Immune-mediated necrotizing myopathy complicated by rhabdomyolysis syndrome and central nervous system cytomegalovirus infection: A case report

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Abstract. Immune-mediated necrotizing myopathy (IMNM) complicated by rhabdomyolysis (RM) syndrome and central nervous system (CNS) infection is extremely rare, difficult to diagnose and associated with a poor prognosis. The present study reports a case of anti-signal recognition particle (anti-SRP) antibody-positive IMNM complicated by RM and CNS cytomegalovirus (CMV) infection to provide a reference for its clinical diagnosis and treatment. The clinical data, laboratory findings, muscle pathology, imaging findings and electromyography results are retrospectively analyzed for a 19-year-old female patient who presented with progressive fatigue and muscle weakness, and the case is discussed in the context of the relevant literature. The patient's serum creatine kinase level peaked at 32,213 U/l, and anti-SRP antibodies were positive. Muscle biopsy revealed myogenic damage with muscle fiber necrosis and C5b-9 deposition. The diagnosis of IMNM was confirmed by muscle biopsy and strongly positive anti-SRP antibodies. Metagenomic next-generation sequencing of the cerebrospinal fluid detected CMV with a relative abundance of 100%; however, viral load quantification and read numbers were not available from the assay. Serum CMV-immunoglobulin (Ig)M (213.30 AU/ml; positive) and CMV-IgG (>1,000.00 AU/ml; positive) further supported active CMV infection. The patient's muscle strength progressively improved following combined immunotherapy, antiviral therapy and supportive care. At the 6-month follow-up, upper limb muscle strength had recovered to grade 4+, while lower limb muscle strength had recovered to grade 3. In conclusion, for patients presenting with rapidly progressive muscle weakness and increased muscle enzyme levels, myositis-specific antibodies should be tested for as early as possible. When complicated by a CNS infection, differentiation from Guillain-Barré syndrome is essential. Early comprehensive immunotherapy combined with antiviral therapy may improve the prognosis.

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Introduction

Immune-mediated necrotizing myopathy (IMNM) is a distinct subset of idiopathic inflammatory myopathy, characterized by proximal muscle weakness, markedly elevated serum creatine kinase levels and histopathological features of muscle fiber necrosis with minimal inflammatory infiltrate (1-3). IMNM is classified into three major subgroups: Anti-signal recognition particle (anti-SRP) antibody-positive, anti-3-hydroxy-3-methylglutaryl-coenzyme A reductase (anti-HMGCR) antibody-positive and seronegative IMNM (3). Among these, anti-SRP antibody-positive IMNM is associated with severe myonecrosis and often requires aggressive immunosuppressive therapy (2,3). Rhabdomyolysis (RM) syndrome is a potentially fatal complication of severe skeletal muscle injury, most commonly triggered by trauma, exertion, drugs or infection, but increasingly recognized as a rare yet clinically significant consequence of immune-mediated myopathies (4,5). When RM complicates IMNM, the resulting extensive myonecrosis portends a more complicated clinical course with a higher risk of systemic complications such as acute kidney injury (AKI) (6). Furthermore, cytomegalovirus (CMV), a ubiquitous herpesvirus that may reactivate under immunosuppression, can occasionally invade the central nervous system (CNS) (7). The coexistence of IMNM (which mandates aggressive immunosuppression) and CNS CMV infection therefore creates a formidable therapeutic dilemma, as these two conditions demand seemingly contradictory treatment strategies. To the best of our knowledge, the simultaneous

occurrence of anti-SRP antibody-positive IMNM, severe RM and CNS CMV infection in a single patient has rarely been reported. A recently published case similarly described a temporal association between anti-SRP IMNM and recent CMV infection, highlighting the potential interplay between viral triggers and autoimmunity (8). However, the scenario in which all three conditions coexist, and particularly the diagnostic challenge it poses in differentiating from Guillain-Barré syndrome (GBS), remains poorly characterized. The present case report therefore aims to describe the clinical presentation, diagnostic workup and multidisciplinary management of a 19-year-old female patient with anti-SRP antibody-positive IMNM complicated by RM and CNS CMV infection, with the hope of raising clinical awareness and informing timely diagnosis and treatment of this rare and complex combination.

Case report

First admission (February-March 2024)

Patient presentation. A 19-year-old female patient was admitted to the Affiliated Traditional Chinese Medicine Hospital, Southwest Medical University (Luzhou, China) in February 2024 due to an abnormal muscle enzyme profile, including increased serum creatine kinase (CK) levels of up to 31,180 U/l (reference value, 40-200 U/l), detected during a routine physical examination 20 days earlier. Initial treatment at a local hospital (Yibin, China) had resulted in no notable improvement. At 1 week before admission to the Affiliated Traditional Chinese Medicine Hospital, Southwest Medical University, the patient developed progressive muscle weakness and myalgia, which eventually progressed to an inability to ambulate.

The patient had no notable past medical history, with no history of statin use or autoimmune disease before the onset of the current symptoms. The patient had no notable personal history, had received no recent vaccinations and had not suffered any viral illnesses prior to symptom onset. There was no family history of hereditary muscle diseases or neuromuscular disorders.

Upon admission, vital signs were stable, and cardiopulmonary and abdominal examinations were unremarkable. Notably, respiratory and swallowing functions were intact, with no dyspnea, orthopnea or dysphagia. Muscle tone in all four limbs was normal. Muscle strength in the distal limbs was graded as 1/5 using the Medical Research Council scale (9), while muscle strength in the proximal limbs was nearly 0/5.

