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Constipation in transverse myelitis

Justine Chinnappan , Abinash Parajuli, Huda Marcus, Ghassan Bachuwa

Department of Internal
Medicine, Hurley Medical Center,
Flint, Michigan, USA

Correspondence to
Dr Ghassan Bachuwa;
gbachuw2@hurleymc.com

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SUMMARY

Transverse myelitis is an extremely rare neuroinflammatory disorder. About half of the affected patients develop paraplegia associated with urinary and bowel dysfunction. The bowel dysfunction is thought to be benign and is usually managed with dietary management and laxatives. We report a case of a man in his 60s presenting with transverse myelitis and the acute disease course complicated with treatment refractory intestinal dysfunction resulting in intestinal perforation, eventually leading to his death. Thus, this case helps us weigh the fact that intestinal dysfunction in the case of transverse myelitis is not always benign but can lead to deadly outcomes as well.

BACKGROUND

Transverse myelitis is a rare neuroinflammatory condition affecting the spinal cord. Clinical features include acute to subacute onset of neurological symptoms involving sensory, motor and autonomic dysfunction. Though any level of the spinal cord can be affected, the thoracic spine is the most common area of involvement, affecting bilateral lower extremities exhibiting upper and lower motor neuron signs with bladder and bowel disturbances.¹ The symptoms peak within 4 hours–21 days and recovery starts at 1–3 months. Only around 33% recover with little to no residual deficits, while the rest of the population suffers from long-term debilitating neurological deficits. During the acute phase, almost 50% of the patients are paraplegic with associated bladder and bowel dysfunction.² Urine retention is treated with indwelling catheters, while constipation is treated with high fibre and laxatives until recovery.³ Transverse myelitis with severe bowel dysfunction unresponsive to usual treatment to the extent of causing bowel necrosis and perforation is rare. We present a case of such significance to emphasise the deadly outcome of severe bowel dysfunction in patients with transverse myelitis in the acute phase.

CASE PRESENTATION

A man in his 60s presented with bilateral lower extremity weakness (right > left) and abdominal pain. Significant medical history includes oxygen-dependent chronic obstructive pulmonary disease, cardiac arrest 4 years before presentation with good recovery, peripheral vascular disease with stenting in the left leg in the past, recently diagnosed untreated hepatitis C infection with remote intravenous drug use, hypertension, heart failure with preserved ejection fraction, splenectomy and multiple abdominal surgeries due to haemorrhagic complication of a motor vehicle accident. His outpatient medication

includes aspirin, atenolol, furosemide, budesonide nebuliser, albuterol and umeclidinium inhaler. The patient has been experiencing progressive weakness in the right leg followed by the left leg with bilateral lower extremity numbness associated with twitching that started around 10 hours before presentation. He has also been experiencing dyspnoea and productive cough with greenish sputum for 3-week duration for which he received outpatient treatment with cefuroxime. On presentation, he was hypertensive 226/121 mm of Hg, afebrile with bilateral wheeze on respiratory examination and right upper quadrant tenderness of the abdomen. On neurological examination, he was alert and oriented, had normal finger–nose test and no dysmetria; had intact cranial nerve function, unsteady on feet, right lower extremity strength $\frac{3}{5}$ while left lower extremity strength was $\frac{4}{5}$; fasciculation, impaired light touch sensation, diminished deep tendon reflexes and positive Babinski sign on the bilateral lower extremity. Initial laboratory workup revealed white cell count 15×10^9 cells/L, aspartate aminotransferase/alanine transaminase 44/83 U/L, B_{12} 955 pg/mL. Microbiology workup revealed respiratory pathogen panel test was positive for human enterovirus/rhinovirus, hepatitis C virus viral load of 5 523 781 IU/mL with genotype 1a, while HIV antibody and syphilis were negative. Cerebrospinal fluid (CSF) analysis showed white cell count 36 cells/cu.mm, glucose 60 mg/dL and protein 68 mg/dL, IgG index 0.71 mg/dL, negative meningitis profile, culture, oligoclonal bands, Venereal disease research laboratory test (VDRL), paraneoplastic antibodies and IgM West Nile antibodies. On repeat examination within 10 hours of presentation, he demonstrated progressive bilateral lower extremity weakness and acute urinary retention without the sensation of bladder fullness; hence, indwelling urinary catheter was placed. Within 2 days of presentation, symptoms progressed to complete loss of sensation and flaccid paralysis, absent deep tendon reflexes of bilateral lower extremities and $\frac{4}{5}$ strength in bilateral upper extremities, while the sensory level ascended to level T7 with progressive difficulty taking deep breath, and patient care was escalated to intensive care. On day 4 of the presentation, there was impaired pinprick sensation demarcated at the level of T4 and remained stable.

