Section: Internal Medicine

Case Report

MENINGEAL MAYHEM: AN ATYPICAL MANIFESTATION IN YOUNG FEMALE

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Corresponding Author:
Dr. Satyaki Basu,
Email: destinybasu@gmail.com

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Girin Ray[1], Satyaki Basu[2], Chandrapaul Gupta[3], Soumyadeep Maity[4], Arijit Saha[5], Adnan Bin Nasir[6]

[1,3,4]PGT, Department of Internal Medicine, KPC Medical College and Hospital Kolkata, India
[2]Assistant Professor, Department of Internal Medicine, KPC Medical College and Hospital Kolkata, India.
[5,6]Intern, KPC Medical College and Hospital Kolkata, India.

ABSTRACT

Background: Herpes simplex virus type 2 (HSV-2) uncommonly presents as extensive myelopathy with concurrent meningeal involvement. We report a case demonstrating remarkable clinical and radiological recovery following prompt antiviral therapy. **Case Presentation:** A 24-year-old previously healthy woman developed acute viral meningomyelitis with extensive long-segment myelopathy spanning C5-L2 vertebral levels and neurogenic bladder dysfunction. Clinical presentation included progressive lower extremity weakness, urinary retention, and altered mental status over two weeks. Cerebrospinal fluid analysis revealed lymphocytic pleocytosis (150 cells/μL, 90% lymphocytes) with positive HSV-2 polymerase chain reaction. Magnetic resonance imaging demonstrated extensive T2 hyperintensity and gadolinium enhancement across multiple spinal segments from C5 to L2. **Management and Outcome:** Treatment consisted of high-dose intravenous acyclovir (10 mg/kg every 8 hours for 14 days) and pulse methylprednisolone (1g daily for 3 days), resulting in near-complete neurological recovery. Motor strength improved from 2/5 to 5/5 bilaterally in lower extremities over 8 weeks. Follow-up magnetic resonance imaging at 8 weeks demonstrated significant resolution of myelitis with minimal residual T2 hyperintensity and complete resolution of gadolinium enhancement. Neurogenic bladder dysfunction persisted despite motor recovery. **Conclusions:** This case demonstrates the potential for complete motor recovery from extensive HSV-2 myelopathy with prompt antiviral therapy, emphasizing the critical importance of early diagnosis and aggressive treatment in HSV-associated central nervous system infections.

INTRODUCTION

Viral meningomyelitis represents a rare but potentially devastating neurological condition characterized by concurrent inflammation of the meninges and spinal cord. Herpes simplex virus type 2 (HSV-2) is a well-established cause of central nervous system infections, typically manifesting as aseptic meningitis or encephalitis.[1,2] However, its presentation as extensive long-segment myelopathy with concurrent meningeal involvement remains uncommon in the medical literature.

HSV-2-associated myelitis occurs in fewer than 1% of HSV-2 infections, with most cases presenting as isolated sacral myelitis or cauda equina syndrome.[3] Extensive long-segment myelopathy spanning multiple vertebral levels is exceptionally rare, with only isolated case reports in the literature.[4,5] The concurrent presentation of meningitis with extensive myelopathy poses significant diagnostic and therapeutic challenges, particularly given the potential for severe neurological sequelae.

We present a case of HSV-2-induced viral meningomyelitis with extensive long-segment involvement from C5 to L2, demonstrating remarkable clinical and radiological recovery following prompt antiviral therapy. This case highlights the importance of maintaining high clinical suspicion for HSV-2 in young adults presenting with concurrent meningitis and myelitis, particularly when neurogenic bladder dysfunction is present.

Case Presentation

Patient Information and Chief Complaint

A 24-year-old previously healthy Caucasian woman presented to the emergency department with a two-week history of progressive neurological deterioration, characterized by weakness, urinary retention, and confusion.

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History of Present Illness

The patient's illness began insidiously with low-grade fever (maximum temperature 38.4°C) and generalized malaise. Over the subsequent 48-72 hours, she developed severe, throbbing, generalized headache accompanied by photophobia and phonophobia. During the week prior to presentation, family members observed progressive confusion and disorientation.