Key laboratory findings during hospitalization are summarized in Tables I-III. Notably, all time points mentioned in this report (e.g., 'day X') are calculated with February 2024 (the date of first hospital admission) defined as day 0. This chronological framework remains consistent throughout the patient's hospital course, including the intervening discharge and second admission, to provide a unified clinical timeline. On Day 0 (admission), routine examinations, including routine blood test, myocardial markers, kidney function, electrolytes, blood fat, myozymogram, routine urinalysis and thyroid function, were performed. Results revealed an elevated white blood cell count (13.12×10^9/l; reference range, 3.5-9.5x10^9/l), markedly elevated CK-MB mass (>287.00 ng/ml; reference range, 0.6-6.3 ng/ml) and myoglobin (Myo; >3,958.0 ng/ml; reference value, ≤65.8 ng/ml), as well as elevated aspartate aminotransferase (AST, 930 U/l; reference range, 13-35 U/l), alanine aminotransferase (ALT, 590 U/l; reference range, 7-40 U/l), lactate dehydrogenase (LDH, 2,267 U/l; reference range, 120-250 U/l), CK-MB (817 U/l; reference range, 0-25 U/l) and α-hydroxybutyrate dehydrogenase (1,898 U/l; reference range, 72-182 U/l), and a profound increase in CK (32,213 U/l; reference range, 40-200 U/l). Regarding RM-related complications, the following were documented: Serum Myo was markedly elevated (>3,958.0 ng/ml; reference value, ≤65.8 ng/ml), but routine urinalysis showed negative results for blood and protein, indicating no gross myoglobinuria at the time of testing, which was likely attributable to early aggressive fluid resuscitation. Electrolyte abnormalities included hyperphosphatemia (inorganic phosphate, 1.63 mmol/l; reference range, 0.85-1.51 mmol/l) and mild hyponatremia (sodium, 135 mmol/l; reference range, 137-147 mmol/l). The serum creatinine level was 37 μmol/l (reference, 41-73 μmol/l), which was below normal, but this remained stable throughout hospitalization, indicating no AKI and preserved renal function. The patient did not develop compartment syndrome. As AKI did not occur, conventional renal replacement therapy was not required, although plasma exchange was performed for immunomodulation. On day 24, autoantibody screening was conducted, showing positive (+++) results for both anti-SRP and anti-SSA/Ro-52 antibodies (Table II). On day 26, serum virology testing detected positive CMV-IgM and CMV-IgG. On day 29, cerebrospinal fluid (CSF) analysis was performed (Table III).

Histopathological examination. A right thigh muscle biopsy was performed with the following histopathological findings: The muscle fiber diameter ranged from 5 to 55 μm, with marked variability in fiber size. Muscle fibers of varying diameters were intermixed, and scattered degenerating, necrotic and regenerating muscle fibers were observed. Internalized nuclei were present. There was no definite lymphocytic aggregation. Mild connective tissue hyperplasia was observed in some muscle fascicles, along with moderate adipose tissue hyperplasia between some muscle fascicles. Tissues were fixed in 4% paraformaldehyde (for 24 h at 37˚) at a section thickness of 5-10 μm, and then stained with hematoxylin (for 10 min at 37˚) and eosin (for 3 min at 37˚). Images were acquired under a light microscope at x4 and x20 magnification (Fig. 1).

Special staining. Gomori staining (4% paraformaldehyde fixation for 24 h; 2% iron alum staining for 10 min and Gomori ammonia-silver staining for 3 min) showed histopathological changes consistent with those aforementioned. Nicotinamide adenine dinucleotide-tetrazolium reductase staining (fresh frozen sections, 7-μm thick; incubated in NADH-TR medium at 37˚C for 30 min in the dark) demonstrated aggregation or loss of enzyme activity in degenerating and necrotic muscle fibers. Periodic acid-Schiff staining (4% paraformaldehyde fixation for 24 h; Schiff reagent staining for 10-20 min in the dark at 37˚ and hematoxylin staining for 2 min) revealed no abnormalities in glycogen distribution within the muscle fibers. Oil Red O staining (fresh frozen sections, 10- to 15-μm thick with no fixation; Oil Red O working solution applied for 5-15 min and hematoxylin for 2 min) showed no abnormalities in lipid accumulation within the muscle fibers. Acid phosphatase

Table I. Key laboratory findings during hospitalization.

Laboratory examinations	Results	Normal values
Routine blood test		
WBCs ($x10^9$/l)	13.12	3.5-9.5
RBCs ($x10^{12}$/l)	5.37	3.8-5.1
HGB, g/l	157	115-110
GR, %	83.6	40-75
Myocardial marker		
CKMB-mass, ng/ml	>287.00	0.6-6.3
Myo, ng/ml	>3,958.0	≤65.8
hs-cTnT, ng/ml	0.084	0.000-0.0175
Kidney function		
SCr, μmol/l	37	41-73
Electrolytic		
Na, mmol/l	135	137-147
Cl, mmol/l	100	99-110
IP, mmol/l	1.63	0.85-1.51
Blood fat		
TG, mmol/l	4.07	<2.26
CHOL, mmol/l	6.36	≤5.2
HDL-C, mmol/l	1.12	>1.04
LDL-C, mmol/l	4.08	<3.34
Myozymogram		
AST, U/l	930	13-35
ALT, U/l	590	7-40
LDH, U/l	2,267	120-250
CK, U/l	32,213	40-200
CK-MB, U/l	817	0-25
α-HBDH, U/l	1,898	72-182
Thyroid function		
TSH, μIU/ml	5.910	0.25-4.20
FT3, pmo1/l	1.11	3.1-6.8
FT4, pmo1/l	24.40	12-22
Routine urinalysis		
BLD	Negative	Negative
LEU	Negative	Negative
PRO	Negative	Negative
Virus		
CMV antibody-IgM, AU/ml	213.30	<8
CMV antibody-IgG, AU/ml	>1,000.00	<10

WBC, white blood cell; RBC, red blood cell; HGB, hemoglobin; GR%, granulocyte; CKMB-Mass, creatine kinase-MB mass; Myo, myoglobin; hs-cTnT, high-sensitivity cardiac troponin T; SCr, serum creatinine; Na, sodium; Cl, chloride; IP, inorganic phosphate; TG, triglyceride; CHOL, cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Ig, immunoglobin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; CK, creatine kinase; α-HBD, α-hydroxybutyric dehydrogenase; TSH, thyrotropin; FT3, triiodothyronine; FT4, tetraiodothyronine; BLD, blood; LEU, leucocyte; PRO, protein; CMV, cytomegalovirus.

Table II. Examination of myositis spectrum and autoantibody spectrum of patients.

