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Neurological Disorders

Case Report

Concurrent acute sensorimotor axonal neuropathy and disseminated encephalitis associated with *Chlamydia pneumoniae* in an adult patient with anti-MOG and anti-sulfatide antibodies: a case report

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***Abstract*:** Acute disseminated encephalomyelitis and Guillain–Barré syndrome refer to post-infectious or post-vaccination inflammatory demyelinating disorders of central and peripheral nervous system, respectively. We report the case of a 60-year-old male patient presenting with irritability, gait difficulty, asymmetric quadriparesis (mostly in his left extremities), distal sensory loss for pain and temperature in left limbs, and reduced tendon reflexes in his upper limbs and absent in his lower limbs, following an upper respiratory tract infection, 3 weeks earlier. Brain magnetic resonance imaging revealed abnormal T2 signal and peripherally enhancing lesions in hemispheres, brainstem, and cerebellum. Nerve conduction studies were compatible with acute motor and sensory axonal neuropathy. Serology revealed positive IgM and IgG antibodies for *Chlamydia pneumoniae*, and he also tested positive for myelin oligodendrocyte glycoprotein (MOG) and sulfatide antibodies. Treatment with intravenous immunoglobulin and methylprednisolone led to clinical and radiological recovery within weeks. Even though several cases of combined central and peripheral demyelination have been reported before, it is the first case report with seropositive anti-sulfatide and anti-MOG acute sensorimotor axonal neuropathy and disseminated encephalitis associated with *C. pneumoniae.*

Keywords: ADEM, AMSAN, case report, *Chlamydia pneumoniae*, MOG, sulfatide

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Background

Acute disseminated encephalomyelitis (ADEM) and Guillain–Barré syndrome (GBS) refer to immune-mediated syndromes of the nervous system.[1,2] ADEM is a demyelinating disorder of the central nervous system (CNS) causing encephalopathy and multiple white matter lesions in the brain, brainstem, and/or spinal cord.[1] GBS is an inflammatory demyelinating disorder of the peripheral nervous system (PNS), targeting peripheral nerves and their spinal roots, causing progressive symmetrical motor weakness of more than one limb, with hyporeflexia or areflexia, often presented with several variants, such as acute axonal neuropathies.[2] Even though both entities share an acute post-infectious or post-vaccination inflammatory demyelinating pathogenesis, they represent distinct neurological disorders, while simultaneous co-occurrence of both disorders as an immune response to the same stimuli is very uncommon. A small number of patients presenting combined central and peripheral demyelinating syndrome have been reported in the literature, most commonly regarding pediatric population.[3–16]

The aim of our paper is to report a rare case of concurrent ADEM and acute sensorimotor axonal

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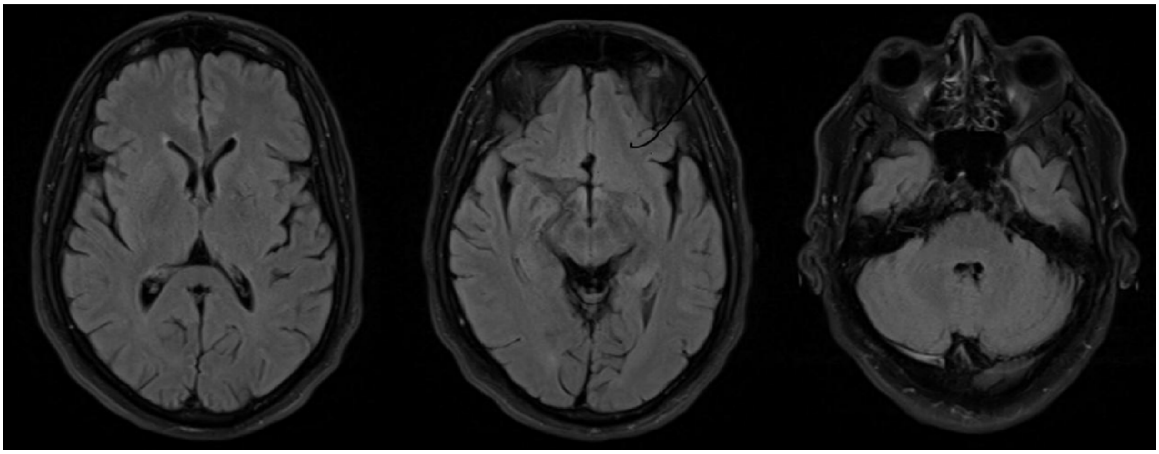