Concurrently, the patient reported neck stiffness and back pain radiating bilaterally to the lower extremities. Progressive bilateral lower extremity weakness developed, initially causing ambulatory difficulties and progressing to complete inability to bear weight. Urinary retention requiring catheterization occurred three days prior to admission. The patient denied any history of recent travel, tick exposure, or recent illness in contacts. She had no prior history of neurological disease, autoimmune conditions, or immunosuppression.

Physical Examination

On admission, vital signs revealed a temperature of 38.8°C, blood pressure 118/76 mmHg, heart rate 102 beats per minute, and oxygen saturation 98% on room air.

Neurological Examination

- Mental status: Lethargic but arousable, disoriented to time and place (Glasgow Coma Scale 10/15: E3M5V2)
- Cranial nerves: Intact bilaterally with mild photophobia; pupils equal, round, and reactive to light
- Motor examination: Upper extremities demonstrated normal strength (5/5) bilaterally; lower extremities showed marked weakness (2/5) bilaterally with hypotonia, more pronounced distally
- Sensory examination: Decreased pinprick and vibration sensation below the T10 dermatome bilaterally
- Reflexes: Lower extremity hyperreflexia (3+) with absent ankle jerks and bilateral Babinski signs
- Meningeal signs: Positive nuchal rigidity, Kernig's sign, and Brudzinski's sign
- Bladder examination: Suprapubic distension consistent with urinary retention

General Examination: No skin rashes, lymphadenopathy, or organomegaly were present.

Diagnostic Assessment

Laboratory Investigations: Complete blood count revealed leukocytosis with white blood cell count of 12,500/μL (normal: 4,000-11,000/μL) with lymphocytic predominance (65%), hemoglobin 12.1 g/dL, and platelet count 285,000/μL. Basic metabolic panel was within normal limits. Inflammatory markers were elevated: erythrocyte sedimentation rate 45 mm/hr (normal: <20 mm/hr) and C-reactive protein 15 mg/L (normal: <3 mg/L).

Cerebrospinal Fluid Analysis: Lumbar puncture revealed an opening pressure of 280 mmH_2O (normal: 70-180 mmH_2O). The fluid appeared clear and colorless. Cell count was 150 cells/μL (normal: <5 cells/μL) with 90% lymphocytes and 10% neutrophils. Protein level was elevated at 85 mg/dL (normal: 15-45 mg/dL), and glucose was 45 mg/dL with a CSF:serum glucose ratio of 0.55 (normal: >0.6). Gram stain was negative, and bacterial cultures showed no growth at 48 hours. Viral polymerase chain reaction panel was positive for HSV-2.

Serology: HSV-1 and HSV-2 antibodies demonstrated positive IgM and negative IgG for HSV-2, consistent with acute primary infection.

Additional Testing: Comprehensive autoimmune workup was negative, including anti-NMO (neuromyelitis optica) antibodies, anti-MOG (myelin oligodendrocyte glycoprotein) antibodies, and antinuclear antibodies.

Imaging Studies: Magnetic resonance imaging of the brain revealed focal demyelination at the cervicomedullary junction with findings suggestive of viral encephalitis. MRI of the spine demonstrated extensive T2 hyperintense signal changes with gadolinium enhancement extending from C5 to L2 vertebral levels, consistent with long-segment myelitis (Figure 1).

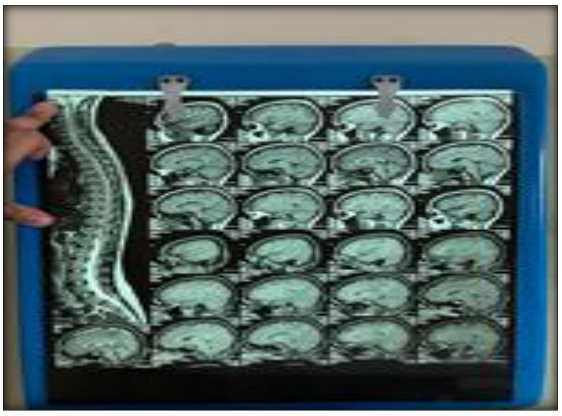

Figure 1: Magnetic resonance imaging of the spine. (A) Sagittal T2-weighted images demonstrating extensive hyperintense signal changes throughout the spinal cord from C5 to L2 vertebral levels, consistent with long-segment myelitis. (B) Sagittal T1-weighted post-gadolinium images showing enhancement of the affected spinal cord segments, indicating active inflammation.

