SUNTEXT REVIEWS

SunText Review of Case Reports & Images
ISSN: 2766-4589

Open Access
Case Report
Volume 7:2

Anti-VGCC Antibody–Associated Autoimmune Cerebellitis: A Case Report and Review of Diagnostic and Surveillance Strategies

Tuwei Y[1], Offord O[2] and Kumar P[3,*]

[1]*Department of Medicine, Mackay Base Hospital and Mater Hospital Mackay, Australia*
[2]*Medical Student, James Cook University, Mackay, Queensland, Australia*
[3]*Department of Medicine, Mackay Base Hospital and Mater Hospital Mackay, Australia*

[*]**Corresponding author:** Kumar P, Department of Respiratory and Sleep Medicine, Mater Medical Suites, 123 Willetts Rd, North Mackay QLD 4740, Australia; E-mail: drpranavkumar@gmail.com

Received date: 03 April 2026; **Accepted date:** 07 April 2026; **Published date:** 12 April 2026

Citation: Tuwei Y, Offord O, Kumar P (2026) Anti-VGCC Antibody–Associated Autoimmune Cerebellitis: A Case Report and Review of Diagnostic and Surveillance Strategies. SunText Rev Case Rep Image 7(2): 180.

DOI: https://doi.org/10.51737/2766-4589.2026.180

Copyright: © 2026 Tuwei Y, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Objective: Autoimmune cerebellar ataxia is a potentially reversible cause of progressive adult-onset ataxia and forms part of the spectrum of immune-mediated cerebellar syndromes. Voltage-gated calcium channel (VGCC) antibodies, particularly P/Q-type, are strongly associated with paraneoplastic neurological syndromes, most notably those associated with small cell lung carcinoma. Neurological manifestations may precede tumour detection, and an initial negative malignancy screening test does not preclude occult neoplasia.

Case Presentation: A 68-year-old woman presented with a three-year history of progressive gait ataxia, episodic vertigo, and chronic tinnitus. Cerebrospinal fluid analysis revealed elevated protein with minimal pleocytosis, indicative of inflammatory pathology. Serum testing confirmed elevated anti-VGCC antibodies (171 pM). Comprehensive autoimmune, infectious, and genetic investigations were unremarkable, and cross-sectional imaging did not definitively identify malignancy.

Management and Outcome: The patient received intravenous immunoglobulin therapy and multidisciplinary rehabilitation. Early immunotherapy has been linked to improved neurological outcomes in immune-mediated cerebellar syndromes. Structured longitudinal malignancy surveillance was implemented in accordance with current paraneoplastic guidelines. Partial symptom stabilization was achieved.

Conclusion: This case highlights the importance of recognizing immune-mediated cerebellar ataxia, initiating prompt immunomodulatory therapy, and maintaining ongoing oncological surveillance despite initial negative tumour screening.

Keywords: Autoimmune cerebellar ataxia; Voltage-gated calcium channel antibodies; Paraneoplastic neurological syndromes; Lambert–Eaton myasthenic syndrome; Intravenous immunoglobulin; Immune-mediated ataxia; Oncological surveillance

Introduction

When adults present with subacute or progressive cerebellar ataxia, it is essential to consider immune-mediated and paraneoplastic causes, as timely intervention may prevent irreversible neurological disability [1-4]. Autoimmune cerebellar ataxia arises when antibody-mediated immune mechanisms disrupt cerebellar synaptic transmission, impairing Purkinje cell function and leading to progressive cerebellar dysfunction [1]. Early recognition is critical, as prompt immunotherapy can limit permanent neuronal injury and improve clinical outcomes [1,8]. Antibodies directed against voltage-gated calcium channels (VGCC), particularly the P/Q-type, are most associated with Lambert–Eaton myasthenic syndrome (LEMS) and paraneoplastic neurological syndromes, especially those linked to small cell lung carcinoma (SCLC) [2,4]. However, cerebellar syndromes can manifest without overt neuromuscular junction involvement and may precede tumour detection by months or years [4,5]. Consequently, a negative initial malignancy screen does not exclude occult neoplasia, necessitating structured longitudinal

oncological surveillance [5,6]. We report a case of anti-VGCC antibody–associated autoimmune cerebellar ataxia in a patient with negative initial cancer screening, emphasizing diagnostic considerations and the importance of early immunomodulatory therapy and continued malignancy surveillance.