Figure 1. Axial brain magnetic resonance imaging T2 FLAIR sequences2 weeks prior patient's admission to our hospital without showing abnormal findings.
FLAIR, fluid-attenuated inversion recovery.

neuropathy (AMSAN) in an adult patient with anti-myelin oligodendrocyte glycoprotein (MOG) and anti-sulfatide antibodies after an upper respiratory infection, and enrich the existing literature regarding combined central and peripheral demyelination (CCPD) syndrome.

Case presentation

A 60-year-old man presented to the Emergency Department of our hospital with irritability, gait difficulty, generalized muscle weakness and numbness, mostly in his left extremities, for 2 days. He was not on medications and had no medical history (non-smoker, no alcohol, or psychotropic substances abuse). He only reported an admission to another hospital for 7 days due to an upper respiratory tract infection 3 weeks ago. A brain magnetic resonance imaging (MRI) with gadolinium and neurological examination were performed during his admission to the other hospital, due to reported headache, and provided to us. His brain MRI (Figure 1) and neurological examination were reported normal [normal orientation, mental status, muscle strength (muscle power scale 5/5 in all extremities), coordination, gait/posture, tendon reflexes, no meningeal signs, and modified Rankin Scale (mRS) 0]. No pathogen isolation was reported during his former hospitalization, and the patient was treated with 500 mg of intravenous (IV) azithromycin once daily for 5 days.

Two days before his admission to our hospital, he acknowledged symptoms of numbness and weakness in his left limbs, gradually expanding to all extremities. The same day that his symptoms began, he reported visiting a neurologist, who suggested the performance of a new brain MRI and reevaluation. Brain MRI was performed after 2 days and revealed abnormal T2 and fluid-attenuated inversion recovery (FLAIR) sequence signal lesions in both hemispheres and cerebellum, as well as peripherally enhancing lesions in hemispheres and cerebellum, in T1 with gadolinium sequences (Figure 2); thus, the patient was admitted to our hospital for further evaluation and treatment. Neurological examination revealed disorientation, drowsiness, asymmetric quadriparesis (reduced muscle strength in all four limbs, but mostly in his left extremities – Medical Research Council (MRC) scale 4/5 and 3/5, respectively), distal sensory loss for pain and temperature in left limbs, no meningeal signs, and no cranial nerves' lesions (mRS 4). Tendon reflexes were reduced in his upper limbs and absent in his lower limbs, without Babinski sign.

A lumbar puncture was performed and cerebrospinal fluid (CSF) studies showed mild pleocytosis of lymphocytic type with 12 white cells per cubic millimeter (ref: 0–5 cells/mm^3), markedly elevated protein (107 mg/dl, ref: 5–45 mg/dl), normal CSF/serum glucose ratio, negative culture, absence of oligoclonal bands, and IgG index 0.55. Nerve conduction studies (NCS) of motor