Diagnosis

Based on the clinical presentation, cerebrospinal fluid findings, positive HSV-2 PCR, and characteristic imaging findings, a diagnosis of viral meningomyelitis secondary to HSV-2 infection with extensive long-segment myelopathy and neurogenic bladder dysfunction was established.

Treatment and Management

Acute Phase Treatment

Antiviral Therapy: High-dose intravenous acyclovir was initiated at 10 mg/kg every 8 hours for 14 days, with dose adjustment for renal function.

Anti-inflammatory Therapy: Given the extensive myelitis, intravenous methylprednisolone 1g daily

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for 3 days was administered, followed by a prednisone taper over 2 weeks.

Supportive Care: Management included placement of an indwelling urinary catheter for neurogenic bladder dysfunction, deep vein thrombosis prophylaxis, physical therapy consultation for mobility assessment, and occupational therapy for activities of daily living. Symptomatic treatment for headache and fever was provided.

Monitoring and Follow-up

Serial neurological examinations were performed to monitor treatment response. Repeat MRI of the spine was scheduled at 2 weeks and 8 weeks post-treatment. Urodynamic studies were planned for the post-acute phase to evaluate bladder function.

Clinical Course and Outcomes

Early Response (Days 1-14)

Clinical improvement was noted within the first week of treatment. Fever resolved by day 5, with progressive improvement in mental status. Headache and meningeal signs gradually diminished over the first two weeks.

Intermediate Recovery (Weeks 3-4)

Significant neurological improvement became apparent during weeks 3-4. Lower extremity strength improved from 2/5 to 4/5 bilaterally. Sensory deficits below T10 began to resolve, with improved pinprick sensation. The patient began to participate more actively in physical therapy.

Late Recovery (Weeks 6-8)

Near-complete motor recovery was achieved by week 8, with lower extremity strength returning to 5/5 bilaterally. The patient regained independent ambulation and could perform activities of daily living without assistance. However, neurogenic bladder dysfunction persisted, requiring continued catheterization despite normal urodynamic studies.

Follow-up Imaging

Repeat MRI of the spine at 8 weeks demonstrated remarkable radiological improvement with:

- Significant resolution of long-segment myelitis
- Minimal residual T2 hyperintensity
- Complete resolution of gadolinium enhancement

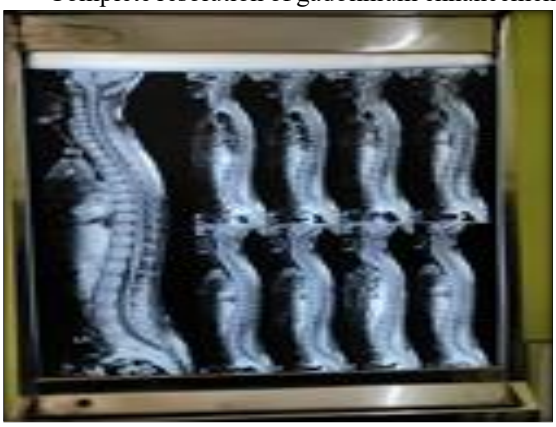

Figure 2: Follow-up magnetic resonance imaging at 8 weeks post-treatment. (A) Sagittal T2-weighted images showing significant resolution of the previously extensive myelitis with minimal residual hyperintensity. (B) Sagittal T1-weighted post-gadolinium images demonstrating complete resolution of enhancement, indicating resolution of active inflammation.

Figure 3: Normal uroflowmetry demonstrating adequate urinary flow parameters. Maximum flow rate of 8 ml/sec with normal voiding pattern and physiological pressure measurements (Pves 55 cmH2O, Pdet 51 cmH2O) effectively rules out obstructive causes. These findings support consideration of non-obstructive etiologies in the differential diagnosis.

Discharge and Long-term Follow-up

The patient was discharged ambulating independently with ongoing outpatient follow-up for neurogenic bladder management. At 6-month follow-up, motor function remained normal, but bladder dysfunction persisted, requiring intermittent catheterization.

DISCUSSION

This case illustrates a rare but clinically significant presentation of HSV-2 infection manifesting as concurrent viral meningitis and extensive myelitis. The extensive nature of the myelopathy spanning from C5 to L2 with associated neurogenic bladder dysfunction represents an uncommon manifestation of HSV-2 central nervous system infection.

