

First Reported Coexistence of Acetylcholine Receptor-Positive Myasthenia Gravis and Genetically Confirmed Spinocerebellar Ataxia Type 6

Review began 09/30/2026

Review ended 10/12/2026

Published 10/17/2026

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DOI: 10.7759/cureus.118085

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Abstract

When a patient with a long-standing neurological diagnosis develops new symptoms, those symptoms are frequently attributed to the disease that is already known, and a second, unrelated disorder may remain unrecognized for years. We describe a patient in whom myasthenia gravis (MG) and spinocerebellar ataxia type 6 (SCA6) occurred together, a combination that has not, to our knowledge, been previously reported.

A 57-year-old man with acetylcholine receptor antibody-positive MG diagnosed in 1983 presented with progressive imbalance despite largely stable MG symptoms. His MG remained well controlled on low-dose prednisone and pyridostigmine following thymectomy, with intermittent azathioprine therapy. Neurological examination demonstrated cerebellar signs including dysmetria, dysidiadochokinesia, gaze-evoked nystagmus, and dysarthria. Brain MRI showed atrophy of the cerebellar hemispheres and vermis with associated brainstem volume loss. Extensive evaluation for acquired causes of cerebellar ataxia/degeneration, including paraneoplastic and autoimmune antibody testing, was unrevealing. Progressive ataxia continued despite largely stable neuromuscular symptoms. Family history revealed ataxia in his father and a similar disorder in his identical twin brother. Several years before the diagnosis was established, he experienced an MG exacerbation with bulbar and generalized weakness that responded to plasmapheresis. Genetic testing subsequently identified a pathogenic CACNA1A CAG repeat expansion of 22 repeats, confirming SCA6, just over a decade after the initial onset of his ataxia symptoms and establishing the coexistence of two distinct and rare neurological disorders.

This case highlights the diagnostic challenge of evaluating new neurological symptoms in patients with established chronic, rare neurological disease. Progressive cerebellar dysfunction was initially overshadowed by the patient's longstanding MG, resulting in delayed recognition of a hereditary ataxia. Clinicians should maintain a broad differential diagnosis when symptoms evolve in a manner that is atypical for the known disease process. Recognition of coexisting neurological disorders is essential for accurate diagnosis, prognostication, genetic counseling, and multidisciplinary management. Coexistence of an acquired neuromuscular disorder and a hereditary cerebellar ataxia is possible; antibody testing to exclude an acquired ataxia and genetic confirmation of the inherited disorder are both required, and recognition of both diagnoses directly informs immunotherapy decisions, rehabilitation, and family counselling.

Categories: Neurology, Psychiatry, Neurosurgery

Keywords: acetylcholine receptor antibodies, cacna1a, cerebellar ataxia, diagnostic overshadowing, myasthenia gravis, sca6, spinocerebellar ataxia type 6

Introduction

Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction characterized by fluctuating and fatigable skeletal muscle weakness resulting from antibodies directed against postsynaptic proteins, most commonly the acetylcholine receptor (AChR) [1,2]. Clinical manifestations may involve ocular, bulbar, limb, and respiratory muscles, and diagnosis is supported by characteristic clinical findings, electrophysiologic studies, and autoantibody testing [1,2]. The reported incidence of MG ranges from approximately 4.1 to 30 cases per million person-years, with a prevalence of approximately 150 to 200 cases per million [1]. Extraocular muscle involvement is the presenting feature in approximately two-thirds of patients, usually progressing to involve bulbar and limb musculature [2]. Pyridostigmine remains the first-line symptomatic treatment, most patients with generalized disease additionally require immunosuppression, and thymectomy is recommended in selected patients [3]. MG also carries an increased risk of concomitant autoimmune disease, most commonly autoimmune thyroid disease, systemic lupus erythematosus, and rheumatoid arthritis [4,5].