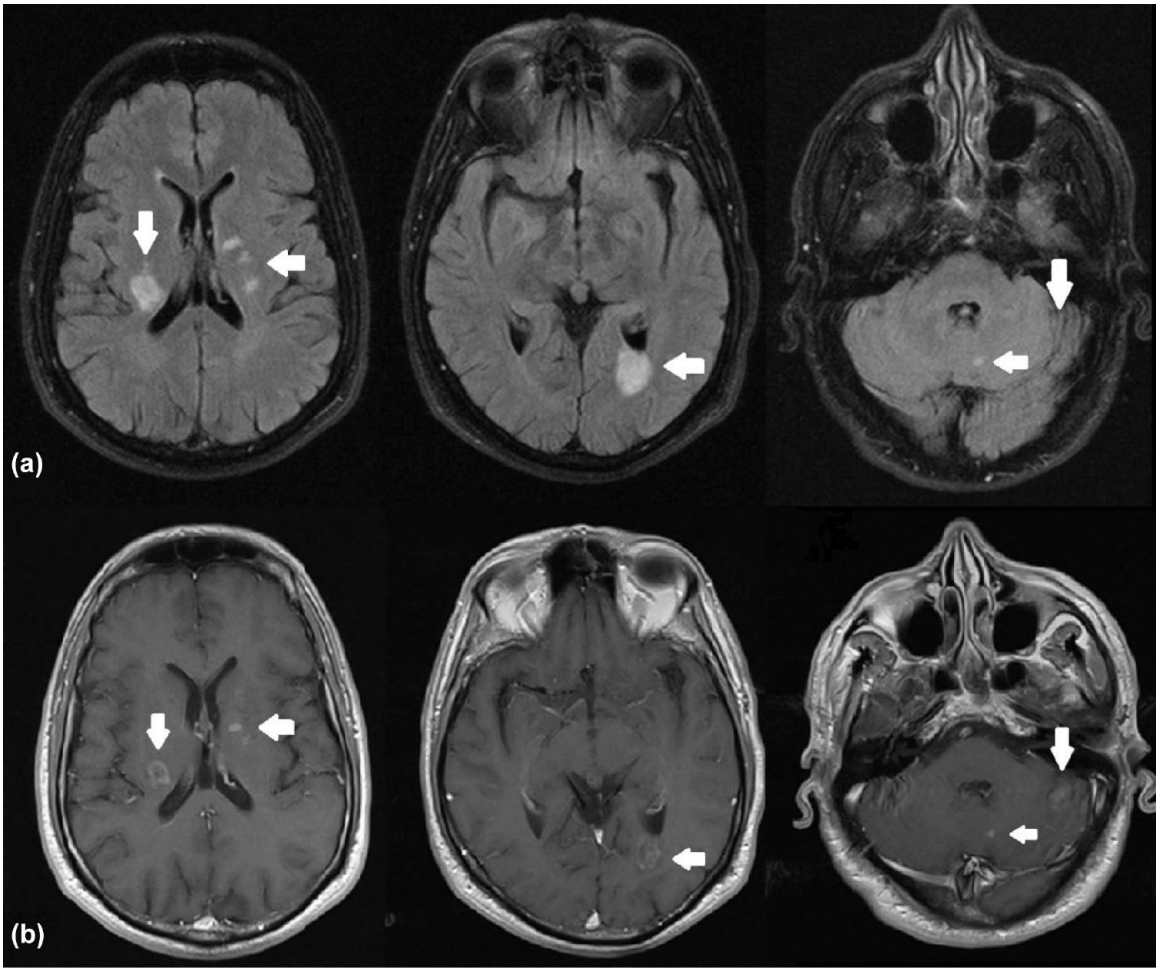

Figure 2. Axial brain magnetic resonance imaging the day of patient's admission to our hospital. (a) T2 FLAIR sequence showing abnormal T2 signal lesions in hemispheres and cerebellum. (b) T1 + gadolinium sequences showing peripherally enhancing lesions in hemispheres and cerebellum.
FLAIR, fluid-attenuated inversion recovery.

(median, ulnar, peroneal, and tibial) and sensory nerves (median, ulnar, radial, and sural) performed the day of his admission, using a Medtronic Keypoint Net apparatus showed only mildly lower amplitude of sensory nerve action potential (SNAP) in sural nerves. Spinal (cervical and thoracic) MRI was unremarkable. Patient underwent extensive laboratory testing for routine, vitamin, and autoimmune diagnostics (fasting blood glucose, Hb1Ac, urea, creatinine, ferritin, vitamin B12, thiamine, homocysteine, folate, thyroid stimulating hormone (TSH), protein and immune electrophoresis, anti-nuclear antibody, cryoglobulins, antineutrophil cytoplasmic antibodies, serum angiotensin-converting enzyme. Screening for infection pathogens was also performed, testing serology and polymerase chain reaction (serum and CSF) for herpes simplex virus 1 and 2, varicella-zoster virus, cytomegalovirus, West Nile virus, Epstein–Barr virus, enterovirus, adenovirus, influenza, echovirus, mumps, measles, rubella, *Chlamydia pneumoniae*, hepatitis B virus, hepatitis C virus, human immunodeficiency virus, *Campylobacter jejuni*, human herpes virus 6–7–8, and *Borrelia burgdorferi*), as well as chest and abdominal computerized tomography, tumor biomarkers, serum paraneoplastic antibodies (Hu, Yo, Tr, GAD, SOX1, CV2, PNMA2, recoverin, zic4, amphiphysin), which were all found negative, except *C. pneumoniae* serum IgM and IgG antibodies, which were found positive (28.3 and 59.7 U/ml, respectively; positive

Table 1. Nerve conduction studies performed 2 weeks after patient's admission.