Epidemiology and Pathophysiology

HSV-2 myelitis is exceptionally rare, with most cases presenting as isolated sacral myelitis or cauda equina syndrome related to sacral radiculitis.[6] Extensive long-segment myelopathy as demonstrated in this case is even rarer, with only scattered case reports in the literature.[7,8] The pathophysiology involves direct viral invasion of neural tissue with subsequent inflammatory response, leading to demyelination and axonal damage.

Clinical Presentation and Diagnosis

The insidious onset with fever, headache, and meningeal signs, followed by progressive lower extremity weakness and urinary retention, suggested a neuroinfectious etiology. The cerebrospinal fluid findings of elevated opening pressure, lymphocytic pleocytosis, and elevated protein were characteristic of viral meningoencephalitis. The positive CSF PCR for HSV-2 was crucial for definitive diagnosis and targeted therapy initiation.

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Differential Diagnosis

Myelopathy refers to spinal cord dysfunction that may result from a wide array of etiologies. The diagnostic approach involves systematic exclusion of compressive causes, vascular, inflammatory, infectious, metabolic, paraneoplastic, and developmental disorders, using both imaging and laboratory investigations. In the presented case of **extensive long-segment myelopathy with neurogenic bladder**, the final diagnosis of **HSV-2 viral meningomyelitis** was established through comprehensive evaluation and targeted diagnostic testing.

1. Compressive Causes

First and foremost, compressive etiologies must be ruled out through **MRI of the spinal cord with and without contrast**.

- Epidural, intradural, or intramedullary neoplasms
- Epidural abscess
- Epidural hemorrhage
- Cervical spondylosis
- Herniated intervertebral disc
- Posttraumatic compression due to vertebral displacement or hemorrhage

2. Vascular Etiologies

Spinal cord infarction or vascular malformations typically present acutely and may mimic infectious or inflammatory myelitis. Imaging is critical.

- Arteriovenous malformations, dural fistulas
- Antiphospholipid syndrome and other hypercoagulable conditions
- Spinal cord infarction—often anterior and abrupt in onset

3. Inflammatory Disorders

Inflammatory causes generally present subacutely and are associated with CSF abnormalities or distinct spinal cord MRI lesions.

- Multiple sclerosis (MS)
- Neuromyelitis optica spectrum disorders (NMO-SD) – Anti-aquaporin-4 antibodies
- Sarcoidosis – Consider serum ACE, calcium, chest imaging, biopsy
- Systemic autoimmune diseases:
  - Systemic lupus erythematosus (SLE)
  - Sjögren's syndrome
  - Behçet's disease
  - APL antibody syndrome
  - Vasculitides
- Other CNS inflammatory conditions:
  - Anti-MOG, anti-GFAP antibodies
  - CLIPPERS
  - Paraneoplastic myelitis (e.g., anti-CRMP5, anti-Hu, anti-amphiphysin)
  - Erdheim-Chester disease

4. Infectious Myelitis

Viral myelitis, including HSV-2, must be considered especially in cases with CSF pleocytosis, systemic symptoms, and long spinal cord lesions on MRI. This case was ultimately confirmed as **HSV-2 meningomyelitis**.

- **Viral:** HSV-2, HSV-1, VZV, CMV, EBV, HIV, HHV-6, HTLV-1
- **Bacterial/mycobacterial:** Borrelia, Listeria, Treponema pallidum, Brucella, Chlamydia pneumoniae, Bartonella, Mycoplasma pneumoniae
- **Parasitic:** Schistosoma, Toxoplasma, Cysticercus

CSF PCR and cultures play a central role in establishing the infectious etiology.

5. Developmental Conditions

- Syringomyelia
- Meningomyelocele
- Tethered cord syndrome

These are typically congenital and present with chronic symptoms.

6. Metabolic Disorders

- **Vitamin B_{12} deficiency** – Causes subacute combined degeneration
- **Copper deficiency**
- **Folate deficiency** These are often associated with dorsal column dysfunction and should be evaluated via serum levels.

7. Paraneoplastic Syndromes

Remote effects of malignancy due to immune-mediated mechanisms, associated with:

- Antibodies to CRMP5, Hu, amphiphysin
- Typically evaluated with autoantibody panels and cancer screening

8. Diagnostic Evaluation Strategy

A complete diagnostic workup is essential to differentiate the underlying etiology. In this case, imaging and laboratory investigations were used to **rule out compressive, inflammatory, autoimmune, neoplastic, and metabolic causes**, while **confirming HSV-2 infection** as the cause of meningomyelitis.