Autoantibodies	Result	Normal result
Anti-JO-1 antibody	Negative	Negative
Anti-PL-7 antibody	Negative	Negative
Anti-PL-12 antibody	Negative	Negative
Anti-EJ antibody	Negative	Negative
Anti-SRP antibody	Positive (+++)	Negative
Anti-Mi-2 antibody	Negative	Negative
Anti-MDA5 antibody	Negative	Negative
Anti-TIF1γ antibody	Negative	Negative
Anti-HMGCR antibody	Negative	Negative
Anti-SSA/Ro-52 antibody	Positive (+++)	Negative
Anti-SAE1/2 antibody	Negative	Negative
Anti-NXP2 antibody	Negative	Negative

Antibody positivity was graded as: + (weak), ++ (positive) or +++ (strong). SRP, signal recognition particle; JO-1, histidyl-tRNA synthetase; PL-7, threonyl-tRNA synthetase; PL-12, alanyl-tRNA synthetase; EJ, glycyl-tRNA synthetase; Mi-2, nucleosome remodeling deacetylase complex; MDA5, melanoma differentiation-associated gene 5; TIF1γ, transcriptional intermediary factor 1γ; HMGCR, 3-hydroxy-3-methylglutaryl-CoA reductase; SSA/Ro-52, Sjögren's syndrome antigen A/Ro52; SAE1/2, small ubiquitin-like modifier activating enzyme 1/2; NXP2, nuclear matrix protein 2.

staining (paraffin sections, 5- to 10-μm thick; pre-cooled ACP fixation for 10 min at 2-8˚C; ACP incubation medium applied at 37˚C for 50 min and hematoxylin for 2 min) demonstrated increased enzyme activity in degenerating and necrotic muscle fibers, inflammatory cells and some small blood vessels. All slides were examined under a light microscope at x4 and x20 magnification (Fig. 1).

Immunohistochemistry. Immunohistochemistry was performed on paraffin-embedded tissue sections at a 4-μm thickness with 4% paraformaldehyde fixation at 37˚C for 24 h. Blocking was performed with 5% BSA (Beijing Solarbio Science & Technology Co., Ltd.) at room temperature for 20 min, with subsequent incubation with the following primary antibodies: Anti-human leukocyte antigen (HLA) Class 1 ABC (rabbit monoclonal; 1:400; cat. no. EPR22172; Abcam) and Anti-C5b-9 (mouse monoclonal; 1:100; cat. no. ab55811; Abcam) at 4˚C for 24 h. Incubation was then performed with the following secondary antibodies: Goat Anti-Rabbit IgG H&L (HRP-conjugated; 1:500; cat. no. ab6721; Abcam) and Goat Anti-Mouse IgG H&L (HRP-conjugated; 1:500; cat. no. ab6789; Abcam) at 37˚C for 1 h. The results showed that HLA-ABC expression was weakly positive, and C5b-9 deposition was observed on the muscle fiber membranes (Fig. 1). All sections were examined under a light microscope at x4 and x20 magnification.

Pathological diagnosis. Overall, the skeletal muscle biopsy demonstrated necrotizing myopathy with macrophagic (histiocytic) infiltration and C5b-9 deposition, fulfilling the histopathological criteria of the European Neuromuscular Centre (ENMC) for IMNM.

Table III. Patient obtained CSF examination results through lumbar puncture.

Inspection item	Result	Normal result
Germ	Negative	Negative
Fungus	Negative	Negative
Parasite	Negative	Negative
Virus	Cytomegalovirus (mNGS; relative abundance 100%)	Negative
CSF albumin, mg/l	328	100-300
CSF total protein, mg/l	644	150-450
CSF pressure, mmH_2O	Not measured (natural drip rate ~60 drops/min)	80-180
WBC (x10^6/l)	1	0-8
HB (x10^6/l)	0	/
Glucose, mmol/l	3.79	2.22-3.89
Cl, mmol/l	127	120-132
MN, %	/	/
PMN, %	/	/
Pandy test	Extremely weak positive (±)	Negative
Acid-fast stain (acid-fast bacilli smear)	Negative (no AFB observed)	Negative

CSF, cerebrospinal fluid; mNGS, metagenomic next-generation sequencing; MN, monocyte; PMN, polymorphonuclear leukocyte; WBC, white blood cell; HB, hemoglobin; Cl, chloride; AFB, acid-Fast Bacilli.

Imaging. Lower extremity T2-weighted MRI (Fig. 2) performed on day 3 demonstrated increased T2-weighted imaging (T2WI) signal intensity in the affected muscles, with a feather-like appearance and heterogeneous signal intensity, consistent with RM.

Electromyography (EMG) was performed on day 2. Electrophysiological findings were consistent with myogenic injury involving both the upper and lower limbs.

Treatment. On day 6, treatment with 40 mg intravenous methylprednisolone was started once a day. This was increased to 80 mg on day 7 and the patient was discharged on day 10 with a daily dose of 60 mg oral prednisone.

Second admission (March-April 2024)

Patient presentation. The patient was re-admitted 15 days after discharge due to worsening muscle weakness. EMG was performed, showing evidence of peripheral neurogenic injury involving motor nerve fibers in both the upper and lower limbs.

Diagnosis and treatment process. The patient received methylprednisolone (40-80 mg intravenously once daily, with dose adjustments during the treatment course). Intravenous Ig (IVIG) therapy was administered at 20 g/day (0.4 g/kg/day based on a body weight of 50 kg) for 6 days, in two courses on days 27-29 and 38-40. On day 27, the serum CMV-IgM level was markedly elevated (213.30, AU/ml; normal value, <8 AU/ml; positive) and the CMV-IgG level was >1,000.00 AU/ml (normal value, <10 AU/ml; positive), indicating active CMV reactivation. On day 30, a lumbar puncture was performed; CSF was sent for routine tests, and biochemistry, microbiology and metagenomic next-generation sequencing (mNGS) analyses. mNGS was performed by Guangzhou Jinqixi Biotechnology Co., Ltd. DNA/RNA samples were prepared using the Library Preparation Kit (cat. no. KS130-CAPJK-48), Reverse Transcription Reagent (cat. no. KS630-NZL-48) and MetaCAP Hybridization Capture Reagent (cat. no. KS129-CAPBH-6). Sample quality and integrity were assessed using a fluorescence quantitative analyzer and a nucleic acid protein analysis system. Sequencing was conducted as single-end 100 bp. The sequencing reaction was performed using the Universal Sequencing Kit (Reversible Termination Sequencing Method; cat. no. KS107-CXR). The final library loading concentration was 1-1.5 pmol/l. Data were analyzed using the Pathogenic Microorganism Sequencing Data Analysis Software (MetaCAP; Version V6.5.3; Guangzhou Jinqixi Biotechnology Co., Ltd.). EMG performed on the same day revealed persistent myogenic injury, with new evidence of peripheral neurogenic involvement in the motor fibers of both upper and lower limbs. Empirical ganciclovir (0.25 g intravenously) was initiated on the same day as the lumbar puncture, administered from day 30 to day 49, and resumed again on day 69. mNGS reported a relative abundance of 100% for CMV DNA, confirming CNS CMV infection.