Nerve (right side)	Amplitude (sensory uV–motor mV)	Velocity (m/s)	Latency (ms)	F wave latency (ms)
Sensory				
Sural reference	>10	>45	<3.5	–
Sural	3.2	50.3	2.1	–
Radial reference	>25	>50	<3.0	–
Radial	11.1	57.2	1.7	–
Median reference	>18	>50	<3.5	–
Median	9.3	53.2	3.1	–
Ulnar reference	>17	>50	<3.7	–
Ulnar	10.6	54.0	2.5	–
Motor				
Peroneal reference	>3	>42	<6.0	<47
Peroneal	0.76	44.8	3.7	43.5
Tibial reference	>5	>41	<6.5	<50
Tibial	0.89	43.6	4.6	45.6
Median reference	>7	>51	<4.0	<32
Median	3.1	56.0	3.0	28.4
Ulnar reference	>6	>51	<4.0	<35
Ulnar	4.3	57.1	2.8	23.9

Nerves of both sides have been studied with similar findings.

ref: >11 U/ml). No further antibiotic treatment was provided to our patient.

Under the suspicion of a post-infectious syndrome due to his history involving CNS and PNS, a serum specimen was tested for antibodies against MOG using fixed cell-based assay (anti-aquaporin-4 antibodies were not tested), myelin-associated glycoprotein (MAG) and gangliosides (GM1, GM2, GM3, GM4, GD1a, GD1b, GQ1b, GD2, GD2, GT1a, GT1b, and sulfatide) using Western blot, and was found positive for anti-MOG IgG (titer 1:80, positive ref: >1:10) and anti-sulfatide antibodies IgG (titer 1:1000, positive ref: >1:100); thus a diagnosis of concurrent ADEM and possible GBS, associated with *C. pneumoniae*, was made. Patient was treated with IV immunoglobulin (30 g per day for 5 days) and IV methylprednisolone (1000 mg per day for 3 days), followed by a calculated oral prednisone dosage (1 mg/kg per day), tapered slowly over 12 weeks.

NCS were repeated 2 weeks after his admission to our hospital and showed reduced amplitudes of SNAP of median, ulnar, radial, and sural nerves, as well as reduced amplitudes of compound muscle action potential (CMAP) in all tested motor nerves, normal F waves of median and tibial nerves, as seen in Table 1, compatible with acute motor and sensory axonal neuropathy (AMSAN). Before discharge, neurological examination revealed clinical improvement regarding his muscle strength and level of consciousness (mRS 2),

