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CASE REPORT

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Guillain–Barre syndrome complicating knee infectious arthritis

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Key Clinical Message

Guillain–Barré syndrome (GBS) is a rare immune status, which affects the nerves, leading to muscle weakness and tingling, and, in some cases, paralysis. Our case showed that GBS may happen after knee infectious arthritis.

KEYWORDS

Guillain–Barré syndrome, infectious arthritis, rheumatology, septic arthritis

1 | INTRODUCTION

Guillain–Barré syndrome (GBS) is a rare immune disorder, which attacks the nerves, causing weakness and tingling, and eventually paralyzing. In most patients, an infection in the six weeks preceding is accused, although its exact cause is still now unobvious.[1,2] Our case is GBS developed during the course of knee infectious arthritis.

2 | CASE REPORT

A 43-year-old married handworker man, nonsmoker, nonalcoholic, presented to the Emergency Department with a history of fever, right knee pain, and swallowing since yesterday, without a previous trauma or fall on the knees.

The patient had no significant medical history including traveling or connection with a person infected with a contagious disease, nor family history.

On admission, the physical examination showed the following: The patient was febrile (39.2°C), had tachycardia 110/m, normal blood pressure, and a painful swallowed left knee with local fever. The rest of the examination was unremarkable.

Laboratory test results revealed the following: elevated white blood cells (18,000 mm^3), elevated erythrocyte sedimentation rate (78 m/h), and elevated C-reactive protein levels (46 mg/dL). Urine analysis was normal.

The knee arthrocentesis showed that synovial fluid was opaque and cloudy in appearance, leukocytes >50,000/μL, and more than 90% neutrophils. Gram stain was positive for *Staphylococcus aureus*, and blood cultures grew also *S. aureus*. Intravenous antibiotics with vancomycin were initiated, and the patient had admitted to the Emergency Department, with a diagnosis of knee septic arthritis, according to American College of Rheumatology criteria.[3,4] The next day of admission, his Glasgow Coma Scale dropped to 8/15, with spontaneous movement of the limbs, and he was transferred to intensive care after intubation.

The neurological assessment in the intensive care unit revealed decreased lower limb reflexes with normal tone. Spontaneous movement of the upper limbs was found the next day only. He continued until he was extubated on Day 7. Areflexia, hypotonia, and no power in the lower limbs were the findings of the neurological examination. Upper limb neurology was intact. According to these findings, GBS was suspected to have developed after the knee infectious arthritis with *S. aureus*.[5]

White cell count was 25.0×10^9 cells/L, platelet count was 39.0×10^9/L, and a raised INR at 1.6. According to the above laboratory tests, we could not perform the lumbar puncture.

Nerve conduction studies showed severe proximal motor polyneuropathy, without the presence of active denervation (Figures 1–10).

Magnetic resonance imaging revealed T2 signal hyperintensity and enhancement consistent with GBS (Figures 11 and 12).

He was treated with intravenous immunoglobulin for 5 days, and vancomycin.

The patient was discharged after 2 months. Upon review, he continued to improve. Fifteen days later, he was able to serve himself and walks backward for 5 min. His balance was improving. After 3 months, he could move with help, and he was catheterized his bladder. He did