Plasma exchange was performed on day 31 (once daily for 3 consecutive days, with fresh frozen plasma replacement). Methotrexate (7.5 mg orally once weekly) was added on day 37 as a steroid-sparing agent, after the active CNS CMV infection was controlled.

The patient's muscle strength gradually improved and they were discharged on day 70 with continued oral prednisone taper starting at 50 mg/day, reduced by 5 mg every 2 weeks.

Final diagnosis, outcome and follow-up. The final diagnosis was immune-mediated necrotizing myopathy (SRP antibody-positive), RM and CNS cytomegalovirus infection.

After 3 months of treatment, the patient was discharged with upper limb muscle strength of grade 4+, demonstrating the ability to raise both arms, clench both fists and move against resistance. Lower limb muscle strength was grade 2+,

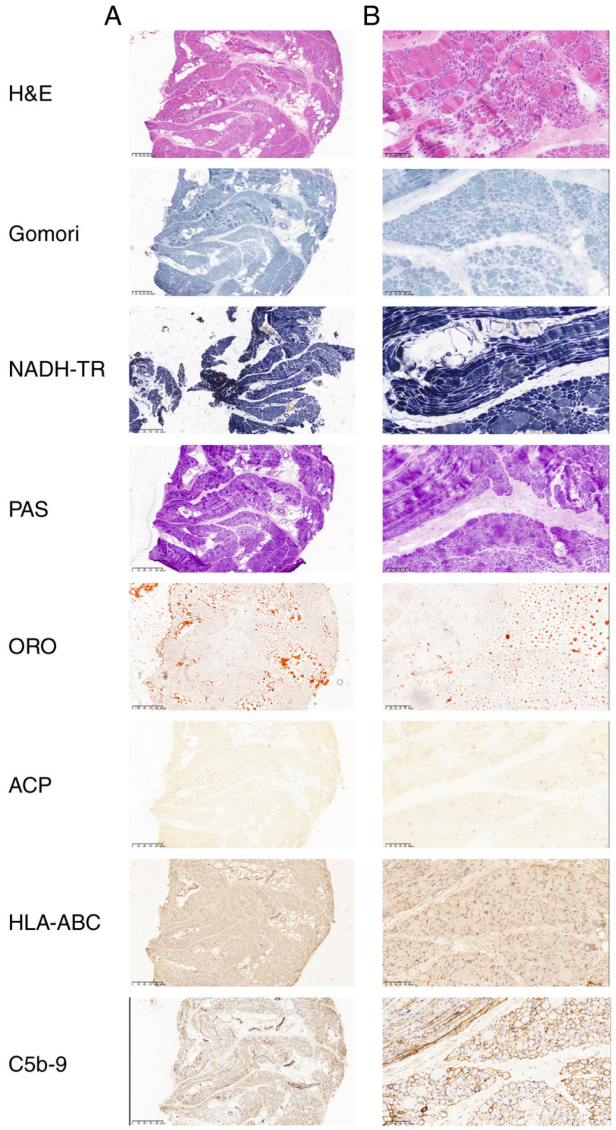


Figure 1. Histopathological and histological staining of the patient's muscle biopsy. A number of different staining methods were used, including H&E, Gomori, NADH-TR, PAS, ORO, ACP, HLA-ABC and C5b-9. (A) Scale bar, 400 μm; low-power view. (B) scale bar, 100 μm; high-power view. H&E, hematoxylin and eosin; NADH-TR, NADH-tetrazolium reductase; PAS, periodic acid-Schiff; ORO, Oil Red O; ACP, acid phosphatase; HLA-ABC, human leukocyte antigen class I; C5b-9, complement membrane attack complex.

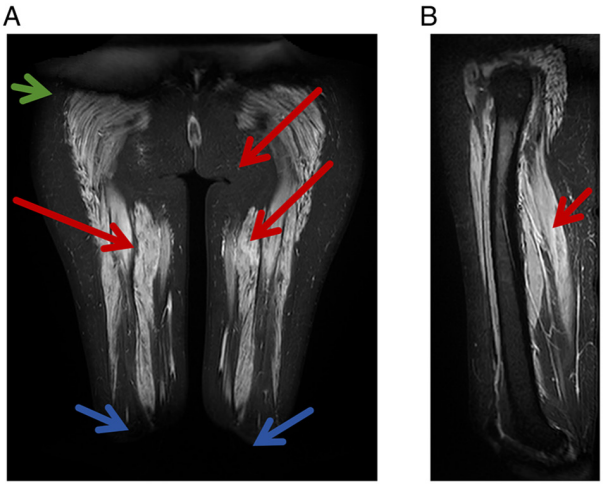


Figure 2. Lower extremity T2-weighted MRI acquired on admission in February 2024. (A) Axial view at the mid-thigh level. (B) Sagittal view encompassing the thigh and calf. The colored arrows indicate distinct imaging features: Green arrows point to abnormal thickening of the subcutaneous fat layer in the lateral thigh region; red arrows indicate areas of muscle edema, where diffuse, patchy high-signal (bright white) areas are visible within muscle groups such as the quadriceps, representing characteristic findings of acute muscle injury, cellular edema and inflammatory exudation; blue arrows mark areas of reduced muscle volume (atrophy), as the calf muscles show decreased bulk, contour concavity and heterogeneous signal intensity, suggesting atrophic changes following muscle tissue necrosis and loss. The combination of subcutaneous fat thickening, diffuse muscle edema and reduced muscle volume collectively constitutes the typical MRI presentation of rhabdomyolysis syndrome.

allowing the patient to sit independently but not to stand. At the 6-month follow-up, the condition of the patient had further improved. Both lower limbs could be lifted off the bed and ankle dorsiflexion could be performed. Upper limb muscle strength remained at grade 4+, while lower limb muscle strength improved to grade 3. Muscle tone was normal in all four extremities (Fig. 3).