INVESTIGATIONS

MRI of the head was negative for acute infarction. MRI of the cervical, thoracic, and lumbar spine with and without contrast revealed multilevel T2 hyperintense lesions in the thoracic spinal cord involving the central grey structure concerning for infectious myelitis (figures 1–3). CT of the abdomen



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Figure 1 MRI of the thoracic spine sagittal T2-weighted image showing multilevel hyperintense signal change involving the central grey matter.

and pelvis showed dilated ascending colon measuring 10.9 cm in largest diameter with evidence of pneumatosis without signs of bowel obstruction or free intraperitoneal air (figure 4).

DIFFERENTIAL DIAGNOSIS

Given the sudden onset, cerebrovascular accident was first ruled out by MRI of the brain and CT angiography of the head and neck. Further working diagnoses included Guillain-Barre syndrome (GBS) and bacterial meningitis, which were ruled out clinically due to bowel and bladder involvement as well as by CSF studies. Then, the patient underwent MRI of the cervical, thoracic and lumbar spine to evaluate for post-viral autoimmune

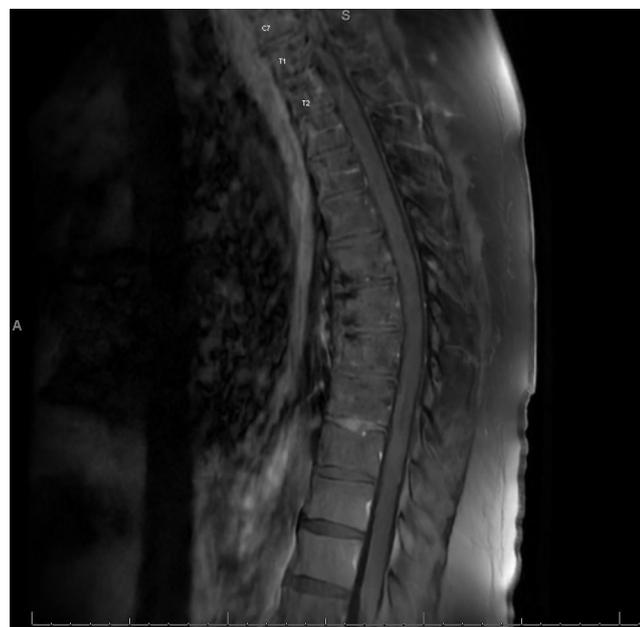


Figure 2 MRI of the thoracic spine sagittal post-contrast T1-weighted image showing no contrast enhancement.

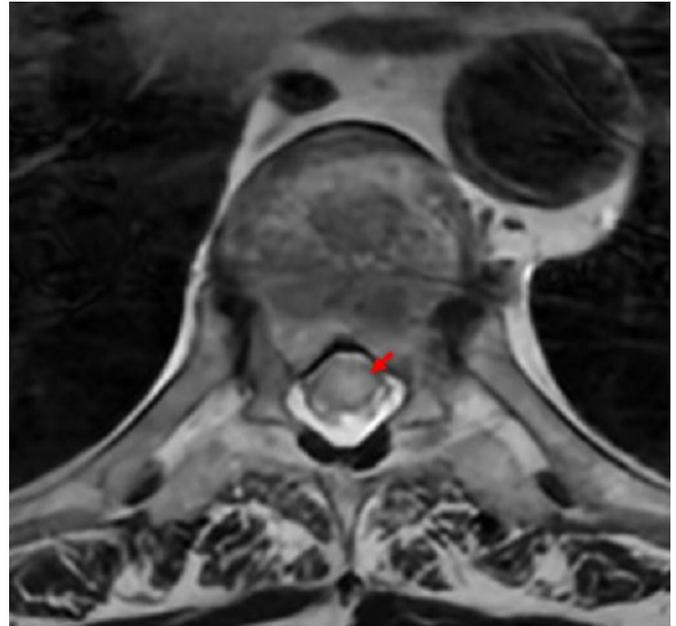


Figure 3 MRI of the thoracic spine axial T2-weighted image showing hyperintense lesion involving the central grey matter (red arrow) of the spinal cord.

