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Reactivation of HSV-2 Leading to Elsberg Syndrome: A Case Report Highlighting Diagnostic and Management Challenges

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Abstract

Elsberg syndrome, or HSV-2 lumbosacral radiculitis, is a rare and underrecognized neurologic condition that mimics cauda equina syndrome (CES). It typically presents with symptoms such as urinary retention, saddle anesthesia, and bowel incontinence. This case report describes a 59-year-old immunosuppressed male with idiopathic pulmonary fibrosis who developed Elsberg syndrome due to re-activation of latent HSV-2. The patient experienced progressive lower extremity sensory deficits and genitourinary dysfunction, culminating in a vesiculopustular rash. Diagnosis was confirmed via cerebrospinal fluid analysis and PCR testing of skin lesions. Despite early imaging findings being unremarkable, subsequent MRI revealed enhancement of the conus medullaris and cauda equina. Treatment with intravenous acyclovir, corticosteroids, and supportive therapy led to gradual functional improvement, though sensory deficits and neuropathy persisted. This case highlights the diagnostic challenges and importance of clinical suspicion for HSV-2 reactivation in immunosuppressed patients, as well as considerations for long-term symptom management.

Keywords

Elsberg Syndrome, HSV-2 Reactivation, Lumbosacral Radiculitis, Immunosuppression

1. Introduction

Elsberg syndrome is an underrecognized neurologic disorder characterized by acute or subacute lumbosacral radiculitis, with or without myelitis of the lower

spinal cord [1]. Patients with this condition typically present with a constellation of symptoms resembling cauda equina syndrome (CES), including lower extremity weakness, bowel and bladder incontinence, urinary retention, saddle anesthesia, and/or sensory impairment in the lower extremities [2]. While several infectious etiologies have been implicated, Herpes Simplex Virus 2 (HSV-2) is the most common cause, occurring as either a primary infection or reactivation of latent virus [2] [3]. The presence of a vesiculopustular rash, alongside symptoms of CES, should raise clinical suspicion for Elsberg syndrome, also known as HSV-2 lumbosacral radiculitis. While HSV-2 has the potential to remain dormant in neurons throughout the central nervous system, prevalence of latent HSV-2 is noted to be greatest in the sacral ganglia [4]. Reactivation of the virus is frequently associated with immunocompromised states, including those caused by diseases such as HIV or malignancies, genetic disorders, or immunosuppressive medications [3] [5].

Although Elsberg syndrome is generally considered a self-limited condition, with severe symptoms often resolving within weeks, complete neurologic recovery is uncommon [2] [4] [6]. To date, there is no consensus on the optimal treatment regimen. Most patients are treated with antiviral medications, such as acyclovir, typically administered for 10 - 20 days [7]. The role of corticosteroids remains controversial; however, a short course is often employed to reduce inflammation and potentially shorten symptom duration [1] [2] [7].

In this report, we describe a case of subacute Elsberg syndrome in an immunocompromised patient.

2. Case Presentation

This patient was a 59-year-old male with a past medical history notable for coronary artery disease requiring stenting, chronic obstructive pulmonary disease, and idiopathic pulmonary fibrosis (on nintedanib and 10 mg Prednisone daily). He initially presented to the emergency department with a 3-day history of right leg numbness and intermittent shooting pains that radiated down the posterior aspect of his right thigh. He denied any bladder or bowel incontinence, saddle anesthesia, or muscle weakness. He also denied any tick bites, rashes, or illnesses prior to onset of symptoms. CT scan done at the time revealed only degenerative changes of the lumbar spine, most pronounced at L4-L5, with moderate spinal canal and bilateral foraminal stenosis. He was discharged with 50 mg Prednisone for 5 days and 5 mg Oxycodone for 3 days.

On day 5, the patient returned to the emergency department for progression of symptoms. He had now developed difficulty with ambulation due to right leg numbness in addition to genital and rectal numbness. He continued to deny any bladder and bowel incontinence, and the physical exam revealed 5/5 strength in bilateral lower extremities. Due to concern for cauda equina syndrome, an MRI of the lumbosacral spine without contrast was performed. The impression of this MRI was "Multilevel degenerative changes from L1-L5 superimposing what is likely a congenitally narrow canal. Findings are most pronounced at L4-L5 with

at least moderate central canal narrowing." The orthopedic spine service deemed no surgical intervention was necessary, and the patient was discharged home with instructions to follow-up with primary care.