Spinocerebellar ataxia type 6 (SCA6) is a rare autosomal dominant neurodegenerative disorder caused by a

How to cite this article

Abou Zeid J, Shin K, Sleiman P, et al. (October 17, 2026) First Reported Coexistence of Acetylcholine Receptor-Positive Myasthenia Gravis and Genetically Confirmed Spinocerebellar Ataxia Type 6. Cureus 18(10): e118085. DOI 10.7759/cureus.118085

CAG trinucleotide repeat expansion in the CACNA1A gene, which encodes the $\alpha 1A$ subunit of the P/Q-type voltage-gated calcium channel [6,7]. SCA6 typically presents in adulthood with slowly progressive gait ataxia, dysarthria, limb incoordination, nystagmus, and other cerebellar manifestations [7]. Normal CACNA1A alleles contain four to 18 CAG repeats, whereas pathogenic alleles contain approximately 19 to 33 repeats [6,8]. The estimated worldwide prevalence of SCA6 is fewer than one per 100,000 individuals, and the reported age of onset ranges from 19 to 73 years, with a mean between 43 and 52 years [7]. Gait unsteadiness and imbalance are the initial symptoms in approximately 90% of patients, while dysarthria, upper-limb incoordination, intention tremor, and dysphagia develop as the disease progresses [7]. Neuropathologically, SCA6 is characterized by diffuse loss of Purkinje cells, predominantly involving the cerebellar vermis [6]. No disease-modifying therapy is currently available, and diagnosis rests on recognition of the clinical phenotype followed by targeted repeat-expansion testing [9].

Because a number of clinical features are shared by these two disorders, the presence of one may obscure recognition of the other. Dysarthria, dysphagia, gait impairment, and limb weakness may arise either from failure of neuromuscular transmission or from cerebellar degeneration [1,2,7]. These overlapping features create the conditions for diagnostic overshadowing, in which new or evolving symptoms are misattributed to an already established diagnosis and recognition of a second condition is delayed [10].

A literature search did not identify any previously published cases of genetically confirmed SCA6 occurring in a patient with AChR antibody-positive MG. We report a patient with longstanding AChR antibody-positive MG who developed progressive cerebellar dysfunction and was ultimately found to have genetically confirmed SCA6, highlighting the importance of reconsidering the differential diagnosis when neurological symptoms evolve beyond those expected from a known disease process.

Case Presentation

A 57-year-old man with AChR antibody-positive MG, diagnosed in 1983, presented with progressive imbalance. His MG had remained well controlled on prednisone 10 mg daily and pyridostigmine. He had previously undergone thymectomy in approximately 1988 and had been intermittently treated with azathioprine.

Because the clinical course of this patient extended over almost four decades, the sequence of events is presented as a longitudinal timeline indexed by calendar year and patient age rather than by day of admission (Table 1).

Time point	Clinical event	Findings and investigations	Management
1983	Diagnosis of acetylcholine receptor antibody-positive myasthenia gravis	Positive acetylcholine receptor antibodies	Pyridostigmine; prednisone
c. 1988	Thymectomy	Not applicable	Intermittent azathioprine
Age 57	Presentation with progressive imbalance despite largely stable myasthenic symptoms	Persistently positive acetylcholine receptor and striated muscle antibodies; normal thiamine; negative serum and cerebrospinal fluid autoimmune and paraneoplastic antibodies	Prednisone 10 mg daily and pyridostigmine continued
Age 61	Admission for worsening gait instability	Dysmetria on finger-to-nose and heel-to-shin testing, dysdiadochokinesia, gaze-evoked nystagmus and dysarthria, without fatigable weakness; brain MRI showing cerebellar hemispheric and vermian atrophy with brainstem volume loss (Figures 1 and 2)	Supportive management
Age 64	Family history obtained; myasthenic exacerbation with bulbar and generalized fatigable weakness	Father affected by ataxia; identical twin brother with a similar progressive ataxic disorder	Five sessions of plasmapheresis; azathioprine and maintenance-dose prednisone
Age 68	Admission for abdominal pain and suspected complicated urinary tract infection; genetic testing performed	Incidental complete heart block, considered likely related to pyridostigmine; pathogenic CACNA1A CAG repeat expansion (22 repeats) confirming SCA6	Permanent pacemaker; myasthenia optimized before surgery; multidisciplinary care

TABLE 1: Longitudinal Clinical Timeline of Myasthenia Gravis and Spinocerebellar Ataxia Type 6 in the Present Patient.