or inflammatory process and compressive myelopathy, which showed multilevel T2 hyperintense lesions in the thoracic spine with no evidence of compression.

Thus, the presence of motor, sensory as well as autonomic dysfunction, bilateral involvement, reaching a nadir in 4 days with clearly delineated sensory level at T4, CSF pleocytosis and the MRI finding without evidence of compression lead us to the diagnosis of transverse myelitis.

TREATMENT

Given the initial working diagnosis of GBS, he was treated with intravenous immunoglobulin. The hospital course was complicated further with persistent constipation. The patient failed to have bowel movement until day 6 with multiple bowel regimens including enema and intravenous neostigmine to enhance bowel motility. This led to increased abdomen pressure causing abdominal compartment syndrome with measured intra-abdominal pressure 18 mm Hg and renal failure requiring renal

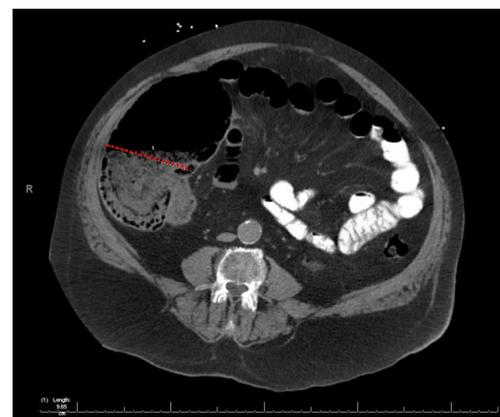


Figure 4 CT of the abdomen and pelvis showing dilated ascending colon with evidence of pneumatosis (red dotted line) on day 6 of admission.



Figure 5 CT of the abdomen and pelvis showing free intraperitoneal air (red arrows) on day 10 of admission.

replacement therapy. CT of the abdomen and pelvis revealed dilated ascending colon measuring 10.9 cm in largest diameter with evidence of pneumatosis without signs of bowel obstruction or free intraperitoneal air. Hence, nasogastric tube and colonoscopic decompression with the placement of the rectal tube were done to relieve constipation, but he did not have any bowel movement until day 10. He rapidly continued to deteriorate with hypotension requiring pressor support and leucocytosis, leading to further evaluation, which revealed intraperitoneal free air on CT of the abdomen (figure 5). The patient underwent emergent exploratory laparotomy, which revealed multiple areas of focal bowel necrosis and perforation. Hence, right hemicolectomy was done. Later, he underwent three more abdominal surgeries including transverse and descending colectomy due to severe dilatation and necrosis, ileostomy creation and multiple washout before complete closure of the abdomen on day 17.

OUTCOME AND FOLLOW-UP

The postoperative course was complicated by bleeding from the ileostomy with an output of around 500 mL per 24 hours. The patient's condition continued to deteriorate with multi-organ failure, requiring multiple pressor support due to septic shock, and the patient code status changed to comfort measures only per family request. The patient died 19 days after initial presentation.

DISCUSSION

Transverse myelitis is a rare neuroinflammatory disorder with an estimated incidence of 1–8 new cases/1 000 000 people per year with no gender predominance.^{4–7} The aetiology is varied and idiopathic is the most common with no identified underlying cause.⁸ Other causes are as follows: post-infectious due to infections but not limited to enterovirus, West Nile virus, varicella-zoster virus and herpes virus⁹; systemic autoimmune disorders like systemic lupus erythematosus, ankylosing spondylitis, sarcoidosis, Sjögren syndrome and rheumatoid arthritis; and finally, central nervous system autoimmune diseases like multiple sclerosis, myelin oligodendrocyte glycoprotein antibody-associated disease, neuromyelitis optica spectrum disorder and acute disseminated encephalomyelitis.⁹