Case Presentation

A 68-year-old woman presented with a three-year history of progressively worsening gait imbalance, episodic vertigo, and tinnitus, resulting in recurrent falls and increasing functional limitation. She reported significant balance deterioration in low light and frequently required wall support when walking at night. She described episodes of objective vertigo, recently intensified chronic tinnitus, and motion-induced nausea while travelling in vehicles. She also reported persistent cognitive difficulties following a traumatic brain injury sustained at 28 years of age, characterized by episodic disorientation and increased forgetfulness. Her past medical history was notable for cervical dystonia managed with botulinum toxin injections. She also reported longstanding gastrointestinal disturbances characterized by episodic vomiting and diarrhoea associated with dairy intolerance. At 28 years of age, she sustained significant traumatic injuries in a motorcycle accident, including multiple pelvic fractures, contributing to baseline mobility limitations. She was a lifelong non-smoker and consumed alcohol only occasionally. On neurological examination, she demonstrated a broad-based ataxic gait with impaired postural stability. Romberg's test was positive. Head movements provoked vertigo accompanied by horizontal unidirectional nystagmus. Limb strength was preserved, and no focal cranial nerve deficits were identified. Coordination testing revealed dysmetria and impaired heel–shin performance, consistent with cerebellar dysfunction. Diplopia was absent. The constellation of progressive gait ataxia, vestibular symptoms, and objective cerebellar signs was consistent with an acquired cerebellar syndrome, prompting evaluation for immune-mediated and paraneoplastic causes [1,3].

Investigations

Routine biochemistry demonstrated mild hyponatremia (sodium 129 mmol/L; reference range 135–145) and low urea (2.8 mmol/L; reference range 2.5–7.5). Renal function was preserved (creatinine 54 µmol/L; eGFR >90 mL/min/1.73 m²). Corrected calcium, magnesium, phosphate, and glucose were within normal limits. Full blood count showed hemoglobin 116 g/L (mild anaemia), white cell counts 3.8 ×10⁹/L (low-normal), and platelet count 219 ×10⁹/L. Inflammatory markers were elevated (ESR 52 mm/h; CRP 14 mg/L).

Serum testing demonstrated elevated anti–voltage-gated calcium channel (VGCC) antibodies at 171 pM (reference <30 pM). Thyroid peroxidase antibodies (45.6 IU/mL; reference <5.6) and thyroglobulin antibodies (29.9 IU/mL; reference <4.1) were elevated, consistent with euthyroid autoimmune thyroiditis (thyroid function tests normal). Anti-VGKC antibodies, ANA, ANCA (C-ANCA and P-ANCA), rheumatoid factor, and serum ACE were negative. Active vitamin B12, folate, copper, and ceruloplasmin levels were normal. Genetic testing for common spinocerebellar ataxias (including SCA1, 2, 3, 6, 7, 8, 17) did not reveal pathogenic repeat expansions.

Serum protein electrophoresis demonstrated polyclonal hypergammaglobulinemia (gamma globulin 26 g/L) without monoclonal paraprotein on immunofixation.

Lumbar puncture demonstrated elevated cerebrospinal fluid (CSF) protein (770 mg/L; reference 150–450) with normal glucose and minimal pleocytosis (5 white cells/mm³). Cytology showed no malignant cells, and microbiological studies (bacterial, viral, fungal, and mycobacterial) were negative. Oligoclonal bands were not tested (add if available).

Imaging

- Brain MRI (September 2024): globally reduced cerebellar volume below the 10th percentile for age and sex, without focal lesion or mass effect (Figure 1).
- Repeat MRI brain with contrast and MR angiography (February 2026): minimal small-vessel ischemic change without diffusion restriction, pathological enhancement, or radiological evidence of active cerebellitis (Figures 2,3).
- Contrast-enhanced CT chest, abdomen, and pelvis: a 3 mm left upper lobe fissural pulmonary nodule, a 6 mm right upper lobe subpleural pulmonary nodule, and a right perihilar lymph node measuring 14 × 11 mm. No suspicious pulmonary mass, significant mediastinal or abdominal lymphadenopathy, or intra-abdominal malignancy was identified (Figure 4).

