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Mixed Etiology Dementia in an Elderly Male: Vitamin B12 Deficiency, Vascular Changes, and Possible Idiopathic Normal Pressure Hydrocephalus- A Case Report

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KEYWORDS

Vitamin B12 deficiency, NPH-like features, reversible dementia, gait disturbance, elderly cognition

ABSTRACT:

Background: Dementia in older adults has multiple etiologies, including reversible causes that require careful detection. Vitamin B12 deficiency can present with cognitive impairment, gait disturbance, and neuropsychiatric symptoms. Additionally, neuroimaging features mimicking normal pressure hydrocephalus (NPH) may complicate the diagnostic picture.

Case Presentation: We describe a 71-year-old man presenting with rapidly progressive cognitive impairment, gait unsteadiness, and frequent falls. MRI suggested NPH-like features, and laboratory evaluation revealed severe Vitamin B12 deficiency. A CSF tap test was performed, followed by parenteral Vitamin B12 supplementation and symptomatic pharmacotherapy. His MMSE improved from 13/30 to 20/30 over one month without adverse events.

Conclusion: This case highlights the importance of assessing reversible metabolic causes even when imaging suggests NPH. Vitamin B12 deficiency can produce NPH-like clinical and radiological features, and correction may lead to significant cognitive recovery.

INTRODUCTION

Dementia is a clinical illness marked by a progressive deterioration of cognitive abilities that disrupts everyday functioning and autonomy[1]. Globally, approximately 55 million individuals are affected, with projections reaching 152 million by 2050 due to population aging[2,3]. Alzheimer's disease (AD) is the most prevalent form, followed by vascular dementia, Lewy body dementia, and frontotemporal dementia[4]. Cognitive impairment in the elderly is often multifactorial, with reversible causes including vitamin B12 deficiency, hypothyroidism, depression, and medication effects[5,6]. Vitamin B12 deficiency, prevalent in older adults, can impair memory, executive function, visuospatial skills, and behavior[7,8]. Chronic vascular lesions, such as lacunar infarcts and microangiopathic changes, contribute to subcortical cognitive impairment and gait disturbances[9]. Idiopathic normal pressure hydrocephalus (iNPH), a treatable cause of dementia, presents with the classic triad of gait difficulty, urinary incontinence, and cognitive decline, often confounding differential diagnosis[10-12]. This case highlights the importance of comprehensive assessment in elderly patients with cognitive decline, integrating neuropsychological testing, laboratory evaluation, and structural imaging.

CASE PRESENTATION

An informed written consent was taken from the son as patient did not have capacity to consent. A 75-year-old man presented with an eight-month history of progressive memory impairment, disorientation, and difficulty navigating both familiar and unfamiliar environments. Over the preceding month, he developed urinary incontinence, gait disturbances, and behavioural changes, including increased irritability and

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inappropriate language. There was no history of head trauma, infection, or prior psychiatric illness.

Neurological examination revealed bradykinesia, dysdiadochokinesis, and impaired tandem walking with ataxic gait. Higher mental function assessment showed impaired and fluctuating orientation, intact attention but reduced concentration, and deficits in immediate, recent, and remote memory. Comprehension was preserved, but arithmetic abilities were impaired. MMSE was 13/30, with deficits in orientation, concentration, memory, language, and visuospatial function. ACE-III score was 44/100: attention 6/18, memory 6/26, fluency 2/14, visuospatial 7/16, and language 23/26.

Laboratory investigations revealed severe vitamin B12 deficiency (133 pg/mL), elevated homocysteine (51.8 µmol/L), and HbA1c of 7.4%. MRI brain demonstrated chronic lacunar infarcts in bilateral frontal and corona radiata regions, hypertensive microangiopathic bleeds in the bilateral lentiform nuclei and right thalamus, periventricular and subcortical ischemic changes, and age-related cerebral and cerebellar atrophy. These findings suggested a mixed etiology, including reversible metabolic deficiency, vascular contributions, and possible iNPH.