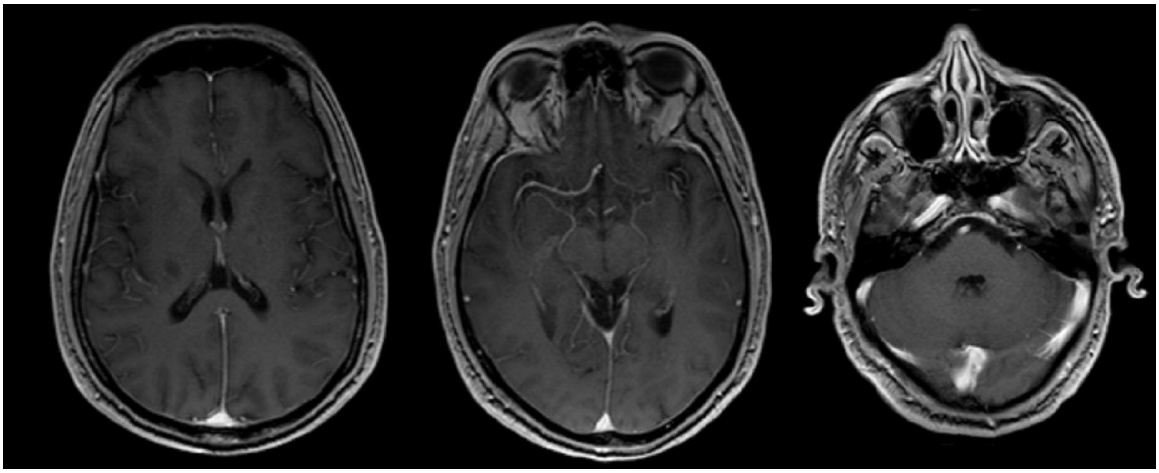

Figure 3. Axial brain magnetic resonance imaging T1 + gadolinium sequences 3 months after patient's discharge from our hospital, showing radiological improvement.

and the patient entered an intensive rehabilitation program for 3 months.

At 3-month follow-up, the patient was re-evaluated, and a remarkable clinical and radiological improvement was noted (Figure 3). His neurological examination revealed only reduced Achilles tendon reflexes and mild weakness (MRC scale 4+/5) in dorsiflexion of the left foot (mRS 1). NCS were repeated that day and showed increase in all prior reduced amplitudes of SNAPs and CMAPs. He was also retested for serum anti-MOG and anti-sulfatide antibodies and was found negative. No further immunomodulatory therapy was provided to the patient.

Conclusion

CCPD syndrome refers to a rare neurological disorder with simultaneous occurrence of CCPD.[3] Brain and spinal MRI scans, CSF tests and NCS studies are typically performed to confirm CCPD, while other laboratory tests are used to exclude other possible diagnoses, as performed in our patient.[3,4] Demyelinating disorders of CNS most commonly related to CCPD include multiple sclerosis (MS), neuromyelitis optica spectrum disorders (NMOSDs), and ADEM, while we found a small number of reports with confirmed MOG antibody-associated disease (MOGAD) and CCPD.[17–21] As regards PNS involvement, chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) has been most commonly associated with CCPD, while GBS variants [acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), AMSAN and conduction blocks] have also been described, even though AMAN and AMSAN variants refer to axonal damage and not demyelination.[3,10,12,13]

Anti-MOG disease is an autoimmune disorder in which the immune system mistakenly attacks MOG, a protein located on the surface of myelin, an insulating layer that surrounds nerve cell axons and enhances signal conduction between them.[22] The spectrum of demyelinating disorders with IgG antibodies to MOG, known as MOGAD, includes many core clinical phenotypes, such as optic neuritis, transverse myelitis, brainstem and/or cerebellar deficits, cerebral monofocal or polyfocal deficits, ADEM, and cerebral cortical encephalitis often with seizures.[23] Our patient reported signs, symptoms, and hospitalization for an upper respiratory tract infection 3 weeks prior to his admission to our hospital. Moreover, even though no lesions were detected in the spinal cord, brain MRI findings showed bilateral lesions with ill-defined borders and peripheral enhancement, which typically appear in ADEM-MOG encephalomyelitis.[24] Our patient was found seropositive for anti-MOG IgG antibodies and had a clinical course compatible with ADEM, fulfilling the newly proposed international MOGAD criteria,[25] as well as he did not fulfill diagnostic criteria for MS and NMOSD.[26,27]

Sulfatide (galactosylceramide-3-*O*-sulfate) represents the major acidic glycosphingolipid in central and peripheral nerve myelin sheath membrane, interacting with GalCer, in the presence of $Ca^{2}+$, which is highly dependent on ceramide composition of both GalCer and sulfatide.[28] Antibodies against sulfatide antigen have been reported in a variety of systemic disorders, such as diabetes, acquired immunodeficiency syndrome, idiopathic thrombocytopenic purpura, autoimmune chronic active hepatitis, and MS.[29,30] Furthermore, an association between highly elevated titers of these antibodies and peripheral neuropathy is well known.[31–40] These neuropathies refer to predominantly sensory or sensorimotor axonal neuropathies, small and large fiber sensory neuropathies, and acute and chronic inflammatory demyelinating polyradiculopathies, while patients with a concomitant IgM monoclonal gammopathy have also been reported.[41–44] In particular, high titers of anti-sulfatide antibodies have been observed in AIDP, CIDP, AMAN, and AMSAN; thus, it is considered that these antibodies could play an important role in the pathogenesis of neuropathy in patients with GBS.[33–36] Our patient had a concurrent PNS disease course with quadriparesis, distal sensory loss, and abolished osteotendinous reflexes, while repeated NCS showed ascending sensory–motor impairment, all responding to immunosuppressive treatment at his follow-up. Anti-sulfatide IgG antibodies were found seropositive, and he fulfilled diagnostic criteria for AMSAN variant of GBS.[45–47]