Key components include:

- **Neuroimaging:**
  - **MRI of the spine with and without contrast** (to exclude compressive and vascular causes)
  - **MRI brain** (to evaluate for multiple sclerosis or CNS involvement)
  - **CT myelogram, spinal angiography** (if vascular cause suspected)
- **CSF Analysis:**
  - Cell count, protein, glucose, IgG index, oligoclonal bands
  - PCR for HSV-2, VZV, CMV, EBV, enteroviruses
  - Gram stain, AFB stain, cultures (bacterial, fungal, mycobacterial)
  - VDRL, antibody testing for *Borrelia*, *Brucella*, *Chlamydia*, *Mycoplasma*
- **Serologic and autoimmune testing:**
  - ANA, ENA, dsDNA, SSA/SSB, rheumatoid factor, p-ANCA
  - Anti-aquaporin-4, anti-MOG, anti-GFAP antibodies
  - Vitamin B_{12}, copper, zinc levels
  - ACE, calcium (for sarcoidosis), ESR, CRP
- **Other Specific Investigations:**

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- Schirmer's test, salivary gland/lacrimal gland biopsy (Sjögren's)
- Slit-lamp exam, gallium scan
- Paraneoplastic panel (anti-Hu, CRMP-5, amphiphysin)

Treatment Response and Prognosis

The excellent neurological recovery, including near-complete resolution of the extensive myelopathy documented by follow-up imaging, underscores the importance of early recognition and prompt high-dose intravenous acyclovir therapy. While corticosteroids may have addressed the inflammatory component, antiviral therapy was likely the primary driver of recovery.

The persistent neurogenic bladder dysfunction despite excellent motor recovery highlights the potential for residual neurological sequelae, particularly affecting autonomic functions. This finding is consistent with previous reports suggesting that bladder dysfunction may be more resistant to treatment than motor symptoms in HSV-associated myelitis.[9]

Clinical Implications

This case emphasizes several important clinical points:

1. **High Clinical Suspicion:** Viral meningomyelitis requires high clinical suspicion, particularly in young adults with concurrent meningeal and spinal cord symptoms.
2. **Comprehensive Diagnostic Workup:** HSV-2 should be considered in young adults presenting with meningitis and myelitis, especially with neurogenic bladder dysfunction.
3. **Therapeutic Urgency:** Early antiviral therapy is paramount for optimal outcomes in HSV-associated CNS infections.
4. **Prognostic Considerations:** Extensive viral myelitis can be reversible with appropriate treatment, although some residual deficits may persist.
5. **Imaging Importance:** Spinal MRI is essential for documenting myelitis extent and monitoring treatment response.

Limitations

While motor recovery was excellent, the persistent neurogenic bladder dysfunction represents a significant limitation in this patient's recovery. Long-term urological follow-up and management remain important considerations. Additionally, the role of corticosteroids in HSV-associated myelitis remains controversial, and the contribution of methylprednisolone to the recovery in this case cannot be definitively established.

CONCLUSION

This case report demonstrates a rare presentation of HSV-2 viral meningomyelitis with extensive long-segment myelopathy and neurogenic bladder dysfunction. The rapid diagnosis through cerebrospinal fluid polymerase chain reaction and prompt initiation of high-dose acyclovir resulted in remarkable neurological recovery with significant radiological improvement.

The case underscores the critical importance of maintaining high clinical suspicion for HSV-2 in young adults presenting with concurrent meningitis and myelitis, particularly when neurogenic bladder dysfunction is present. Timely diagnosis and aggressive antiviral treatment are essential for preventing long-term neurological sequelae and achieving optimal patient outcomes.

The excellent motor recovery observed in this patient provides encouraging evidence for the potential reversibility of extensive HSV-2 myelopathy when appropriate treatment is initiated promptly. However, the persistent bladder dysfunction highlights the potential for residual autonomic deficits despite otherwise complete recovery.

This case contributes valuable insights to the limited literature on HSV-2-associated long-segment myelopathy and may guide clinical decision-making in similar presentations. Future research should focus on understanding the mechanisms underlying differential recovery patterns between motor and autonomic functions in HSV-associated myelitis.

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