On Day 18, the patient presented to the emergency department for a third time. His symptoms had now progressed to include paresthesia of the left leg, a bilateral burning sensation over the plantar aspect of both feet, subjective bilateral lower extremity weakness, erectile dysfunction, bowel incontinence, and urinary hesitancy. He now required the assistance of a cane for ambulation. He had also discontinued nintedanib at this time at the recommendation of his pulmonologist for concerns that it may be contributing to the patient's gastrointestinal symptoms. Physical exam revealed 5/5 strength and 2+ reflexes in all upper and lower extremities with notable bilateral upgoing plantar reflexes. Additionally, the patient had decreased pinprick sensation throughout his legs and decreased vibratory sense in bilateral knees, ankles, and toes. Sensation to light touch and proprioception was intact. All vital signs were within normal limits. He was admitted to the neurology service for further workup.

On admission, complete blood count showed a white blood cell count of 11.2 × 10^3/uL, hemoglobin of 16.6 g/dL, and platelets of 192 × 10^3/uL. The basic metabolic panel was within normal limits. MRI of the cervical, thoracic, and lumbosacral spine with and without contrast was performed due to symptom progression. Results revealed mild, diffuse, non-nodular enhancement to the conus that extended to the cauda equina nerve roots best appreciated on the sagittal post contrast images. There was no abnormal enhancement identified within the cervical and upper thoracic spine. Two days later he also had an MRI brain with and without contrast that showed nonspecific hyperintense T2 white matter signal changes with no acute intracranial process.

On Day 21, the patient developed a small, vesicular rash on an erythematous base on his chest, back, and medial aspect of his left thigh. Only the thigh rash was pruritic. By Day 22, the rash had continued to progress and now included a pustular rash on the dorsal aspect of the left foot. The development of this rash prompted further evaluation via lumbar puncture. Results showed protein of 98 mg/dL, glucose of 52 mg/dL, lactic acid of 1.6 mmol/L, and 67 nucleated cells. Biofire analysis of CSF fluid returned positive serology results for Herpes Simplex Virus 2 (HSV-2). An encephalopathy panel was also performed on the CSF which tested for MPA-R, amphiphysin, AGNA-1, ANNA-1, ANNA-2, ANNA-3, CASPR2-IgG, CRMP-5-IgG, DPPX, GABA-B-R, GAD65, GFAP, mGluR1, IGLON5, LGI1, neurochondrin, NIF, NMDA-R, PCA-TR, PCA-1, PCA-2, PDE10A, SETPTIN-7, and TRIM46 antibodies, all of which were negative. A PCR swab collected from the lesions of the left foot was also positive for HSV-2. Due to the clinical symptomatology, radiographic findings, and serology results, a diagnosis of HSV-2 lumbosacral radiculitis (Elsberg Syndrome) was made at this time. Of note, the patient did have positive IgG titers for HSV-2 over one year prior to this admission, indicating this was not a sequelae of a primary infection, but rather,

secondary reactivation.

The patient was then started on 14 days of IV acyclovir 10 mg/kg Q8H, 5 days of IV methylprednisolone 1000 mg, and cyanocobalamin 1000 mcg. Pain control was managed with Pregabalin 75 mg PO TID and hydrocodone-acetaminophen 5 - 325 mg Q6H as needed while inpatient. The patient was discharged home after completion of the 5-day course of steroids. Post-discharge, the patient experienced very gradual, but significant improvement of functional status with respect to ambulation and continence, however, he still had persistence of some sensory symptoms. He also restarted nintedanib approximately one-month post-discharge for the management of idiopathic pulmonary fibrosis. Outpatient evaluation on day 130 revealed that the patient was ambulating without a cane or any assistive device approximately 90% of the time. Although not as severe as during his admission, the patient still endorsed persistent neuropathy and paresthesias managed with a regimen of duloxetine 60 mg daily, pregabalin 150 mg 3× daily, and valacyclovir 500 mg twice daily for prophylaxis.

3. Discussion

This case report describes the reactivation of latent HSV-2, which was thought to be triggered by an immunosuppressed state secondary to treatment for idiopathic pulmonary fibrosis. The patient's presentation exemplifies many of the diagnostic challenges associated with this condition, as he experienced a gradual progression of symptoms. Characteristic features of cauda equina syndrome did not develop until day 18 of his illness, despite initial unremarkable imaging findings.

The delay in imaging findings is a common and frustrating aspect of Elsberg syndrome. MRI findings, such as enhancement and swelling of the sacral nerve roots, are primarily caused by viral-induced inflammation. The inflammatory response to HSV-2 infection is believed to result in a breakdown of the blood-nerve barrier, leading to gadolinium enhancement on MRI. This enhancement reflects increased vascular permeability and inflammation of the nerve roots. The swelling of the sacral nerve roots is attributed to inflammatory edema and cellular infiltration associated with the viral infection [8] [9].