Differential Diagnosis

In a patient presenting with progressive adult-onset cerebellar ataxia, several differential diagnoses were considered.

- Autoimmune cerebellar ataxia associated with anti-VGCC antibodies was strongly considered given the progressive cerebellar syndrome and antibody positivity [1]. Immune-mediated cerebellar ataxias are recognized causes of subacute or progressive ataxia and may respond to early immunotherapy [1,7,8].
- Paraneoplastic cerebellar degeneration was also considered due to the established association between anti-VGCC antibodies and paraneoplastic neurological syndromes, particularly SCLC [2,4,5]. Although initial malignancy screening was negative, delayed tumour detection is

recognized, necessitating structured longitudinal surveillance [5].

- Vestibular migraine and peripheral vestibular disorders were considered given the prominent vertigo and tinnitus. However, the presence of persistent cerebellar signs, including dysmetria and gait ataxia, supported a central rather than peripheral process [3].
- Post-infectious cerebellitis was considered; however, the chronic, progressive course over three years and the absence of a preceding infectious illness made this unlikely [1].
- Hereditary spinocerebellar ataxia was also considered, given the patient's age at presentation. However, the absence of a family history and the presence of antibody positivity and inflammatory cerebrospinal fluid findings favored an acquired immune-mediated cause [3].

Overall, the clinical features, antibody profile, and exclusion of structural, genetic, and infectious causes supported a diagnosis of immune-mediated cerebellar ataxia [1,8,9].

Management

The patient commenced intravenous immunoglobulin (IVIG) therapy (Privigen, 2 g/kg divided over 5 days), with appropriate pre-medication (paracetamol, antihistamine) and monitoring of vital signs. Baseline and interval laboratory testing, including full blood count, renal function, and electrolytes, was arranged to monitor for IVIG-related adverse effects (e.g., thrombosis, acute kidney injury, aseptic meningitis). Given the progressive cerebellar syndrome characterized by gait ataxia, imbalance worse in darkness, motion sensitivity, and head-movement–provoked dizziness, falls precautions were instituted (e.g., non-slip footwear, walking aids, home environment assessment). Multidisciplinary management, including physiotherapy and occupational therapy, was arranged to optimize balance, improve gait stability, and reduce fall risk. A structured vestibular rehabilitation program was initiated once the patient was clinically stable. In view of the association between anti-VGCC antibodies and paraneoplastic neurological syndromes, ongoing malignancy surveillance was planned. Although initial CT chest imaging did not demonstrate malignancy, the presence of small pulmonary nodules and a right perihilar lymph node warranted interval imaging. Consideration of FDG-PET/CT scanning or short-interval CT chest follow-up (e.g., at 3–6 months) was arranged in accordance with recommendations for paraneoplastic syndromes [5]. Given the antibody profile, evaluation for clinical features suggestive of Lambert–Eaton myasthenic syndrome (LEMS) was also undertaken, including assessment of proximal muscle weakness, hyporeflexia, and autonomic symptoms; no clear neuromuscular junction features were identified at this stage. Repetitive nerve stimulation was not performed but could be considered in future if symptoms emerge.

Outcome

Following completion of IVIG loading therapy, the patient demonstrated partial symptom stabilization. There was subjective improvement in motion sensitivity and a reduction in the severity of dizziness with head movement. Gait instability persisted, but fall frequency decreased with rehabilitation input. No new focal neurological deficits developed during admission. There were no significant IVIG-related complications (e.g., no headache, thrombosis, or renal impairment).

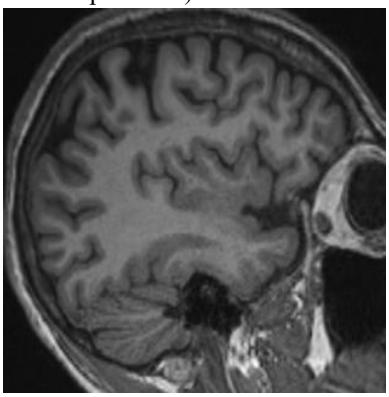


Figure 1: *MRI Brain) – Axial T1-weighted image demonstrating globally reduced cerebellar volume below the 10th percentile for age and sex, with no focal lesion or mass effect.*