Initial evaluation demonstrated persistently positive AChR and striated muscle antibodies. Thiamine levels were normal. Comprehensive antibody testing was performed on both serum and CSF to evaluate for a potential autoimmune or paraneoplastic cerebellar ataxia. These included neuronal nuclear (ANNA1 and 2), Purkinje cell, amphiphysin, CRMP-5, and voltage-gated calcium channel antibodies, all of which were negative.

The laboratory and genetic investigations performed to exclude acquired causes of cerebellar ataxia, together with their reference ranges and the purpose of each test, are summarized in Table 2.

Test	Patient result	Units	Reference range	Purpose
Acetylcholine receptor binding antibody (serum)	Positive, persistently	nmol/L	Negative (below assay cut-off)	Confirmation of acetylcholine receptor antibody-positive myasthenia gravis
Striated muscle antibody (serum)	Positive, persistently	Titre	Negative	Supportive of myasthenia gravis; recognized association with thymoma
Thiamine (serum)	Within reference range	nmol/L	Within laboratory reference range	Exclusion of nutritional (thiamine deficiency) cerebellar dysfunction
Paraneoplastic and autoimmune cerebellar antibody panel (serum): ANNA-1, ANNA-2, Purkinje cell, amphiphysin, CRMP-5	Negative	Titre	Negative	Exclusion of paraneoplastic and antibody-mediated autoimmune cerebellar degeneration
P/Q-type voltage-gated calcium channel antibody (serum)	Negative	nmol/L	Negative	Exclusion of an acquired, antibody-mediated disorder of the same calcium channel encoded by CACNA1A
Cerebrospinal fluid antibody panel: antibodies as listed above	Negative	Titre	Negative	Exclusion of an intrathecal autoimmune or paraneoplastic cerebellar process
CACNA1A CAG repeat analysis (whole blood)	Expanded allele 22 CAG repeats; normal allele 11 CAG repeats	CAG repeats	Normal 4-18; pathogenic 19-33 [6,8]	Confirmation of spinocerebellar ataxia type 6

TABLE 2: Laboratory and Genetic Investigations, With Units, Reference Ranges and Purpose of Each Test

Despite largely stable MG symptoms, the patient experienced gradually progressive gait impairment and ataxia. At age 61, he was admitted for worsening gait instability. Neurological examination demonstrated dysmetria on finger-to-nose and heel-to-shin testing, dysdiadochokinesia, gaze-evoked nystagmus, and dysarthria, consistent with cerebellar dysfunction. The absence of fatigable weakness suggested a cerebellar process rather than MG exacerbation. Brain MRI demonstrated cerebellar hemispheric and vermian atrophy with associated brainstem volume loss (Figures 1, 2).

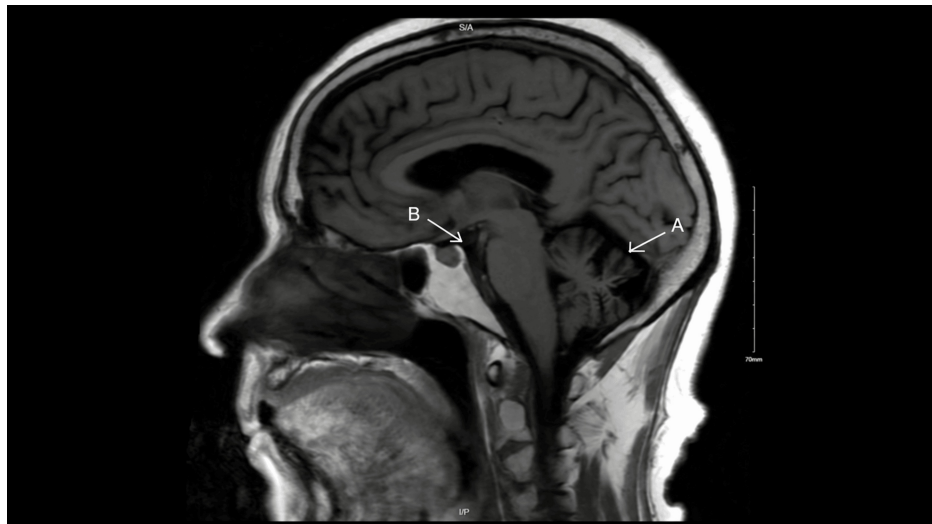


FIGURE 1: Sagittal T1-weighted MRI of the brain

Arrow A indicates marked atrophy of the cerebellar vermis, with thinning of the folia and widening of the interfolial cerebrospinal fluid spaces. Arrow B indicates volume loss of the pons and ventral brainstem, with prominence of the adjacent prepontine cerebrospinal fluid space.