Discussion

IMNM is a rare inflammatory myopathy with a subacute or insidious onset. Although necrotizing myopathy was first reported by Smith in 1969 (1), IMNM was not recognized as a distinct entity until 2003-2004 (2). IMNM complicated by RM has rarely been reported (10). In the present case, EMG demonstrated both myogenic and peripheral nerve involvement. CSF analysis revealed CMV infection and albuminocytological dissociation. Based on the clinical manifestations, laboratory findings, muscle pathology, electromyographic results and CSF analysis, the patient was diagnosed with anti-SRP antibody-positive IMNM complicated by RM and CNS CMV infection. GBS was included in the differential diagnosis as the patient presented with acute limb weakness, decreased muscle tone and mild albuminocytological dissociation, features that can mimic GBS. However, GBS was ultimately excluded by the presence of myopathic EMG changes, normal nerve conduction studies and a markedly elevated serum CK level (peaking at 32,213 U/l), which are inconsistent with typical GBS.

IMNM is characterized by proximal muscle weakness and increased serum CK levels. Histopathologically, IMNM is characterized by prominent muscle fiber necrosis and regeneration, with little or no inflammatory lymphocytic infiltration (3). IMNM is more common in women, with the peak age of onset between 40 and 50 years, and a higher incidence reported during the summer months. IMNM can be classified into three subtypes: Anti-SRP antibody-positive IMNM, anti-HMGCR antibody-positive IMNM and seronegative IMNM. Among the patients with anti-HMGCR antibody-positive IMNM, ~15% have a history of statin use, whereas only 4.2% of patients with anti-HMGCR antibody-negative IMNM have previously used statins. Simvastatin, atorvastatin and rosuvastatin are the statins most commonly associated with RM (4). In 2016, the ENMC (3) proposed the diagnostic criteria for anti-SRP antibody-associated myopathy, which include: i) Increased

