Cytomegalovirus transverse myelitis in a non-immunocompromised patient

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INTRODUCTION

Transverse myelitis is a neurological disorder characterized by inflammation of the spinal cord. The inflammation traverses the spinal cord across one or multiple levels and results in destruction of myelin [1]. Cytomegalovirus (CMV), most commonly infects immunocompromised hosts. Its clinical spectrum includes retinitis, pneumonitis, hepatitis, and may cause transverse myelitis in some cases [2, 3]. We present a rare case of a transverse myelitis in a non-immunocompromised patient secondary to CMV infection.

A previously healthy 34-year-old Filipino male with no significant past medical history presented to the emergency department with fever 103.7°F, nausea, urinary retention, and bilateral hip pain for eight hours. He denied taking any medications. His social history was negative. His family history was not contributory. On physical examination, the patient was alert and in some
distress. The only significant finding on examination was a boggy, non-tender prostate and an enlarged bladder. The neurologic examination was unremarkable. The white blood cell count was 8,700/μL, hemoglobin 14.4 g/dL, platelets 175,000/μL, international normalization ratio (INR) was 1.1, and C-reactive protein was 0.78 mg/dL. Serum electrolytes were remarkable for sodium of 134 mmol/L and chloride of 97 mmol/L.

CASE REPORT

On hospital day-1, the patient developed a rapidly ascending weakness from his toes to the lower abdomen along with diplopia, perioral numbness, and urinary retention. He was started on levofloxacin, acetaminophen, tamsulosin, and intravenous (IV) hydration. A Magnetic resonance imaging of the spine showed a non-enhancing T2 signal abnormality in the cord extending from level C5 to the upper endplate of C7 without cord expansion or edema (Figure 1) and an increase in signal intensity in the mid-thoracic and upper lumbar spinal cord regions (Figure 2). Methylprednisolone 1000 mg IV was administered three times a day for four days. A few hours later, slurred speech and facial numbness were noted with progression to pinprick sensation loss up to T11 dermatome, bilateral leg weakness of 0/5, and diminished tone in lower extremities. There was also mild dysmetria on finger-to-nose testing and loss of rectal tone. Human immunodeficiency virus antibody I and II, Human T-Lymphocyte virus I and II were negative. Urinalysis and urine toxicology screen were negative. No enteric pathogens were detected in the stool sample. Blood cultures showed no growth. Direct flu antigen A and B, herpes simplex virus 1 and 2 DNA were undetectable. Serum Epstein–Barr virus IgG was 1, 270 mg/dL (normal: <0.90). Epstein–Barr virus Antibody Vca IgG titer was 1.84 (normal: <0.90). Epstein–Barr virus antibody Vca IgM titer was 2.96 (normal: <0.90). Serum antibody Lyme titer was undetectable. Human immunodeficiency virus antibody I and II, Human T-Lymphocyte virus I and II were negative. Urinalysis and urine toxicology screen were negative. No enteric pathogens were detected in the stool sample. Blood cultures showed no growth. Direct flu antigen A and B, herpes simplex virus 1 and 2 DNA were undetectable. Serum Epstein–Barr virus IgG was 1, 270 mg/dL (normal: <0.90). Epstein–Barr virus Antibody Vca IgG titer was 1.84 (normal: <0.90). Epstein–Barr virus antibody Vca IgM titer was 2.96 (normal: <0.90). Serum antibody Lyme titer was undetectable. Cerebrospinal fluid analysis was significant for a non-reactive venereal disease research laboratory test (VDRL). The white blood count was 26/mm³; polymorphonuclear cells 32% and mononuclear cells 68%. Lyme antibody titer was negative. Protein was 150 mg/dL. It also contained well defined gamma restriction bands that were also in the corresponding serum sample, but some bands in the cerebrospinal fluid (CSF) were more prominent. The CSF IgG titer was 20.7 mg/dL and IgG synthesis rate was 25.3 mg/24 hours. Cytomegalovirus DNA titer by rapid polymerase chain reaction (PCR) was 40,787. This suggested a transverse myelitis secondary to CMV. Thereafter, five plasmapheresis treatments were undertaken every other day. Ganciclovir 375 mg IV q12h for four days was also given. Despite such measures, his respiratory status started to decline and he was placed on bilevel positive airway pressure (BiPAP) support. Also, intravenous immune globulin 40 g IV every other day for a total of five treatments was prescribed. Subsequently, the respiratory status improved and he no longer required BiPAP support. On hospital day 18, paraplegia persisted with urinary and fecal incontinence and he was transferred to a rehabilitation facility. After 10 weeks, he was able to ambulate with a walker.

DISCUSSION

Transverse myelitis is characterized by inflammation of the spinal cord resulting in the destruction of myelin. The scar formation results in neuronal signal disruption [1]. Cytomegalovirus (CMV), a herpes virus, typically affects immunocompromised hosts. It may cause retinitis, pneumonitis, and hepatitis [2].

Transverse myelitis may result in weakness, sensory loss, and autonomic dysfunction below the level of the spinal cord lesion [3]. The signs and symptoms in the case described above are urinary retention, loss of strength, and sensory loss below the level of the lesion.
sensation from the umbilicus to the toes, and paraplegia in the lower extremities. Magnetic resonance imaging results revealed a non-enhancing T2 signal abnormality involving spinal segments C5 to C7, mid-thoracic and upper lumbar region consistent with transverse myelitis. Our patient met the criteria of the variant longitudinally extensive transverse myelitis (LETM) [4, 5]. Recent literature suggests that 40–50% of transverse myelitis cases may present without significant CSF findings [1, 6, 7]. Additionally, transverse myelitis may not meet diagnostic criteria for signs of inflammation [8]. The absence of inflammatory markers does not rule out the possibility of transverse myelitis when the clinical picture is suggestive of transverse myelitis [8].

There is a reported case of CMV transverse myelitis in a 40-year-old immunocompetent Sri Lankan male who presented with a two-day history of fever with bilateral lower extremity flaccid paralysis without urinary retention, but within five days, the paralysis progressed proximally with significant urinary retention [9]. A spinal MRI scan demonstrated hyperintensities on C5 and T2 cord segments and the CSF had WBC of 350/mm³ (92% lymphocytes). The patient’s lower extremity strength improved from flaccid paralysis to anti-gravity muscle strength after 21 days of intravenous ganciclovir treatment. Of note, both patients were Asian men. This group may be prone to CMV transverse myelitis [10, 11] (Table 1).

Table 1: Diagnostic Criteria for Transverse Myelitis.

| Diagnostic Criteria for Transverse Myelitis |
|--------------------------------------------|
| • Sensory, motor or autonomic dysfunction attributable to the spinal cord |
| • Bilateral signs and/or symptoms |
| • Clearly define sensory level |
| • No evidence of compressive cord lesion |
| • Inflammation defined by cerebrospinal fluid pleocytosis or elevated IgG index or gadolinium enhancement |
| • Progression to nadir between fours and 21 days |

CONCLUSION

Transverse myelitis is a potentially debilitating disease. Approximately one-third of patients with transverse myelitis experience full recovery, one-third experience partial recovery, and the remaining have no recovery. Our patient’s neurological symptoms have improved since initial presentation approximately three months ago. Future studies on pathophysiology of transverse myelitis may be required to develop a standard medical management protocol to prevent morbidity and mortality associated with cytomegalovirus associated transverse myelitis.
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