Chlamydia pneumoniae is a type of bacteria that causes respiratory infections in humans and represents one of the most common causes of community-acquired pneumonia. Serology is considered the preferred method for confirmatory laboratory diagnosis and empirical antibiotic therapy with a macrolide or a fluoroquinolone is considered sufficient to cure the illness.[48] In recent years, an increasing number of publications have reported the detection of *C. pneumoniae* in chronic extrarespiratory diseases, such as common neurological disorders (MS, stroke, Alzheimer's disease), which according to authors could be an irrelevant finding.[49,50] Interestingly, a small number of reports described the possible association between *C. pneumoniae* and ADEM or GBS, most commonly in younger patients with a favorable outcome after administration of antibiotic and/or immunosuppressive treatment, suggesting a strong etiologic link between the microorganism and neurological disorder, as seen in our patient.[50–54] In one of these cases,[54] anti-MOG antibodies were found positive in a child with acute and multiphasic disseminated encephalomyelitis and subclinical *C. pneumoniae* infection, and in a second one,[53] anti-ganglioside GM1 antibodies were found positive in a young woman with GBS following *C. pneumoniae* infection. In both cases, authors suggest the possible induction of these neurological disorders by *C. pneumoniae* and a rather underestimated association.

Molecular mimicry and cross-reactive autoimmune response to myelin protein antigens are considered the most likeable pathogenesis of both GBS (classic and variants) and ADEM.[1,2] However, another hypothesis proposed that an antibody-mediated post-infectious syndrome results to a continuous clinical spectrum involving both the PNS and CNS, suggesting that the responsible pathogen shares an antigen of both peripheral and central myelin, like in Fisher–Bickerstaff syndrome with anti-GQ1b antibodies.[55] Furthermore, a possible common pathogenic mechanism, suggesting that the immune response against a component of the myelin of the CNS may carry cross-antigenicity with the peripheral system, with a significant increase in activated and helper inducer T-cells in both GBS variants and ADEM, has been proposed by others authors.[11] Even though sulfatide antibodies are often associated with a concomitant reactivity to MAG and the rare selective re-activities to sulfatides associated with different forms of neuropathy, no strong association with MOG cross-antigenicity is well founded.[56,57] Nonetheless, our hypothesis was that *C. pneumoniae* infection induced concurrent AMSAN and ADEM, in our case, as a common antigen target for antibodies' production. Since *C. pneumoniae* infection could be associated with anti-MOG positive ADEM and anti-ganglioside positive axonal GBS variant, as discussed above, it is rather possible that it could trigger simultaneous anti-sulfatide AMSAN and anti-MOG ADEM, as seen in our patient, but the exact mechanism remains unknown.

Herein, we present a rare case of concurrent AMSAN and ADEM in an adult patient with a favorable outcome after immunosuppressive therapy. Even though several cases of CCPD have been reported before, to our knowledge, it is the first case report with concurrent AMSAN and ADEM with

both anti-sulfatide and anti-MOG antibodies associated with *C. pneumoniae*. Further research needs to be carried out to clarify the pathogenesis and possible correlation of these entities.

Declarations

Ethics approval and consent to participate

The publication of this case report was approved by the Scientific Committee of G. Gennimatas Hospital (number of approval: 19244; date: 18 July 2023).

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Author contributions

Michail Papantoniou: Conceptualization; Data curation; Investigation; Writing – original draft.

Grigorios Panagopoulos: Methodology; Writing – review & editing.

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Competing interests

The authors declare that there is no conflict of interest.

Availability of data and materials

The corresponding author takes full responsibility for the data, has full access to all the data, and has the right to publish any or all data separate and apart from any sponsor.

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