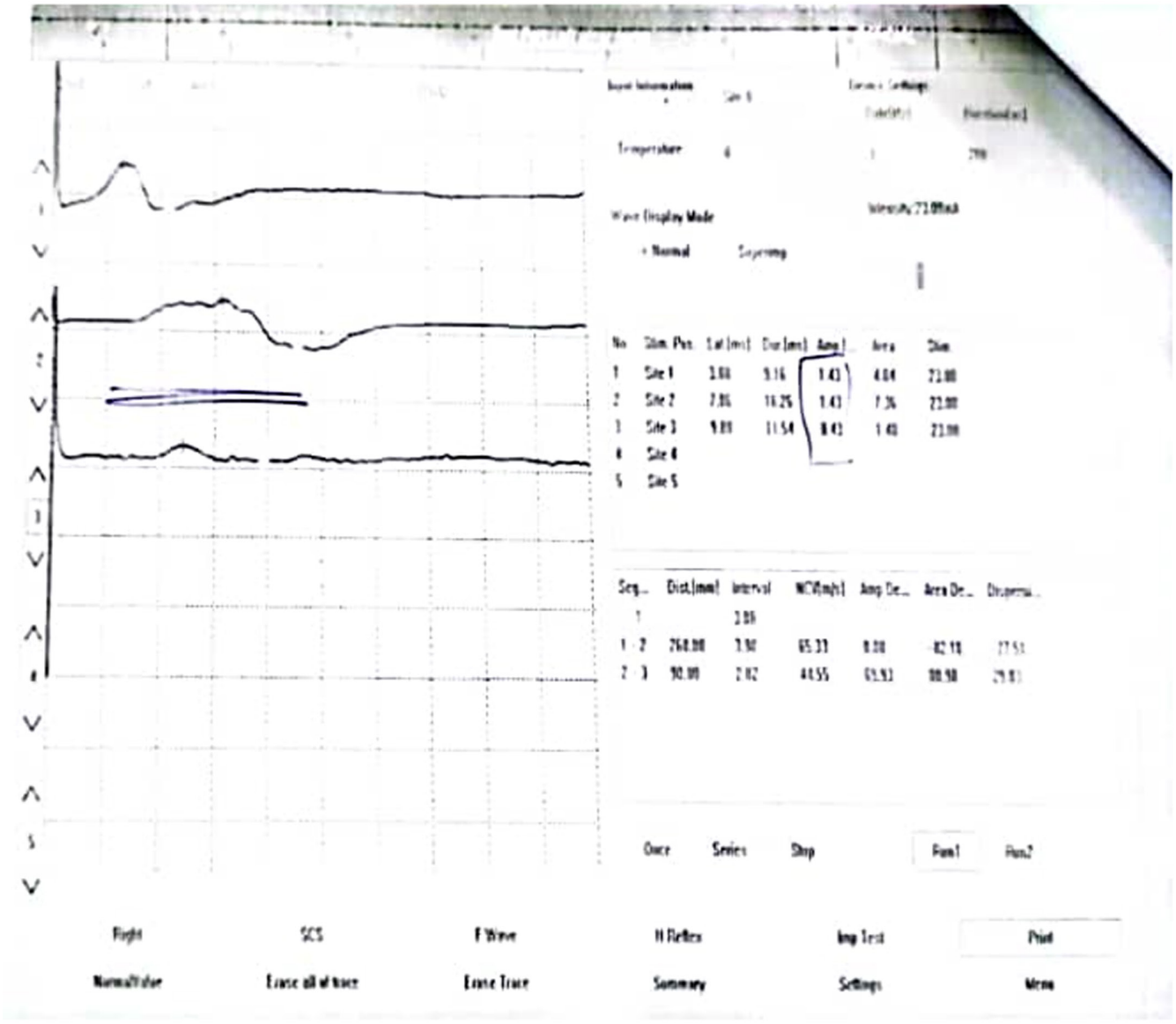

FIGURE 1 Nerve conduction studies showed severe proximal motor polyneuropathy.

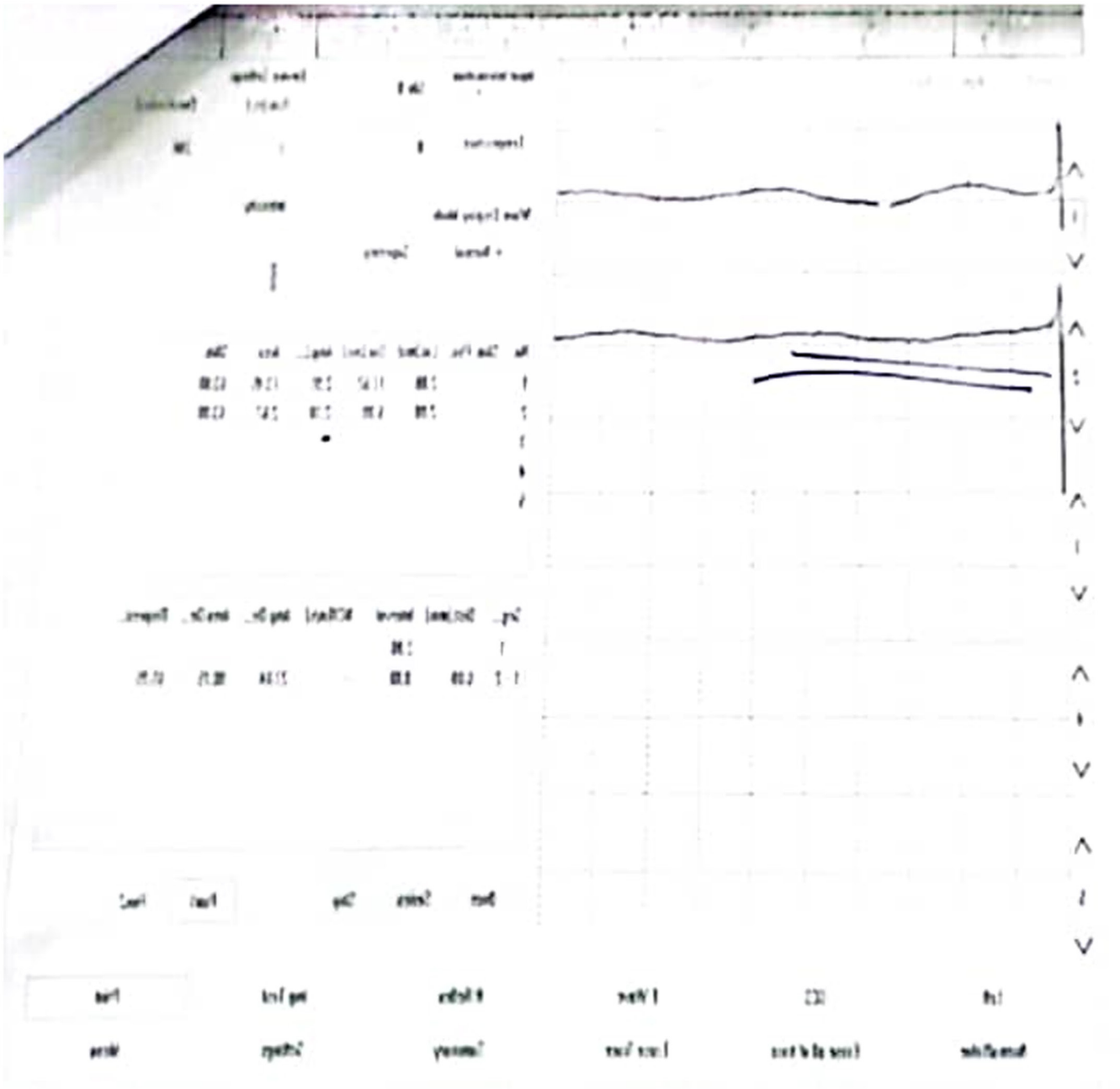

FIGURE 2 Nerve conduction studies showed severe proximal motor polyneuropathy.

poop manually. He had paresthesia like an electrocution pain in both feet. He could elevate his legs and lie down. His hands were fully functional. He scored 3 on the Modified Rankin Scale.[6] He has improved after 1 year since diagnosis without disability (Modified Rankin Scale = 0).