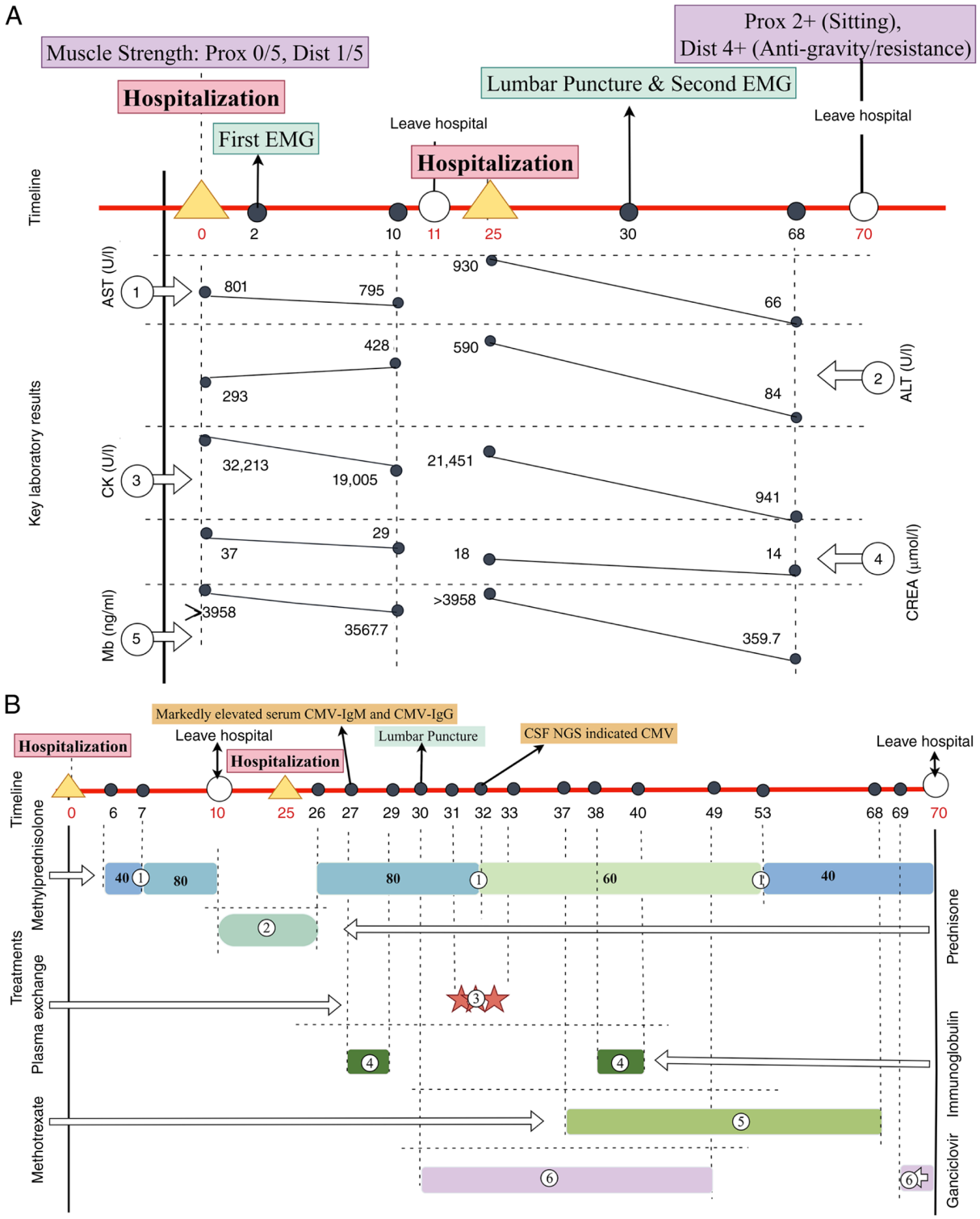


Figure 3. Timeline of the patient's muscle strength, treatments and laboratory examinations. The red horizontal line represents the timeline. Yellow triangles denote hospitalization (admission) events, white circles denote discharge events and black dots indicate specific examination or intervention nodes. To maintain a consistent temporal framework, all numerical labels above or below the timeline represent the number of days relative to the first admission (the first yellow triangle, defined as day 0). For example, the black dot labeled '2' indicates an event occurring 2 days after the first admission (day 2). (A) Clinical course and laboratory trends. Purple boxes indicate serial muscle strength assessments in the four limbs, graded using the Medical Research Council scale. Red boxes mark the two hospital admissions: February and March, 2024. Green boxes highlight key diagnostic procedures: The first EMG assessment on day 2 and the second EMG assessment combined with lumbar puncture on day 30. Beneath the timeline, the trajectories of five laboratory parameters are plotted: ① AST, ② ALT, ③ CK, ④ CREA and ⑤ Mb. (B) Therapeutic interventions. This panel details the administration of specific therapies. ① Methylprednisolone administered intravenously at varying dosages (40, 60 or 80 mg daily, as indicated within the colored boxes). ② Subsequent transition to oral prednisone following discharge. ③ Plasma exchange performed on three consecutive days (on days 31-33). ④ Intravenous Ig therapy administered during two discrete courses: on days 27-29 and on days 38-40. ⑤ Methotrexate therapy initiated on day 37 and continued until day 68. ⑥ Ganciclovir administered during two periods: from day 30 to day 49 and from day 69. Three arrows in the upper middle portion of the timeline indicate the following key events: Markedly elevated serum CMV-IgM and CMV-IgG on day 27; lumbar puncture performed on day 30; and CSF NGS suggesting CMV on day 32. EMG, electromyography; AST, aspartate aminotransferase; ALT, alanine aminotransferase; CK, creatine kinase; CREA, creatinine; Mb, myoglobin; Ig, immunoglobulin; CSF, cerebrospinal fluid; NGS, next-generation sequencing; CMV, cytomegalovirus. Created with Figdraw (www.figdraw.com).

serum CK levels; ii) proximal muscle weakness; and iii) positive results for anti-SRP autoantibodies. These criteria highlight the diagnostic importance of myositis-specific antibodies. A muscle biopsy is not required for the diagnosis of the first two IMNM subtypes (anti-SRP antibody-positive and anti-HMGCR antibody-positive IMNM), whereas patients who are negative for these serum antibodies still require a muscle biopsy to establish the diagnosis. The present patient was a young woman with acute-onset disease, markedly increased serum muscle enzyme levels and prominent proximal muscle weakness. Combined with the markedly increased serum CK level and positive anti-SRP antibodies, the clinical presentation fulfilled the ENMC diagnostic criteria for anti-SRP antibody-associated myopathy. Furthermore, the biopsy findings, characterized by necrotic and regenerating fibers, sparse lymphocytic infiltrate but macrophagic (histiocytic) infiltration, weak HLA-ABC expression and C5b-9 deposition, fulfilled the established histopathological criteria for IMNM. In conjunction with the clinical course and investigative results, alternative diagnoses were systematically excluded: Toxic myopathy was unlikely given the absence of statin or other myotoxic drug exposure; muscular dystrophy was considered improbable due to the acute onset, lack of family history and presence of anti-SRP antibodies; and infectious myositis was ruled out by the absence of viral inclusions or granulomatous changes on biopsy, with the concurrent CMV infection localized to the CNS rather than the skeletal muscle. RM is a clinical syndrome caused by skeletal muscle necrosis, resulting in the release of intracellular muscle constituents into the systemic circulation (5). The causes of RM include trauma, strenuous exercise, drug toxicity, infection, electrolyte disturbances, hereditary metabolic disorders and other conditions. Patients typically present with the classic triad of myalgia, muscle weakness and dark brown urine. In severe cases, RM can lead to acute kidney injury and may even be life-threatening. Some patients may also experience fatigue, fever, tachycardia, nausea, vomiting and abdominal pain. The most common triggers of RM are strenuous exercise, medications, trauma and infection, whereas immune-mediated factors are relatively uncommon (4). MRI findings of RM typically include diffuse swelling of the affected muscles with extensive involvement and ill-defined margins. T1WI usually demonstrates decreased signal intensity, whereas T2WI and short τ inversion recovery sequences show increased signal intensity with patchy or flocculent heterogeneous hyperintensity (11). In the present case, MRI demonstrated increased T2WI signal intensity in the affected muscles, with a feather-like appearance and heterogeneous signal intensity, findings that are consistent with RM. IMNM is a subtype of idiopathic inflammatory myopathy (IIM) (3). Although IIM and RM are distinct disease entities, they share similar clinical manifestations, and IIM is a rare cause of RM (12). Therefore, IMNM, as a subtype of IIM, may also be associated with the development of RM. In addition to the aforementioned evidence, previous studies have reported distinct pathological features in anti-SRP antibody-associated myopathy with and without RM (13). In patients without RM, necrotic muscle fibers are typically angular or rounded, depending on the degree of necrosis, and are randomly distributed throughout the muscle tissue (14). Inflammatory infiltrates are generally minimal or absent, and when present, they are macrophage-predominant ($CD68^+$) with sparse perivascular lymphocytes (14). MHC-I expression is usually absent or only weakly expressed on non-necrotic fibers (14). In cases with extensive muscle fiber necrosis, such as those complicated by RM, the pathological findings may demonstrate more prominent macrophagic (histiocytic) infiltration associated with necrotic fibers, increased endomysial connective tissue proliferation, sarcolemmal MHC-I expression on affected muscle fibers and occasional C5b-9 deposition (15,16). The pathological findings from the right thigh muscle biopsy in the present case demonstrated macrophagic infiltration and positivity for C5b-9, supporting the diagnosis of anti-SRP antibody-associated myopathy complicated by RM.

As detailed in the present report, the patient presented with severe RM yet did not develop AKI or compartment syndrome, or require conventional renal replacement therapy. This relatively favorable clinical course, particularly the preservation of renal function, may be partly attributable to the early initiation of plasma exchange, which we consider to offer dual therapeutic benefits in patients with severe IMNM complicated by RM (17). First, it removes circulating pathogenic autoantibodies, including anti-SRP antibodies, thereby reducing ongoing immune-mediated muscle fiber injury (18). Second, it facilitates the clearance of large amounts of circulating Myo released from damaged muscle, potentially decreasing the risk of cast nephropathy and acute kidney injury (19). In the present case, plasma exchange was initiated promptly (once daily for 3 consecutive days), which likely contributed to the removal of pathogenic antibodies and excess Myo, thereby promoting recovery of muscle strength and preventing renal deterioration. While plasma exchange has been reported to improve outcomes in IMNM (18,20), evidence for its benefit in severe RM is currently lacking (19). Therefore, early initiation may be a reasonable therapeutic consideration in critically ill patients with IMNM complicated by RM, although prospective studies are needed to confirm this hypothesis.