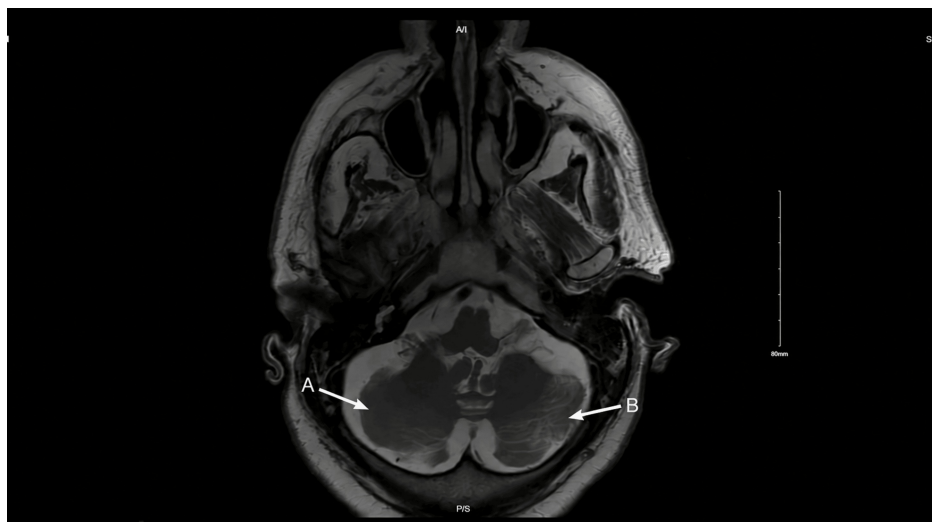


FIGURE 2: Axial T2-Weighted MRI of the Brain at Level of Posterior Fossa

Arrow A indicates atrophy of the right cerebellar hemisphere and arrow B the left cerebellar hemisphere, with narrowed folia, widened interfolial cerebrospinal fluid spaces, and enlargement of the surrounding cerebellar sulci and cisterns.

At age 64, further history revealed that his father had suffered from ataxia and that his identical twin brother had recently been diagnosed with a similar progressive ataxic disorder, raising suspicion for a hereditary cerebellar syndrome. Around this time, the patient also experienced an MG exacerbation with bulbar involvement and generalized fatigable weakness, which improved with five sessions of plasmapheresis. Over the following years, his MG symptoms remained well controlled on azathioprine and maintenance-dose prednisone, while his ataxia continued to progress.

At age 68, he presented with abdominal pain and was evaluated for a suspected complicated urinary tract infection. During this admission, he was incidentally found to have complete heart block, considered likely to be related to his pyridostigmine use, a recognized although uncommon cardiac conduction effect of cholinesterase inhibition [11], and cardiology successfully placed a permanent pacemaker. Neurology was consulted for peri-operative management of his MG. The neurology team managed the MG in preparation for surgery, but also recognized that most of his disability was from his ataxia. They obtained the history of

his longstanding progressive cerebellar syndrome and family history of ataxia. Genetic testing identified a pathogenic CAG repeat expansion in CACNA1A, confirming the diagnosis of SCA6. The expanded allele contained 22 CAG repeats, with a normal allele of 11 repeats.

Discussion

To our knowledge, the coexistence of genetically confirmed SCA6 and AChR antibody-positive MG has not been reported previously. Rare neurological comorbidities have been described in patients with spinocerebellar ataxias, including associations between SCA2 and amyotrophic lateral sclerosis (ALS) [12,13], but no comparable association involving SCA6 and MG has been documented.