3 | DISCUSSION

Guillain–Barré syndrome is a rare condition, manifested with the severe onset of paralysis. In most patients, an infection in the six weeks preceding is accused, although its exact cause is still now unobvious.[1,2]

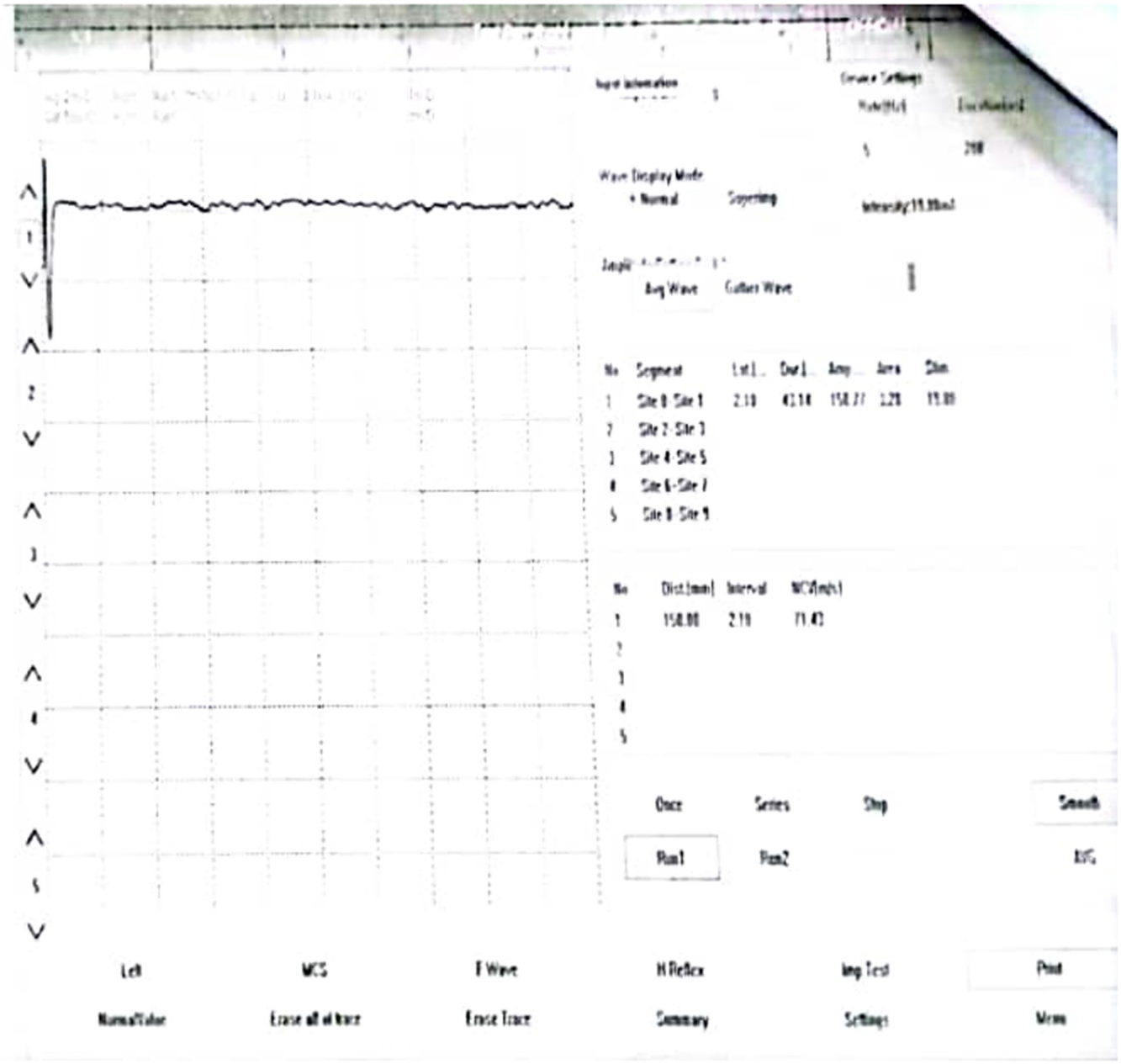

FIGURE 3 Nerve conduction studies showed severe proximal motor polyneuropathy.

It may associate with various pathogens such as viruses and bacteria.[7] A novel finding raised by our patient is the presentation of GBS after *S. aureus* septic arthritis.

Guillain–Barre syndrome can be difficult to diagnose in its earliest stages, and its signs and symptoms are similar to those of other neurological disorders and may vary from person to person, but with good medical history and clinical examination diagnosis can be made in addition to Electromyography and Neuroelectrography, also spinal tap (lumbar puncture) can make the diagnosis.[8]

Other etiologies that may mimic GBS include tick paralysis; also, there would be an initial presentation of neuromuscular junction disorder, acute intermittent porphyria, HIV infection, spinal cord disorder, toxic neuropathies, and even infections (such as West Nile virus or rabies).[8]