In addition, the following principles should be emphasized in the clinical management of patients with IMNM complicated by RM: i) Discontinue strenuous exercise and offending medications, avoid trauma and other precipitating factors, and promptly identify and treat underlying conditions such as infection and immune-mediated disorders (21); ii) initiate aggressive fluid resuscitation with either lactated Ringer's solution or normal saline, guided by the patient's cardiopulmonary status. An initial infusion rate of ~400 ml/h is recommended, with the goal of maintaining a urine output of 1-3 ml/kg/h (18); iii) renal replacement therapy should be considered in patients with a McMahon score $\geq$6 or in those who develop AKI despite adequate fluid resuscitation (22); iv) plasma exchange may facilitate the clearance of circulating macromolecules, including Myo, high-sensitivity cardiac troponin T, CK-MB and LDH, thereby potentially mitigating RM-associated AKI; however, its impact on overall mortality remains unproven (19,23); and v) electrolyte disturbances, particularly hyperkalemia, hyperphosphatemia and hypocalcemia, should be identified and corrected promptly throughout the course of RM (5). The present patient achieved a favorable short-term outcome following treatment; however, the long-term prognosis remains uncertain. Regarding the

selection of immunosuppressive therapy, methotrexate (7.5 mg orally once weekly) was introduced as a steroid-sparing agent after the active CNS CMV infection had been controlled with ganciclovir (24). Rituximab was intentionally avoided during the acute phase due to its profound and prolonged B cell-depleting effects, which could substantially increase the risk of CMV reactivation or exacerbation (24). Mycophenolate mofetil and azathioprine were not prioritized since they are mainly used as maintenance therapies and have a relatively slow onset of action, whereas this patient required rapid control of severe myositis and RM. Intravenous cyclophosphamide, another treatment option for IMNM, was also deferred due to the patient's young age and its potential adverse effects, such as decreased fertility (24,25).

When IMNM is complicated by RM, EMG typically reveals myogenic injury; however, a small proportion of patients may also exhibit evidence of peripheral nerve involvement. In the present case, EMG performed in March 2024 demonstrated peripheral neurogenic injury involving the motor fibers of both the upper and lower limbs, in addition to myogenic abnormalities. The peripheral nerve involvement observed on EMG may be explained by several mechanisms (26). First, prolonged direct nerve compression may result in compressive neuropathy. Second, increased intracompartmental pressure associated with muscle injury or compartment syndrome may lead to nerve compression and subsequent nerve injury. Third, muscle injury itself may induce local neuroinflammation, contributing to peripheral nerve dysfunction. In complex cases where RM is accompanied by peripheral nerve injury, functional recovery is often prolonged, and patients generally require a longer rehabilitation period (27). When RM is not accompanied by peripheral nerve injury, MRI typically demonstrates the following features: i) Diffuse involvement of the affected muscles with heterogeneous abnormal signal intensity, ill-defined margins, mild-to-moderate intermuscular exudation and subcutaneous edema; ii) on T1WI, most lesions (approximately two-thirds) show homogeneous mild hypointensity, whereas the remaining one-third exhibit heterogeneous signal intensity; and iii) on T2WI and fat-suppressed sequences, patchy, flocculent, feather-like or linear areas of heterogeneous hyperintensity are observed, accompanied by muscle swelling and a loose muscle architecture (28). By contrast, when RM is accompanied by peripheral nerve injury, MRI may demonstrate the following features: i) Intramuscular hemorrhage and muscle edema in ~60% of patients; ii) more prominent high signal intensity within the subcutaneous tissue; iii) increased muscle volume with diffuse swelling and a loose muscle architecture; iv) muscle lesions with predominantly rectangular or linear morphology and irregular, sharply demarcated margins; v) variable patterns of muscle involvement, often affecting multiple muscles within the same limb or compartment; and vi) a higher likelihood of compartment syndrome with increased intracompartmental pressure, resulting in secondary peripheral nerve injury (28). In the present patient, after systematic treatment, post-treatment evaluation demonstrated a marked improvement in the patient's serum CK and hemoglobin levels. Upper limb muscle strength recovered substantially, whereas recovery of lower limb muscle strength was slower. MRI revealed a characteristic pattern in which the central portion of the affected muscle bundles showed no enhancement, while the peripheral regions exhibited contrast enhancement. This imaging pattern suggests severe irreversible necrosis in the central muscle tissue with ongoing repair and inflammatory activity at the periphery, which may explain the delayed recovery of lower limb muscle strength. Nevertheless, it is important to acknowledge the limitations of these interpretations. Although the preceding discussion provides evidence-based explanations for the neurogenic changes observed in this case, these conclusions are mainly based on the clinical findings of a single patient and should therefore be interpreted with caution. In complex clinical scenarios, neurogenic findings may result from multiple concurrent mechanisms, including direct nerve compression, RM-related nerve injury, critical illness, treatment-related effects and coexisting peripheral neuropathy (29). Therefore, the aforementioned interpretations should be regarded as hypothesis-generating observations based on this individual case rather than definitive conclusions. The underlying pathophysiological mechanisms in each patient should be evaluated on a case-by-case basis, with treatment strategies tailored to the individual clinical presentation.