The coexistence of MG and SCA6 in a single patient is possible and, given the rarity of both disorders, is best understood as a chance association. MG has an estimated prevalence of 150 to 200 per million [1], while SCA6 is estimated to affect fewer than one per 100,000 individuals [7]. One disorder is acquired and antibody-mediated and arises at the neuromuscular junction, while the other is inherited and degenerative and arises from a CAG repeat expansion in CACNA1A affecting the α1A subunit of the P/Q-type voltage-gated calcium channel and its associated transcription factor [6,8]. No shared pathogenic mechanism is currently recognized, and the occurrence of both conditions in our patient is therefore most plausibly coincidental. Recognizing that such coexistence is possible is nonetheless clinically important, because it is precisely when two disorders are assumed to be mutually exclusive that the second diagnosis is delayed.

Concurrent neuromuscular and hereditary cerebellar disorders have been described previously, although the reported combinations differ from ours. Nanetti et al. described a patient with SCA2 who developed motor neuron symptoms and reviewed similar cases, concluding that motor neuron involvement may represent a manifestation of SCA2 rather than an unrelated comorbidity [13]. Ferrari et al. subsequently reported a case series in which spinocerebellar ataxia was associated with amyotrophic lateral sclerosis and proposed that intermediate ATXN2 repeat lengths may contribute to ALS risk [12]. Motor neuron loss has also been documented at autopsy in SCA6 [14]. These reports differ from the present case in two important respects. First, the neuromuscular component in those patients was degenerative and, in SCA2, mechanistically linked to the same repeat expansion, whereas the neuromuscular disorder in our patient was acquired, antibody-mediated, and genetically unrelated to CACNA1A. Second, in those reports the hereditary ataxia was recognized first and motor neuron involvement emerged subsequently, whereas in our patient the acquired disorder preceded the hereditary one by nearly four decades. A comparison of these reports is presented in Table 3.

Report	Disorders reported	Genetic confirmation	Key observation
Nanetti et al., 2009 [13]	SCA2 with motor neuron disease	ATXN2 CAG repeat expansion	Motor neuron involvement may be a manifestation of SCA2 rather than an independent comorbidity
Ohara et al., 2002 [14]	SCA6 with motor neuron loss	CACNA1A CAG repeat expansion	Motor neuron loss demonstrated at autopsy in genetically confirmed SCA6
Ferrari et al., 2024 [12]	Spinocerebellar ataxia with amyotrophic lateral sclerosis	ATXN2 CAG repeat expansion	Intermediate ATXN2 repeat lengths proposed as a risk factor for ALS
Present case	AChR antibody-positive myasthenia gravis with SCA6	CACNA1A CAG repeat expansion	An acquired, antibody-mediated neuromuscular disorder and a genetically unrelated hereditary cerebellar ataxia in the same patient; not previously reported

TABLE 3: Reported Coexistence of Neuromuscular and Hereditary Cerebellar Disorders Comparison.

ALS: amyotrophic lateral sclerosis, SCA6: spinocerebellar ataxia type 6, AChR: acetylcholine receptor

This case also illustrates the complementary value of antibody testing and genetic confirmation. Comprehensive autoimmune and paraneoplastic antibody testing in serum and CSF served to exclude an acquired cerebellar syndrome (Table 2), a necessary step because immunotherapy would have been indicated had such an antibody been identified. Testing for P/Q-type voltage-gated calcium channel antibodies was of particular relevance, since CACNA1A encodes the α1A subunit of this same channel [6,8]; a negative result argued against an acquired, antibody-mediated channel disorder in a patient who was ultimately found to

have an inherited disorder affecting the same channel. Genetic testing then established a definitive diagnosis that serological and imaging studies alone could not provide, concluded an investigation that had continued for several years, and permitted accurate prognostication and family counselling.