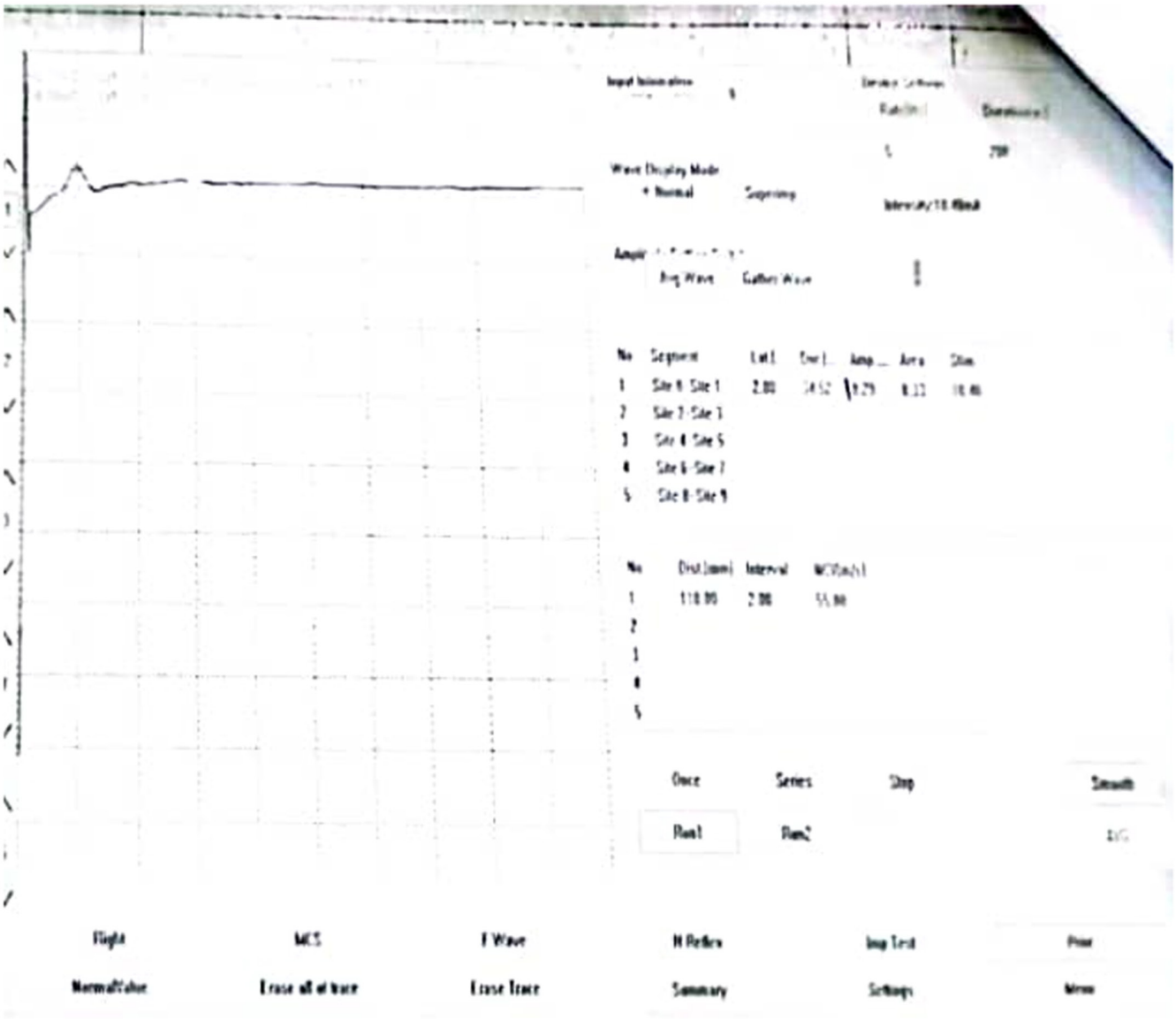

FIGURE 4 Nerve conduction studies showed severe proximal motor polyneuropathy.

In our patient, there were no story of tick paralysis or other viral infections such as West Nile virus or rabies; also, there were no sexual relations or blood transfusion so we excluded HIV infection, and there were no previous spinal cord disorders and the MRI of the spinal cord was normal.

There are several risk factors for septic arthritis such as age, liver diseases, and immunosuppressive medication,[9] but our patient had none of them.

It is well-known that other infections, but not *S. aureus* infection, may trigger GBS,[2] although it was *S. aureus* infection, which likely trigger the patient's subsequent GBS.

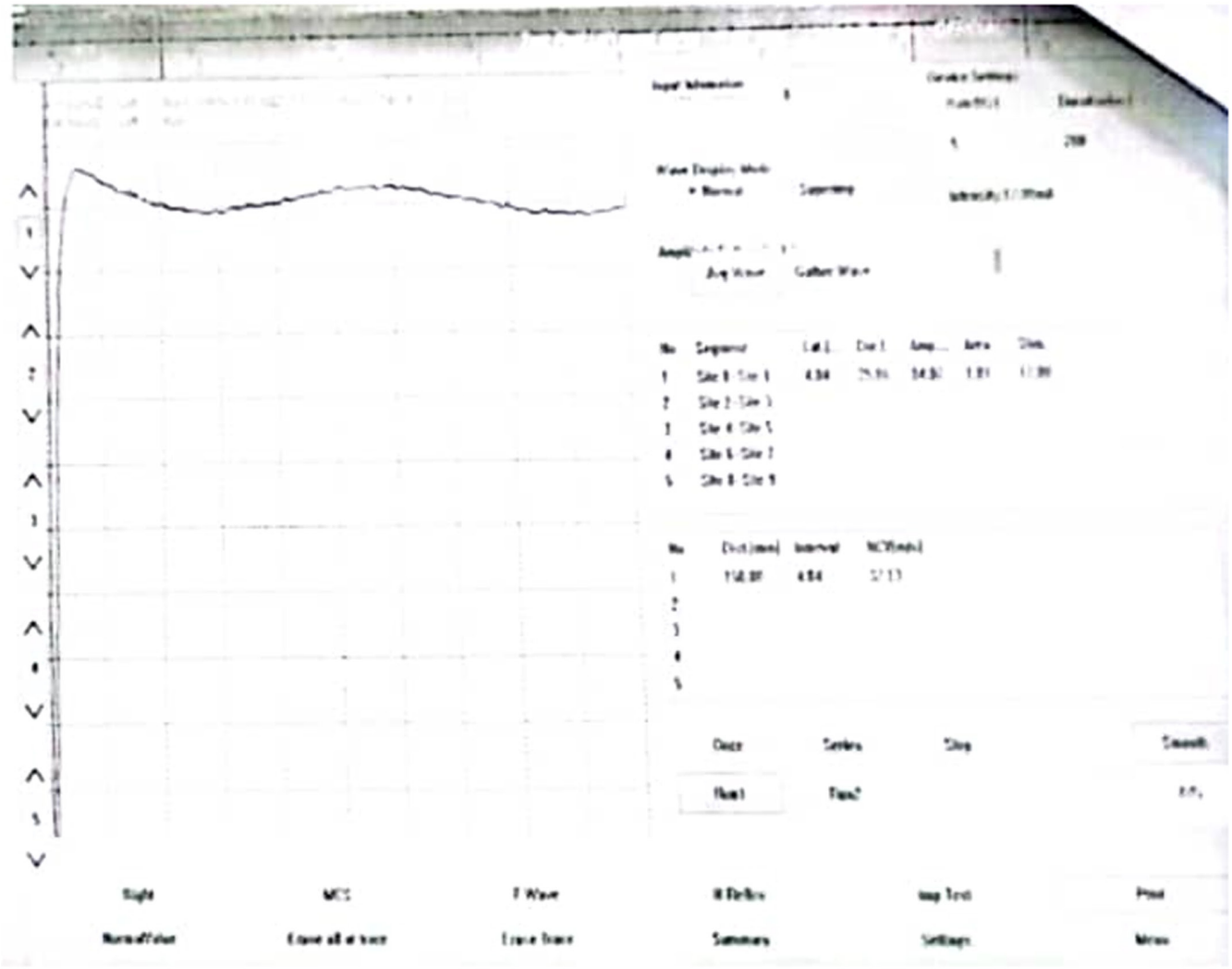

FIGURE 5 Nerve conduction studies showed severe proximal motor polyneuropathy.