Because the patient was not a suitable candidate for ventriculoperitoneal (VP) shunt surgery, a conservative and neuropharmacological approach was adopted. He was initiated on parenteral vitamin B12 supplementation (3000 µg/day intravenously) to correct significant cobalamin deficiency. Cognitive enhancers were started with donepezil 5 mg once daily and memantine 5 mg once daily in the morning. For behavioral disturbances—irritability, sleep impairment, and verbal aggression—he was prescribed quetiapine 50 mg twice daily, which was later decreased to once daily due to morning sedation. His hypertension was treated with amlodipine 5 mg and his diabetes mellitus regimen was modified by shifting from insulin to oral hypoglycemic agents. On one month follow-up, the patient demonstrated a marked cognitive improvement, with the MMSE increasing from 13/30 to 20/30. No adverse events were reported during the follow-up period.

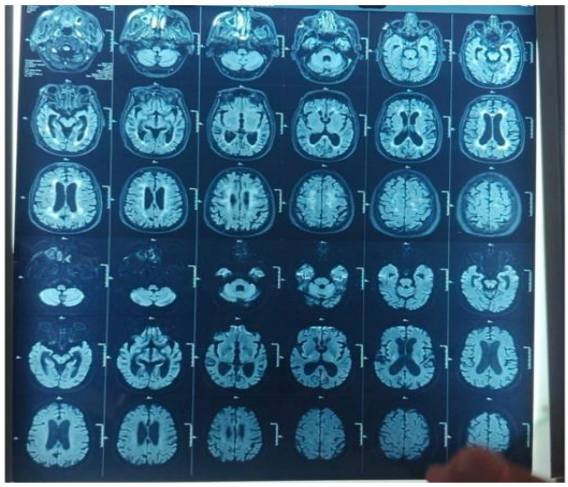

Figure 1: MRI Brain showing chronic lacunar infarcts, hypertensive microangiopathic bleeds, and periventricular ischemic changes.

DISCUSSION

This instance exemplifies the intricate interaction of metabolic, vascular, and hydrocephalic elements in cognitive deterioration in the elderly. Vitamin B12 deficiency is a recognized reversible cause of cognitive impairment, with deficits in memory, executive function, visuospatial ability, and behavioral regulation[7,8]. Hyperhomocysteinemia associated with B12 deficiency may exacerbate vascular injury, compounding cognitive deficits[13]. The presence of chronic lacunar infarcts and microangiopathic changes suggests subcortical vascular contributions, which commonly impair executive function and gait[9,14].

The combination of cognitive decline, gait disturbance, and urinary incontinence raises suspicion for iNPH, a treatable cause of dementia. CSF diversion procedures have demonstrated improvements in gait and functional outcomes, with variable cognitive benefit[10,11]. Early detection and intervention in mixed etiologies are crucial to optimize recovery and quality of life. Multimodal management in this patient would include vitamin B12 supplementation, management of vascular risk factors, cognitive rehabilitation, and assessment for potential surgical intervention for iNPH[7,10].

This instance highlights the necessity of thorough assessment in older people exhibiting cognitive deterioration. Laboratory screening for reversible metabolic deficiencies, detailed neuropsychological

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testing, and neuroimaging to assess vascular and structural abnormalities are essential. Integrating these findings can guide individualized treatment and improve outcomes in patients with multifactorial dementia.

CONCLUSION

Elderly patients with cognitive impairment often have mixed etiologies, including reversible metabolic deficiencies, vascular lesions, and possible hydrocephalus. Early recognition and treatment of reversible causes, along with appropriate management of vascular and hydrocephalic changes, are critical to improving cognitive and functional outcomes. Comprehensive assessment using neuropsychological tests, laboratory evaluation, and neuroimaging is essential for accurate diagnosis and optimal management.

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