Recognition of both diagnoses has direct consequences for management. MG is a treatable disorder, and our patient's exacerbation responded to plasmapheresis and ongoing immunosuppression [2,3]; failure to distinguish myasthenic weakness from cerebellar dysfunction risks either unnecessary escalation of immunotherapy for symptoms that are cerebellar in origin or delayed treatment of a genuine exacerbation. SCA6, by contrast, has no disease-modifying therapy, and management is supportive, centred on physical and occupational therapy, mobility aids, fall prevention, speech therapy, and assessment of swallowing as dysphagia develops [7,9]. Physiotherapy has been shown to improve balance and ataxia severity in degenerative cerebellar disease and therefore remains a cornerstone of care [15]. The two conditions also interact in practical ways: long-term corticosteroid therapy and cholinesterase inhibition carry risks of their own, as illustrated by the complete heart block identified in our patient [11]; cardiac involvement, including conduction abnormalities, is recognized in MG more broadly [16]. Progressive cerebellar dysfunction compounds the functional impact of any residual myasthenic weakness. Long-term care therefore requires coordinated neuromuscular, ataxia, genetics, rehabilitation and, in this patient, cardiology input, together with continued surveillance for additional autoimmune comorbidity given the recognized risk in MG [4,5].

A particularly important aspect of this case is the diagnostic challenge posed by attributing new neurological symptoms to a pre-existing disease. Our patient had carried a diagnosis of MG for nearly four decades before SCA6 was recognized. Symptoms such as dysarthria, gait instability, imbalance, and limb weakness could initially be attributed to MG [1,2]. However, the progressive nature of his ataxia, the presence of cerebellar signs including dysmetria, dysdiadochokinesia, and gaze-evoked nystagmus, together with a significant family history of ataxia, suggested an alternative diagnosis. Subsequent genetic testing confirmed SCA6.

The distinction between cerebellar dysfunction and manifestations of MG is clinically important. Although dysarthria and gait impairment may occur in both conditions, objective cerebellar signs such as dysmetria, dysdiadochokinesia, and gaze-evoked nystagmus are not characteristic features of MG and should prompt evaluation for alternative neurological processes [1,2,7]. In our patient, progressive cerebellar symptoms persisted despite relatively stable MG, providing an important clue that a second diagnosis was present.

This case demonstrates the importance of reconsidering the diagnostic framework when clinical features are not fully explained by a known disease process. It is, therefore, necessary to be mindful of psychological phenomena and biases that may occur and lead to diagnostic overshadowing. Biases related to the concept of diagnostic overshadowing include anchoring, premature closure, and implicit biases, and these may lead to missed diagnoses or ineffective therapies [10]. For our patient, it is important to keep these biases in mind, as MG and SCA6 have overlapping symptoms. In patients with established MG, persistent or progressive cerebellar symptoms should prompt evaluation for hereditary ataxias and other neurodegenerative disorders. Failure to recognize a second neurological condition may delay diagnosis, prognostication, genetic counseling, and appropriate multidisciplinary management.

Autoimmune comorbidities are well recognized in MG, most commonly autoimmune thyroid disease, systemic lupus erythematosus, and rheumatoid arthritis [4]. However, associations between MG and inherited neurodegenerative disorders have rarely been reported. Current evidence does not support a shared pathogenic mechanism between MG and SCA6, and their coexistence in our patient is most likely coincidental. Nevertheless, reporting such cases broadens the spectrum of recognized neurological comorbidities and may assist clinicians in identifying similar presentations in the future.

Genetic testing also carries implications that extend beyond the patient. Because SCA6 is inherited in an autosomal dominant manner, first-degree relatives are at 50% risk, and the patient's children may benefit from genetic counselling before family planning; his identical twin brother, who has a similar progressive ataxic disorder, would be expected to carry the same expansion [7]. The expanded allele in our patient contained 22 CAG repeats, within but at the lower end of the reported pathogenic range of 19 to 33 [6,8], consistent with his comparatively late symptom onset. It should be noted that the CAG expansion in SCA6 is small and comparatively stable between generations, and marked anticipation is not a typical feature, in contrast to SCA1, SCA2, SCA3, and SCA7, in which larger and less stable expansions may produce earlier onset in successive generations [6–8]. Counselling should therefore be tailored to the specific repeat-expansion disorder rather than applying a single expectation across all of them.

Conclusions

This case illustrates the coexistence of two distinct neurological disorders with overlapping clinical manifestations. It highlights the importance of identifying an inherited cerebellar ataxia in a patient with longstanding MG whose progressive symptoms extended beyond the expected manifestations of neuromuscular junction disease. Clinicians should maintain a broad differential diagnosis when evaluating patients with established neurological disorders, particularly when symptoms evolve in a manner that is atypical for the known disease process.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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