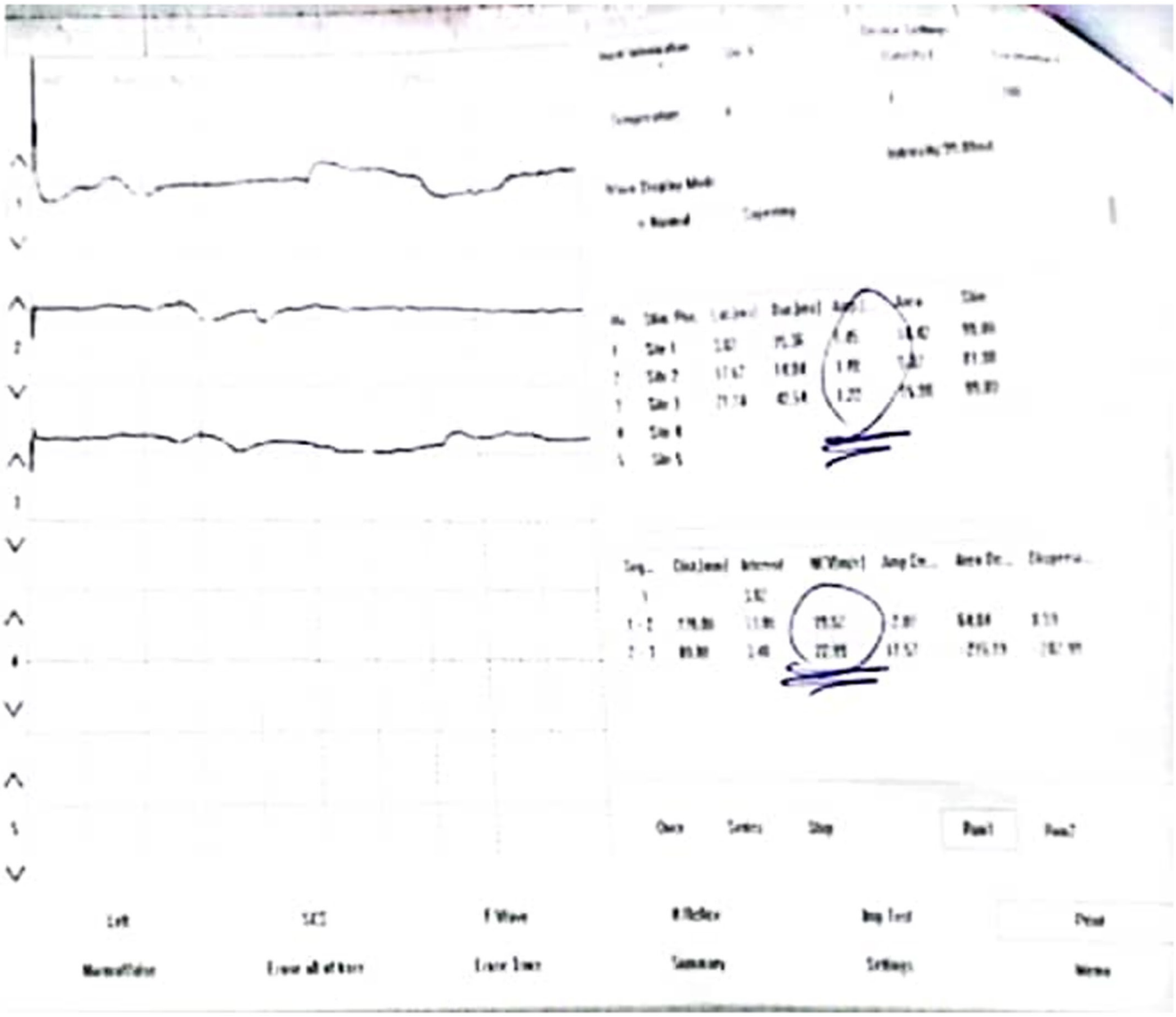

FIGURE 6 Nerve conduction studies showed severe proximal motor polyneuropathy.