Peripheral neuropathy, particularly GBS, can present with clinical features similar to those observed in the current patient, including limb weakness or paralysis and decreased muscle tone. GBS is an acute immune-mediated polyradiculoneuropathy characterized by multiple nerve root and peripheral nerve involvement. Albuminocytologic dissociation in the CSF is one of its characteristic findings (30). In severe cases, paralysis of the respiratory and bulbar muscles may occur, leading to life-threatening respiratory failure (31). The diagnosis of GBS is mainly based on the patient's clinical history, electrophysiological findings and CSF examination, with nerve conduction studies and EMG serving as essential diagnostic tools. Electrophysiological findings in GBS typically include prolonged distal motor latency and F-wave latency, temporal dispersion, prolonged compound muscle action potential duration, reduced nerve conduction velocity and conduction block in non-entrapment nerve segments (32). In the present patient, in March 2024, EMG demonstrated electrophysiological evidence of myogenic injury in the examined muscles, characterized by shortened motor unit potential duration, increased polyphasic motor unit potentials and reduced amplitude. Sensory and motor nerve conduction velocities, F-wave latencies and F-wave persistence were all within normal limits. Although CSF analysis revealed mildly increased protein levels, the increase was modest. Given that systemic CMV reactivation is typically associated with viremia, this mild protein elevation may more plausibly reflect localized CNS CMV infection rather than non-specific systemic inflammation. Furthermore, serum CK levels are typically normal or only mildly increased in patients with GBS, whereas this patient had a markedly increased CK level, peaking at 32,213 U/l, strongly supporting a primary myopathic rather than neuropathic process. Therefore, although the patient presented with limb weakness resembling GBS and exhibited mild albuminocytological dissociation in the CSF, the electrophysiological findings were not consistent with GBS or other peripheral nervous system disorders. Based on these combined findings, particularly the myopathic

EMG changes and the markedly elevated CK level, the patient did not meet the diagnostic criteria for GBS. It is also essential to distinguish clinically significant CMV CNS infection from incidental viral detection or systemic CMV reactivation, particularly in this case, as the patient received both immunosuppressive therapy and ganciclovir, and the differential diagnosis with GBS relied heavily on the interpretation of CSF albuminocytologic dissociation and EMG findings. Several lines of evidence support that the positive CSF mNGS result represented an active CNS infection. First, CMV DNA was detected by mNGS in the CSF on day 32, which was after the initiation of immunosuppressive therapy (methylprednisolone was started on day 6 and IVIG was administered on days 27-29). This timeline is consistent with a treatment-related opportunistic infection, rendering incidental viral detection or systemic reactivation unlikely. Second, although systemic CMV reactivation typically presents with viremia, the detection of viral DNA directly in the CSF suggests a localized pathological process within the CNS. Importantly, the mNGS report showed a relative abundance of 100% for CMV; however, viral load quantification (for example, IU/ml or copies/μl) and read numbers were not available from the assay. Third, serum CMV-IgM was markedly elevated at 213.30 AU/ml (normal value, <8 AU/ml; positive) and CMV-IgG was >1,000.00 AU/ml (normal value, <10 AU/ml; positive), providing serological evidence of active CMV infection. Fourth, acid-fast staining of the CSF was negative, effectively excluding tuberculous meningitis and other acid-fast bacillary infections as the cause of the neurological manifestations. Fifth, the presence of CMV in the CSF provides a plausible infectious explanation for the observed albuminocytological dissociation and the neurogenic findings on EMG, features that might otherwise be attributed solely to GBS or critical illness-related neuropathy. Regarding the pathogenesis of IMNM complicated by CNS CMV infection, two possible mechanisms merit consideration. First, CMV infection may trigger an autoimmune response through specific immunological mechanisms, such as molecular mimicry and bystander activation, potentially contributing to the development of necrotizing myopathy (8,33,34). Alternatively, it may occur as an opportunistic infection following immunosuppression (7). In the present case, CMV DNA was detected in the CSF after the initiation of immunosuppressive treatment, and serum CMV-IgM/IgG levels were already elevated on day 27, suggesting that CMV reactivation likely occurred as a consequence of immunosuppression and subsequently involved the CNS, although a contributing role in triggering autoimmunity cannot be entirely excluded.

The present case report has several limitations that should be acknowledged. First, as a single-case report, its findings may not be generalizable to all patients with anti-SRP antibody-positive IMNM complicated by RM and CNS CMV infection. Second, the follow-up period was relatively short (6 months), and the long-term prognosis, including the risk of relapse, persistent muscle weakness and late neurological sequelae of CNS CMV infection, remains uncertain. Third, the CSF mNGS assay detected CMV with a relative abundance of 100%, but viral load quantification and read numbers were not available from the test report, limiting the ability to precisely quantify viral burden or monitor treatment response. Additionally, the diagnosis of CNS CMV infection was based on a combination of CSF mNGS results, serological evidence (positive CMV-IgM and IgG), and clinical context, rather than being confirmed by conventional virological methods such as CSF CMV PCR or viral culture. Further studies involving larger cohorts and longer follow-up are needed to better characterize the clinical course of this rare condition and to establish evidence-based management strategies.

In conclusion, the present report describes a rare case of anti-SRP antibody-positive IMNM complicated by RM and CNS CMV infection. Although a single case cannot establish definitive management recommendations, it highlights the importance of distinguishing anti-SRP antibody-positive IMNM complicated by RM from GBS in patients presenting with severe muscle weakness and markedly increased muscle enzyme levels. It also underscores the need for thorough evaluation of concomitant infections before and during immunosuppressive therapy to facilitate timely diagnosis and appropriate treatment.

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Availability of data and materials

The mNGS data generated in the present study may be found in the National Center for Biotechnology Information Sequence Read Archive under BioProject accession number PRJNA1525608 or at the following URL: https://www.ncbi.nlm.nih.gov/bioproject/PRJNA1525608.

Authors' contributions

YX, as the attending physician, was responsible for the patient's diagnosis and treatment throughout the course, supervised the histopathological staining procedure, reviewed the stained slides and wrote the original draft. NZ was responsible for data curation, visualization and analysis of mNGS data, and assisted in writing the original draft. JL was responsible for obtaining the medical images (MRI and CT), funding acquisition, supervision and reviewing/editing the manuscript. QZ provided an expert opinion on the patient's treatment and was responsible for reviewing and editing the manuscript. QH analyzed the patient's laboratory data, performed project administration and assisted in reviewing and editing the manuscript. All authors have read and approved the final version of the manuscript. YX and NZ confirm the authenticity of all the raw data.

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of The Affiliated Traditional Chinese Medicine Hospital, Southwest Medical University (Luzhou, China).

Consent for publication

The patient and the patient's guardian (due to the severe nature of the illness) provided written informed consent for the publication of this case report, including all information and images.

Competing interests

The authors declare that they have no competing interests.

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