The diagnostic criteria include the presence of bilateral sensory, motor and autonomic dysfunction in the absence of evidence of cord compression by MRI, with a clearly defined sensory level, positive markers for spinal cord inflammation including CSF pleocytosis, gadolinium enhancement (T2 hyperintense changes) or elevated IgG index and rapidly progressing course reaching a nadir between 4 hours and 21 days.¹⁰ The course of the disease involves partial recovery beginning at 1–3 months from the onset with continued exercise and physical therapy.¹¹ Treatment involves intravenous glucocorticoid therapy, treatment of the underlying cause, plasma exchange or intravenous immunoglobulins in resistant cases.^{1 12 13}

The complication of transverse myelitis can be classified based on the phase of the disease. In the acute phase, the most common complications are neurogenic acute urinary retention and constipation, and spinal shock, while chronic complications include major depression, recurrent or chronic urinary tract infection, and decubitus ulcers from paraplegia.

Though constipation is commonly reported, transverse myelitis causing severe constipation to the extent of resulting in bowel necrosis and perforation is rare. The bowel dysfunction results from involvement of parasympathetic, sympathetic and motor nerves. Parasympathetic involves the vagus nerve, which supplies the foregut–midgut and S2–4 nerves, which supply the hindgut resulting in slow transit time. Sympathetic involves T5–L2 controlling the internal anal sphincter, while the pudendal nerve affects the external anal sphincter and sensory input.¹⁴ In combination, the slow transit and increased sphincter tone, in conjunction with loss of sensation of rectal fullness, can result in severe constipation. Evaluation begins with review of bowel habits prior to the disease onset, medication review, digital rectal examination to assess rectal tone and faecal impaction, and determination of severity with scoring systems like the Cleveland Constipation Score. Investigations, though not always performed, include radiographic colonic transit time testing and anorectal physiological testing.¹⁵

Initial management for constipation in transverse myelitis or any neurogenic bowel dysfunction includes conservative bowel regimen with fluids, bulk-forming agents, stool softeners, osmotic laxatives, colonic stimulants, rectal stimulants and enema.^{3 16} When this fails, transanal irrigation, electrical stimulation, surgical antegrade colonic irrigation and stoma formation may be tried.¹⁵

Once acute colonic pseudo-obstruction sets in, laxatives should be avoided. Acute colonic pseudo-obstruction without signs of bowel ischaemia or perforation is initially treated with supportive care including bowel rest, electrolyte correction and fluid resuscitation. If the supportive measure fails, neostigmine push or colonoscopy decompression can be performed. When all of the above-mentioned methods are futile, and the caecal diameter is more than 12 cm for more than 6 days or there is evidence of bowel ischaemia and perforation, emergent surgery is warranted.¹⁷

We believe our patient developed post-infectious transverse myelitis due to enterovirus infection with the acute phase complicated by bowel dysfunction resulting in colonic pseudo-obstruction, which was refractory to neostigmine and colonoscopy decompression, progressing to bowel necrosis and perforation. This is the first case to report transverse myelitis complicated by acute pseudocolonic obstruction leading to abdominal compartment syndrome and bowel necrosis with perforation. Though constipation is considered benign in transverse myelitis, this case represents the extreme spectrum and emphasises the fact that constipation in individuals with transverse myelitis can lead to deadly outcomes as well.

Learning points

- ▶ Addressing bowel and bladder dysfunction is crucial in transverse myelitis.
- ▶ Treatment for bowel dysfunction includes treating the underlying condition as well as supportive care including increased fluid intake, dietary fibre that has bulking effect, laxatives and enema.
- ▶ Colonic pseudo-obstruction is managed conservatively with bowel rest, electrolyte correction, fluid resuscitation, neostigmine and colonoscopy decompression until caecal diameter is more than 12 cm for more than 6 days, or there is evidence of bowel ischaemia/perforation when emergent surgery is warranted.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

ORCID iDs

Justine Chinnappan <http://orcid.org/0000-0002-6643-2549>

Ghassan Bachuwa <http://orcid.org/0000-0001-8414-0067>

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