Because MRI findings in Elsberg syndrome are not specific, the differential diagnosis can be extensive. In this case, the differential included Guillain-Barré syndrome (GBS), multiple sclerosis (MS), neuromyelitis optica (NMO), and other infectious or autoimmune etiologies. A comprehensive workup was pivotal in confirming the diagnosis. This included ruling out demyelination on imaging, demonstrating normal nerve conduction studies, and detecting HSV-2 DNA in the CSF via PCR. The most crucial diagnostic step was a lumbar puncture, which ruled out MS due to the absence of oligoclonal bands and made GBS less likely given the presence of lymphocytic pleocytosis. NMO and other etiologies were excluded using an encephalopathy panel performed on the CSF.

For this patient, the development of a vesiculopustular rash on an erythematous base ultimately prompted testing for HSV-2 in the CSF and skin lesions. While a

rash is not always present in Elsberg syndrome, its emergence can be an important diagnostic clue in patients presenting with signs of lumbosacral radiculitis. Once the diagnosis was confirmed, treatment with IV acyclovir was initiated—22 days after symptom onset and 19 days after the patient first sought care. The treatment regimen of IV acyclovir 10 mg/kg every 8 hours for 14 days was selected based on CDC guidelines for severe HSV infections with CNS complications [10]. Given the patient's known history of HSV-2, long-term immunosuppression, and the onset of neurologic symptoms, it is arguable that earlier initiation of antiviral therapy might have improved outcomes. A study by Erdem *et al.* underscores the importance of rapid diagnosis and early antiviral treatment in herpetic meningoencephalitis, a condition with pathophysiological similarities to Elsberg syndrome. Delayed treatment is associated with unfavorable outcomes, emphasizing the need for prompt intervention [11]. Due to this patient's ongoing immunosuppressed state and the significant complication he had from HSV, indefinite prophylactic antiviral therapy was initiated to prevent future reactivation.

The progressive nature of the patient's symptoms also warranted treatment with IV methylprednisolone 1000 mg daily for 5 days. Multiple case reports have demonstrated significant improvement in neurologic symptoms in patients with HSV encephalitis and Elsberg syndrome following steroid therapy [12] [13].

Following hospitalization, the patient's care included management of neuropathic symptoms with duloxetine 60 mg daily and pregabalin 150 mg three times daily. While these therapies are not unique to Elsberg syndrome, they have proven effective in this case for mitigating the long-term neuropathic pain associated with the condition. Tavanaei *et al.* have described severe axonal damage and radiculopathy observed in electromyography and nerve conduction studies, which can lead to chronic pain and sensory deficits [8].

This case highlights the diagnostic challenges and therapeutic considerations in managing Elsberg syndrome, particularly in the context of immunosuppressed patients.

4. Conclusion

Elsberg syndrome is a well-established but poorly recognized cause of cauda equina-like symptoms, including urinary retention, saddle anesthesia, and bowel incontinence, as seen in this patient. While its overall prevalence is low, it accounts for up to 10% of all cases of cauda equina syndrome, highlighting the importance of early clinical detection [2]. Diagnosis relies on the constellation of clinical, radiographic, and serologic findings, as absolute diagnostic criteria have not yet been established. MRI is often a valuable tool, frequently revealing contrast enhancement of the sacral nerve roots and hyperintense absorption within the conus medullaris [8]. Early recognition and initiation of intravenous acyclovir, particularly in immunosuppressed patients, are crucial to reducing symptom duration and morbidity [1]. While full neurologic recovery is often not achieved, functional status, incontinence, and severe sensory deficits typically improve,

though residual neuropathy and paresthesia may persist [4] [6]. This case highlights the efficacy of a multi-drug regimen of duloxetine and pregabalin for managing chronic neuropathic pain in the outpatient setting, offering a safer and more favorable alternative to opioid-based therapies. Additionally, antiviral prophylaxis, possibly at the onset of immunosuppressive therapy, can be essential in patients to prevent HSV-2 reactivation [10]. Future research should focus on establishing standardized diagnostic criteria, standardizing treatment regimens, evaluating adjunctive therapies like corticosteroids, and exploring the long-term management of residual symptoms and appropriate antiviral prophylaxis duration. This case contributes to the growing body of evidence on Elsberg syndrome and underscores the clinical implications of early intervention and comprehensive management strategies.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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