Figure 2: *MRI Brain) – Axial FLAIR image showing minimal small-vessel ischemic change in the periventricular white matter, with no signs of active cerebellitis or diffusion restriction.*

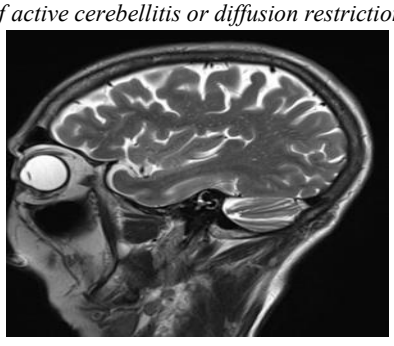


Figure 3: *MRI Brain) – Coronal T2-weighted image confirming absence of cerebellar oedema, enhancement, or acute inflammatory changes.*



Figure 4: *High-resolution CT Chest – Axial image showing a right perihilar lymph node measuring 14 × 11 mm (arrow) and small subpleural pulmonary nodules bilaterally. No definitive pulmonary mass or mediastinal lymphadenopathy.*

Follow-Up Plan

Close neurological follow-up has been arranged at 3-month intervals to assess clinical response, including serial evaluation of gait stability, limb coordination, nystagmus, and functional independence (e.g., SARA scale or ICARS if available). Ongoing assessment will determine the need for maintenance immunotherapy (e.g., repeat IVIG every 4–8 weeks or alternative agents such as rituximab if refractory). Serial malignancy surveillance will continue, recognizing that tumour detection in paraneoplastic syndromes may occur months or years after neurological presentation. Interval chest imaging (CT chest at 6 months, then annually for at least 2–3 years) has been scheduled in line with risk profile and radiological recommendations [5]. FDG-PET/CT is under consideration if any nodule enlarges or new symptoms arise. Vestibular rehabilitation and physiotherapy will continue in the outpatient setting, with reassessment of balance confidence and fall risk. The patient has been advised regarding safety strategies in low-light environments and during rapid head movements. Long-term follow-up will focus on early identification of relapse, monitoring for additional autoimmune manifestations (e.g., LEMS, thyroid disease), and periodic reassessment of paraneoplastic risk.

Discussion

Autoimmune cerebellar ataxia is an important and potentially treatable cause of progressive imbalance in adults. Unlike degenerative ataxias, immune-mediated cerebellar syndromes may stabilize or partially improve with timely immunotherapy [1,8]. Early recognition is therefore critical to limit irreversible neuronal injury. Anti–voltage-gated calcium channel (VGCC) antibodies are classically associated with paraneoplastic neurological syndromes and Lambert–Eaton myasthenic syndrome, particularly in the context of small-cell lung carcinoma [2,4]. However, cerebellar dysfunction may occur in isolation or precede neuromuscular junction involvement. These antibodies impair presynaptic calcium influx, disrupting cerebellar synaptic transmission and contributing to Purkinje cell dysfunction and progressive ataxia [2]. Neuroimaging considerations: The initial MRI, which included automated volumetric analysis, reported cerebellar volume below the 10th percentile for age and sex. A subsequent contrast-enhanced MRI did not demonstrate features of active cerebellitis or acute intracranial pathology. The absence of radiological evidence of active inflammation does not exclude immune-mediated cerebellar dysfunction. Variations between studies may reflect differences in imaging technique, volumetric methodology, or reporting emphasis rather than definitive structural change [1,4]. Notably, chronic immune-mediated injury may present with atrophy without gadolinium enhancement. Cerebrospinal fluid findings supported an inflammatory process. Although CSF abnormalities in autoimmune cerebellar ataxia are frequently non-specific (elevated protein with mild pleocytosis), they help exclude infection and support an immune-mediated aetiology [1]. Extensive evaluation excluded degenerative, metabolic, and genetic causes of adult-onset ataxia, strengthening the likelihood of antibody-mediated cerebellar dysfunction [3].