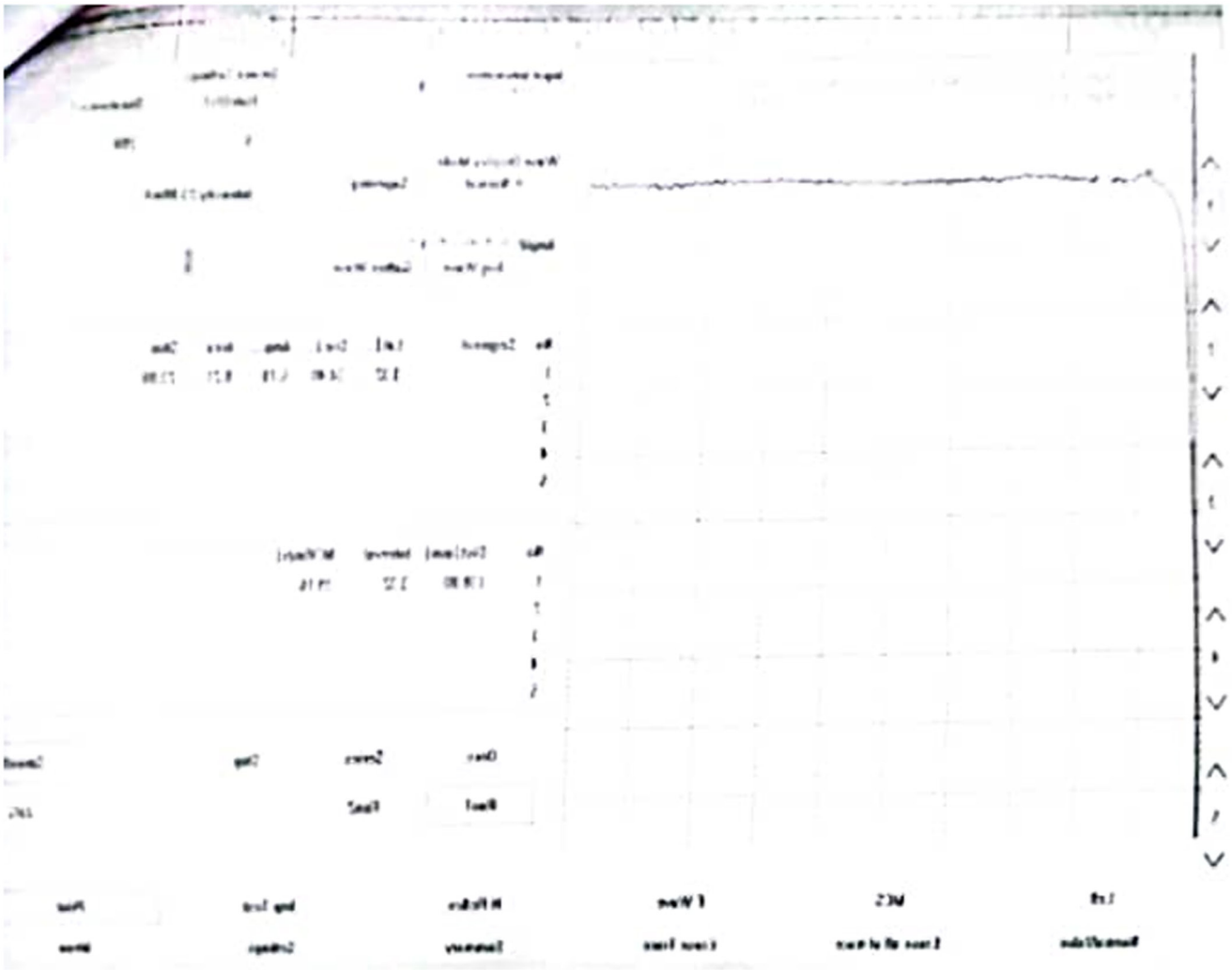

FIGURE 7 Nerve conduction studies showed severe proximal motor polyneuropathy.

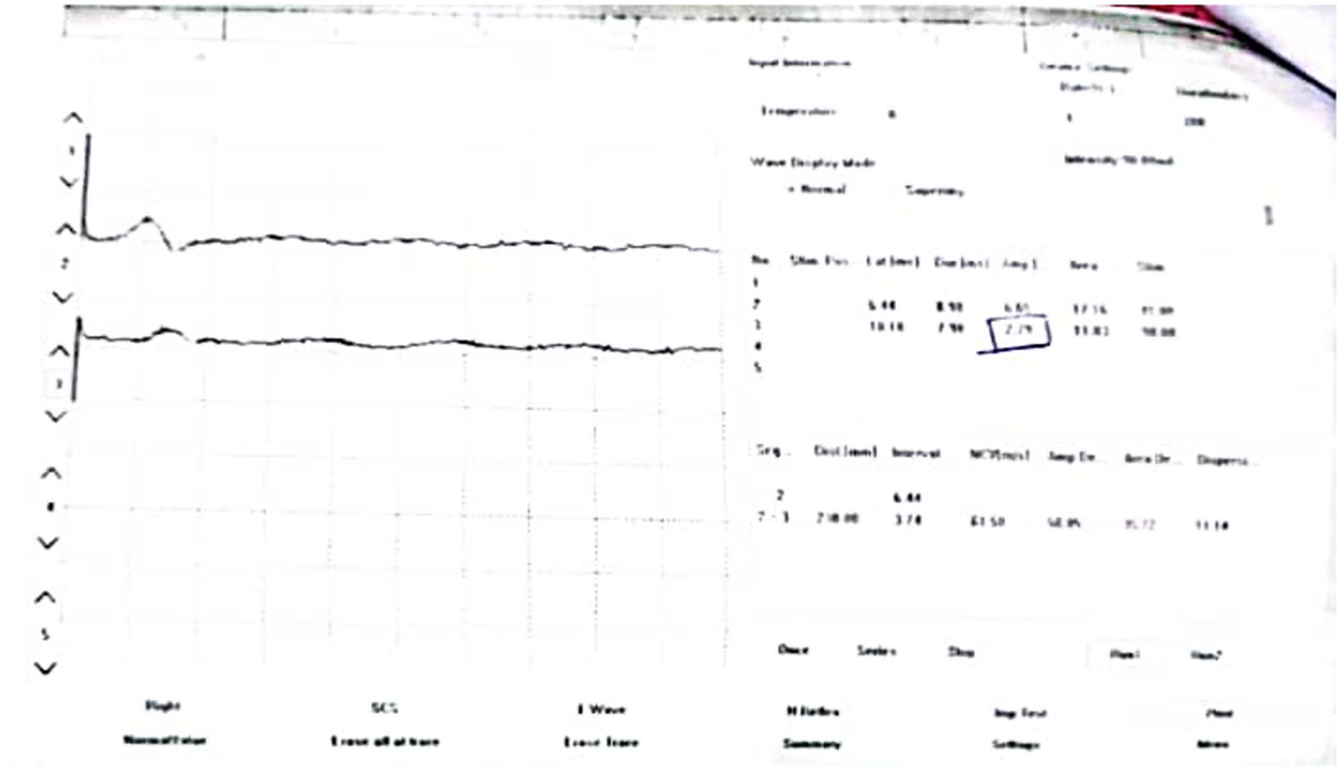

FIGURE 8 Nerve conduction studies showed severe proximal motor polyneuropathy.

