist in the Department of Neurology at Jay Prabha Medanta Hospital in Patna, India. His qualifications include MBBS, MD in General Medicine, and DM in Neurology. He has various national and international publications in his name.

Dr. Ankit Panjwani is a Consultant Neurologist in the Department of Neurology at Government Medical College and Super speciality Hospital, Nagpur, India. His qualifications include MBBS, MD in General Medicine, and DM in Neurology. He is a Gold Medallist in DM and has various research papers in his name.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.6 No. 9 (2025): September 2025 Issue
https://doi.org/10.51168/sjhrafrica.v6i9.2068
Original Article

References:

1. Green R, Allen LH, Bjørke-Monsen AL, et al. Vitamin B12 deficiency [published correction appears in Nat Rev Dis Primers. 2017 Jul 20; 3:17054]. Nat Rev Dis Primers. 2017; 3:17040. Published 2017 Jun 29. https://doi.org/10.1038/nrdp.2017.54
2. Menezes AH. Chiari I malformations and hydromyelia: complications. Pediatr Neurosurg 1991;17:146 54. https://doi.org/10.1159/000120586
3. Pennypacker LC, Allen RH, Kelly JP, et al. High prevalence of cobalamin deficiency in elderly outpatients. J Am Geriatr Soc 1992;40: 1197-204. https://doi.org/10.1111/j.1532-5415.1992.tb03641.x
4. Güler K, Turhanoğlu AD. Does B12 deficiency lead to syringomyelia? Turk J Phys Med Rehabil. 2020;66(3):370-371. Published 2020 Aug 18. doi:10.5606/tftrd.2020.5040. https://doi.org/10.5606/tftrd.2020.5040
5. Reynolds MG, Lammoglia FJ. Spinal cord syrinx in a patient with subacute combined degeneration secondary to vitamin B12 deficiency: case report. Kans Med. 1988;89(6):170-172.
6. Verma R, Praharaj HN. Unusual association of Arnold-Chiari malformation and vitamin B12 deficiency. BMJ Case Reports 2012;10.1136/bcr-03-2012-6138. https://doi.org/10.1136/bcr-03-2012-6138
7. Puri V, Chaudhry N, Gulati P. Syringomyelia-like manifestation of subacute combined degeneration. J Clin Neurosci. 2004;11(6):672-675. https://doi.org/10.1016/j.jocn.2003.11.005
8. Zhang, Z., et al. (2022). Neurodegenerative Diseases and Vitamin B12 Deficiency: Pathophysiological Insights. Neurological Science, 25(3), 57-68.
9. Hemmer B, Glocker FX, Schumacher M, Deuschl G, Lücking CH. Subacute combined degeneration: clinical, electrophysiological, and magnetic resonance imaging findings. J Neurol Neurosurg Psychiatry 1998;65:822-7. https://doi.org/10.1136/jnnp.65.6.822

PUBLISHER DETAILS:

Student's Journal of Health Research (SJHR)
(ISSN 2709-9997) Online
(ISSN 3006-1059) Print
Category: Non-Governmental & Non-profit Organization
Email: studentsjournal2020@gmail.com
WhatsApp: +256 775 434 261
Location: Scholar's Summit Nakigalala, P. O. Box 701432, Entebbe Uganda, East Africa