Paraneoplastic surveillance: Given the recognized association between VGCC antibodies and paraneoplastic syndromes, malignancy screening is essential. Paraneoplastic neurological syndromes may precede tumour detection by months or years, and negative initial imaging does not exclude occult malignancy [4,5]. In this case, CT chest imaging identified small pulmonary nodules and a right perihilar lymph node that were not diagnostic of malignancy but warrant structured longitudinal surveillance. Updated diagnostic criteria emphasise the importance of ongoing tumour screening in antibody-positive patients [5,6]. The presence of thyroid antibodies in this patient is likely coincidental, as autoimmune thyroid disease is common in older adults and does not directly explain the cerebellar syndrome. Immunotherapy remains the cornerstone of management in immune-mediated cerebellar ataxia [1,8]. Early initiation of corticosteroids, intravenous immunoglobulin, or plasma exchange may stabilize symptoms and improve functional outcomes. In this patient, IVIG was chosen due to its favorable safety profile in older adults and absence of contraindications. The partial clinical stabilization observed is consistent with published case series. Rehabilitation: Multidisciplinary rehabilitation, including physiotherapy and vestibular rehabilitation, plays a critical role in reducing falls and maintaining independence [1].

Limitations (added): This is a single case report, and thus findings may not be generalizable. Long-term outcome data beyond the initial treatment course are not yet available. CSF oligoclonal bands and repetitive nerve stimulation were not performed, which could provide additional supportive evidence

Conclusion

This case demonstrates anti–voltage-gated calcium channel antibody–associated autoimmune cerebellar ataxia presenting with progressive gait imbalance and central vestibular symptoms. This report reinforces the importance of considering autoimmune etiologies in adult-onset progressive ataxia, even when neuroimaging findings are subtle or non-specific. It also highlights the need for structured malignancy surveillance in antibody-positive patients and supports early initiation of immunotherapy, along with multidisciplinary rehabilitation, to optimize functional outcomes.

Learning Points

- Autoimmune cerebellar ataxia is an important and potentially treatable cause of progressive imbalance in adults and should be considered in subacute or unexplained cerebellar syndromes.
- Anti–voltage-gated calcium channel (VGCC) antibodies may present with predominant cerebellar dysfunction, even in the absence of overt neuromuscular junction features.
- Normal or non-specific neuroimaging does not exclude immune-mediated cerebellar pathology: structural changes may be subtle or reflect chronic injury rather than active inflammation.
- Detection of neural antibodies should prompt structured malignancy screening and longitudinal surveillance, as paraneoplastic neurological syndromes may precede tumour diagnosis.
- Early immunotherapy combined with multidisciplinary rehabilitation may stabilize symptoms and reduce functional decline.

Conflict of Interest Statement

None declared.

Patient consent

Informed consent obtained from the patient for publication of this case report.

References

1. Hadjivassiliou M, Martindale J, Shanmugarajah P. Immune-mediated cerebellar ataxias. Lancet Neurol. 2015; 14: 91-104.
2. Lennon VA, Kryzer TJ, Griesmann GE, O`Suilleabhain PE, Windebank AJ, Woppmann A, et al. Calcium-channel antibodies in Lambert–Eaton syndrome. N Engl J Med. 1995; 332: 1467-1474.
3. Klockgether T. Sporadic adult-onset ataxia. Lancet Neurol. 2010; 9: 94-104.
4. Darnell RB, Posner JB. Paraneoplastic syndromes involving the nervous system. N Engl J Med. 2003; 349: 1543-1554.
5. Graus F, Vogrig A, Muniz-Castrillo S, Jean-Christophe GA, Desestret V, Dubey D, et al. Updated diagnostic criteria for paraneoplastic neurologic syndromes. Lancet Neurol. 2021; 20: 754-765.
6. Honnorat J, Antoine JC. Paraneoplastic neurological syndromes. Orphanet J Rare Dis. 2007; 2: 22.
7. Lancaster E, Dalmau J. Neuronal autoantigens and autoimmune encephalitis. Lancet Neurol. 2012; 11: 1006-1018.
8. Mitoma H, Manto M, Hampe CS. Immune-mediated cerebellar ataxias. Cerebellum. 2016; 15: 213-232.
9. Shams'ili S, Grefkens J, de Leeuw B, den Bent MV, Hooijkaas H, der Holt BV, et al. Paraneoplastic cerebellar degeneration: clinical features and treatment outcomes. Brain. 2003; 126: 1409-1418.