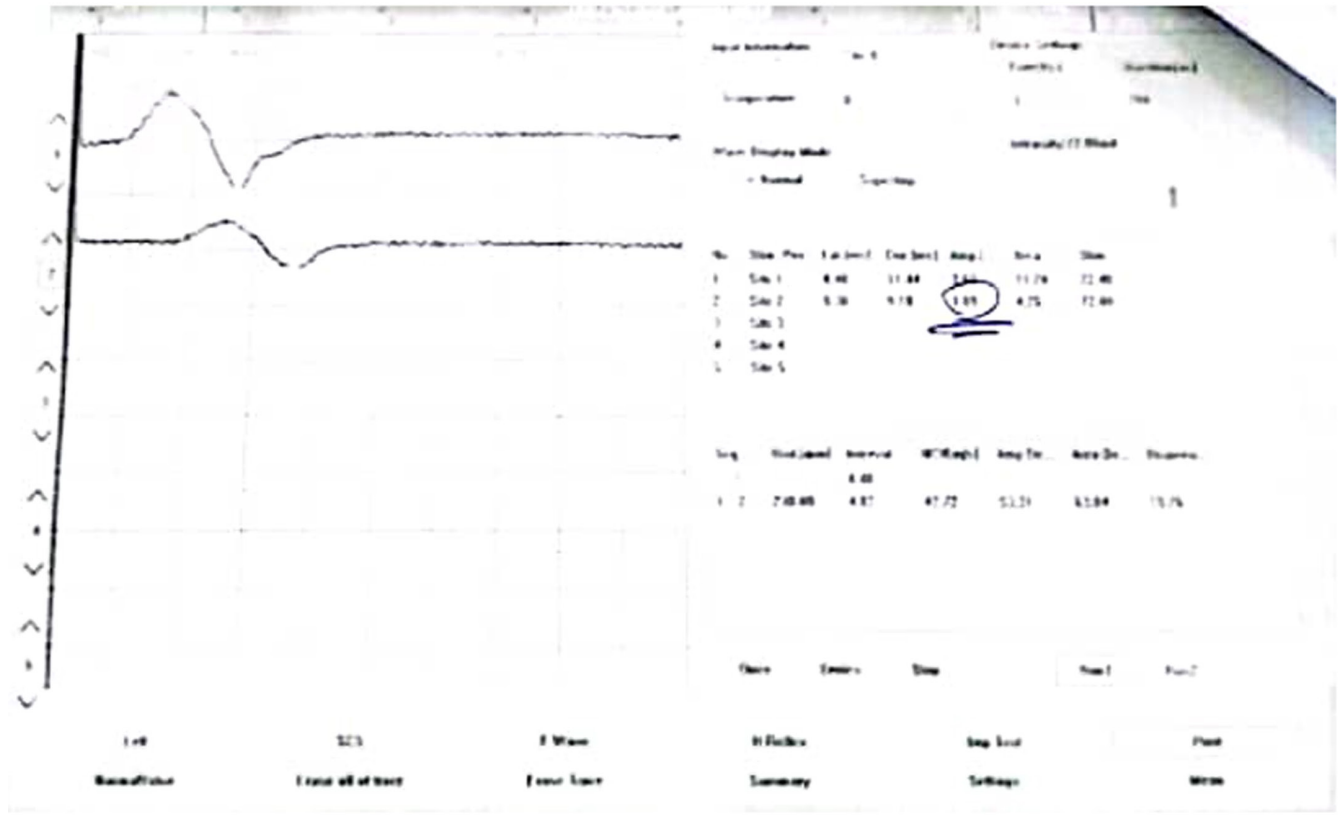

FIGURE 9 Nerve conduction studies showed severe proximal motor polyneuropathy.

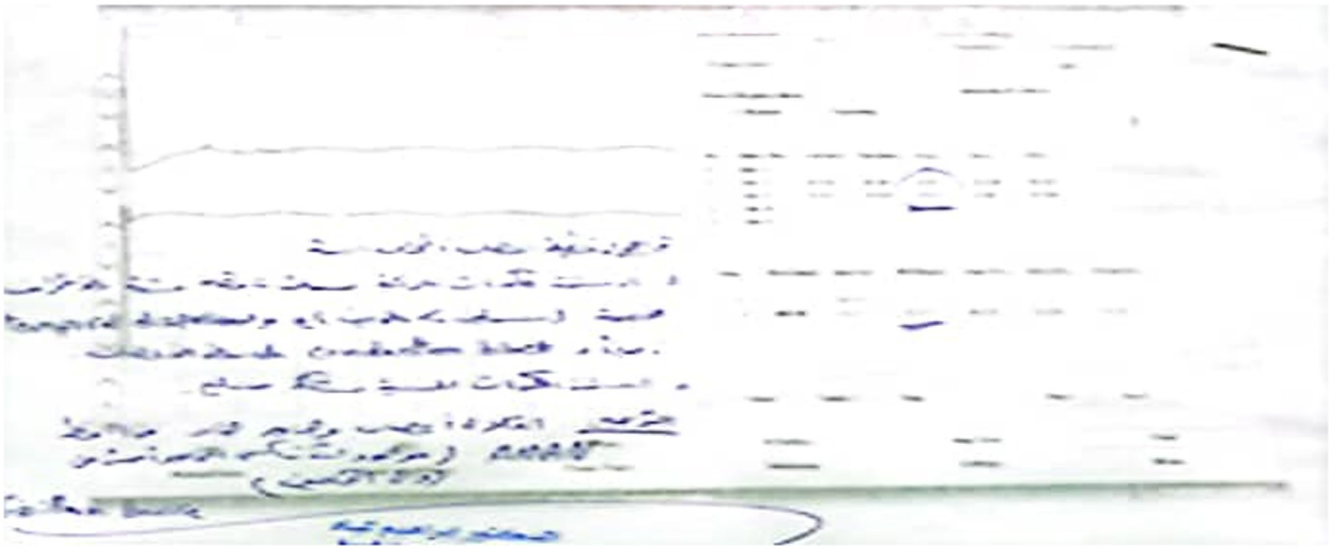

FIGURE 10 Nerve conduction studies showed severe proximal motor polyneuropathy.

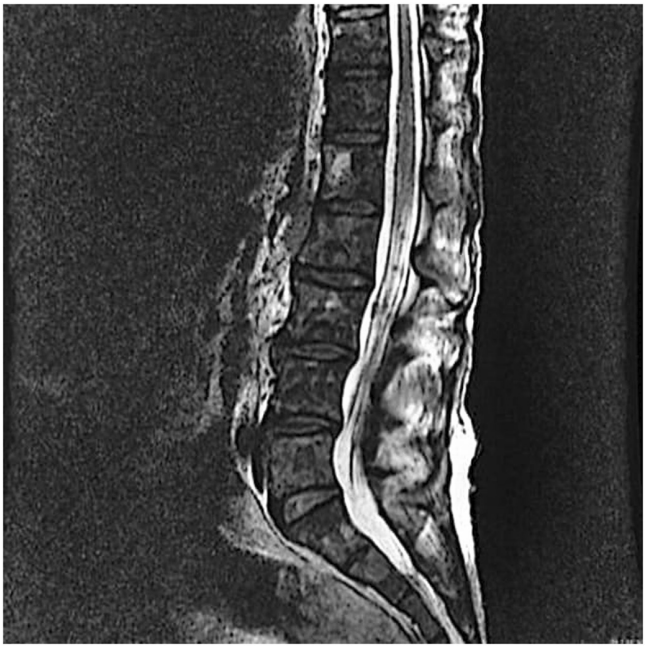

FIGURE 11 Magnetic resonance image shows hyperintensity and enhancement consistent with GBS.

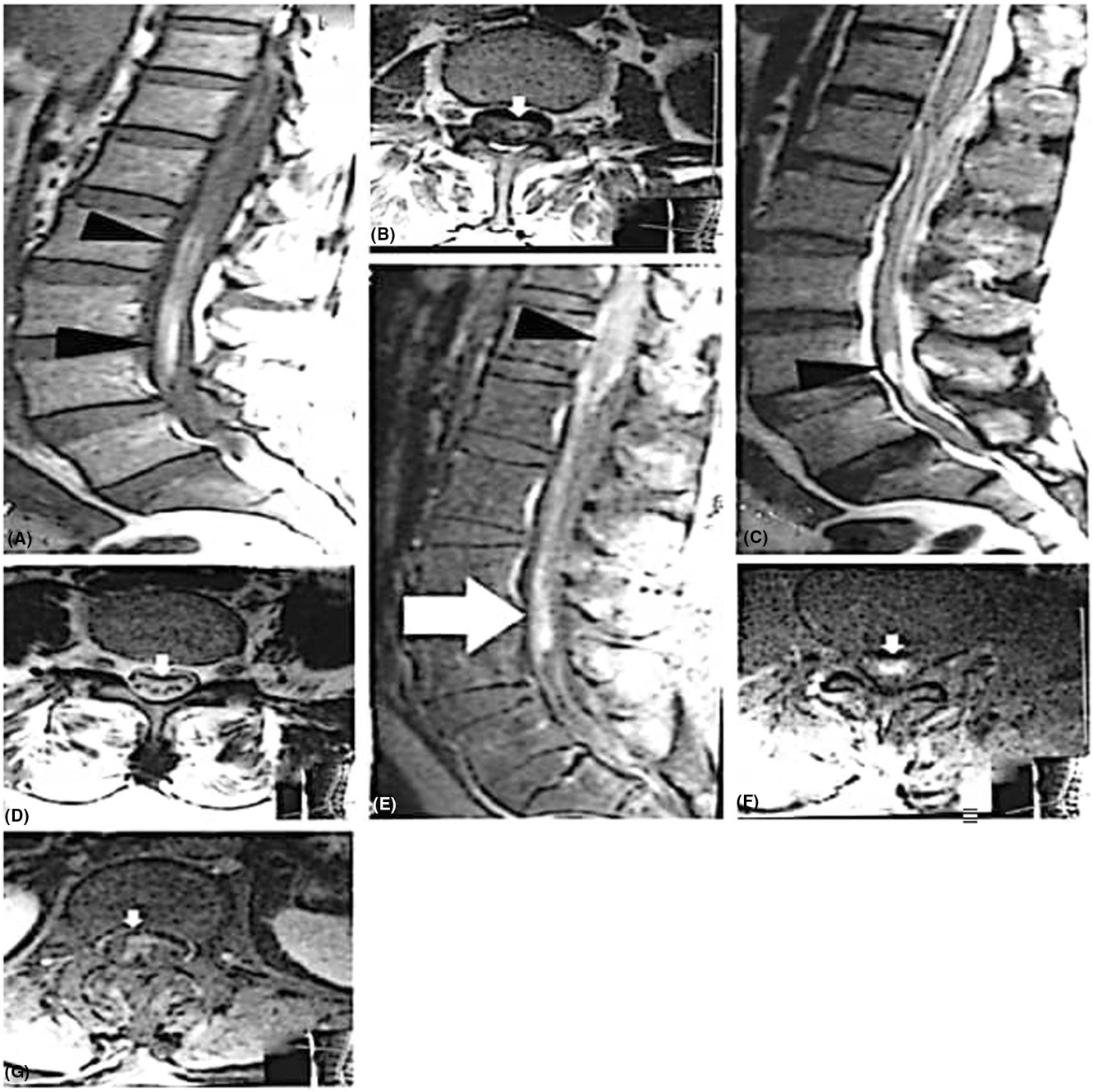

FIGURE 12 Magnetic resonance image shows hyperintensity and enhancement consistent with GBS.

The exact mechanism for GBS after bacterial infection may be explained by molecular mimicry as described for *Campylobacter jejuni*.[10]

In literature reviews, it was found that it caused GBS in neonates of mothers with *S. aureus* infection,[11] although it was the trigger of GBS in a patient with endocarditis caused by these germs.[12]

S. aureus is the most bacteria isolated from synovial fluid cultures in previous studies,[13,14] as in our case and the incidence of septic arthritis caused by methicillin-resistant *S. aureus* has increased.[15] Vancomycin is one of the used antibiotics for these cases,[13,15] as we did.

Guillain–Barre syndrome treatment included in severe cases: intubation, plasmapheresis, intravenous immunoglobulin, and electrolytes management,[8,16] as we did.

The originality of our case is that GBS is observed after septic arthritis with *S. aureus*.

4 | CONCLUSION

Our patient showed that GBS may be observed after septic arthritis with *S. aureus*, although further cases are necessary to identify the mechanism by which GBS develops after a *S. aureus* infection.

AUTHOR CONTRIBUTIONS

Naram Khalayli: Conceptualization; data curation. **Diaa Haj Ali:** Methodology; software. **Mayssoun Kudsi:** Project administration; supervision; writing – original draft; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

No conflicts of interest.

DATA AVAILABILITY STATEMENT

The data are available from the corresponding author upon reasonable request.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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