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Case Report

Transverse Myelitis in a Rural Australian Emergency Department: Case Report

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A B S T R A C T

Acute transverse myelitis (ATM) is a rare neurologic condition with rapid onset of weakness, sensory changes, and occasional bowel or bladder symptoms. This case report will explore a male patient presenting with symptoms consistent with ATM to a rural Australian emergency department. This case report explores the diagnosis of ATM with further discussions on the difficulties and importance of the nurse practitioner–physician relationship surrounding patient care. This case report also outlines the difficulties of providing care to patients in the rural hospital setting, along with a focus on clinical considerations that nurse practitioners encounter in day-to-day practice.

Brief Introduction

Acute transverse myelitis (ATM) is a rare immune-mediated neurologic condition. It involves an inflammatory response to the spinal cord resulting in neurologic injury to the affected area.[1-3] ATM is a rare disease process with incidence rates reported between 1 and 8 new cases per 1,000,000 people.[2] However, a recent retrospective study revealed a point prevalence of 7.86 cases per 100,000 people.[4] There have been reports that ATM has a bimodal peak between ages 10 and 19 years and between ages 30 and 39 years.[5]

Case Presentation

Chief Complaint

A 59-year-old man with the pseudonym *John* reported an acute onset of painless left leg weakness while walking. The patient reported 20 hours of left leg weakness with associated paresthesia and an inability to weight bear. There were no other complaints or symptoms of headache, seizurelike activity, dizziness, blurred vision, facial droop, or upper limb weakness. John's symptomology led him to present to his local rural emergency department for review.

History of Present Illness

John reported first noticing his symptoms the day prior as he was leaving work. On standing to walk, his leg felt weak, dragging gait with no other neurologic symptoms. On waking the following morning, John became increasingly concerned due to the increased severity of left leg weakness to the point of being unable to weight bear. John also reported new-onset paresthesia; however, the description was vague and not isolated to a particular dermatome(s) of the left leg. John declined that the weakness and paresthesia occurred in an ascending pattern. These symptoms were constant, with no aggravation or relieving factors. John also stated that he was incontinent of urine while attempting to walk to the toilet, which was new to him.

Medical History

John's medical history was inclusive of hypertension, type 2 diabetes mellitus, and gout. The patient did not report any previous cardiovascular events or episodes of incontinence.

Medications

The patient had no known allergies and took regular medications inclusive of allopurinol 300 mg once daily, olmesartan 40 mg once daily, amlodipine 10 mg once daily, hydrochlorothiazide 12.5 mg once daily, and metformin 1 g twice daily, all taken orally.

Family History

The patient denies any family cardiovascular history or inherited neurologic disorders.

Personal/Social/Developmental History

The patient is independent with mobility and activities of daily living, lives at home with his wife, and works as a maintenance worker at the local hospital. He reports consuming approximately 4

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standard alcoholic beverages per week. He denies smoking or recreational drug use.

Review of Symptoms

There were no other complaints of headache, confusion, seizurelike activity, dizziness, blurred vision, facial droop, fecal incontinence, or upper-limb weakness. The patient did not report any recent infective symptoms, pain, or fever. There were no complaints of chest pain, palpitations, dyspnea, and diaphoresis.

Pertinent Physical Examination Findings

John's vital signs included a respiratory rate of 18 breaths/min, heart rate of 90 beats/min, temperature of 36.1 °C, blood pressure of 149/66 mm Hg, oxygen saturation of 98% on room air, and blood glucose level of 218.02 mg/dL (12.1 mmol/L). Cardiac examination revealed S1 and S2 regular heart sounds without clicks or murmurs, and the electrocardiogram demonstrated a normal sinus rhythm. Respiratory examination was clear lung auscultation, equal chest rise and fall, with no increased effort or accessory muscle use. His abdomen was soft and nontender, with no palpable masses or hepatomegaly noted. Neurologically, the patient was alert and orientated, with a Glasgow Coma Scale score of 15 and normal cranial nerve examination findings. Upper limbs: equal strength, normal sensation and coordination throughout; lower limbs: right normal strength, sensation, and coordination. The left leg had 4/5 strength throughout with ataxia, hyporeflexia, and gross paresthesia to light touch and pinprick that was not isolated to a particular dermatome(s). Written consent was obtained and the patient had unilateral saddle anesthesia, and anal tone was intact. The lower limbs bilaterally were warm, with strong peripheral pulses throughout and appropriate capillary refill. The paresthesia resolved while in the emergency department; however, left leg neurologic symptoms remained.

Diagnostic Studies

John was triaged as category 2 and seen immediately in the emergency resuscitation bay and was discussed with the telestroke service and underwent computed tomography (CT) of the brain and angiography, which returned normal results. The initial pathology, including full blood cell count, urea and electrolytes, liver function testing, coagulation studies, C-reactive protein, and venous blood gas, were grossly normal. It was decided that a CT perfusion scan should not be performed because transfer for endovascular clot retrieval would take approximately 6 hours, therefore falling outside the clot retrieval time window due to late presentation. The patient was then discussed with an accepting physician on call at the accepting hospital and was transferred urgently through to their emergency department via aeromedical transport. On arrival at the referral hospital emergency department, the patient underwent CT of the thoracolumbar spine. This investigation showed an L2 to L5 disc bulge with L5 to S1 moderate to severe foraminal narrowing with L5 nerve root impingement. The patient was then referred to neurology and admitted for further investigations. The patient received magnetic resonance imaging (MRI) of the brain, which returned results with no acute ischemic change, space-occupying lesion, or intracranial haemorrhage to indicate neurologic symptoms. The patient then received an MRI of their whole spine, which returned results demonstrating a central cord focus between C7 to T1 indicating cord signal abnormality indicative of an ATM diagnosis. Additionally, the MRI of the spine reported lumbar spondylitic changes with an unremarkable cauda equina and no further cord abnormalities. A lumbar puncture was performed under CT guidance, which had normal opening pressure, normal cerebrospinal fluid, with a mildly elevated protein level of 0.08 g/dL (0.83 g/L) (normal, 0.02-0.06 g/dL [0.19-0.65 g/L]). Further pathology revealed an erythrocyte sedimentation rate of 18 mm/hr (normal, 0-15 mm/hr), a ferritin level of 589 ng/mL (589 μg/L) (normal, 30-400 ng/mL [30-400 μg/L]), an iron level of 39.1 μg/dL (7 μmol/L) (normal, 44.7-167.6 μg/dL [8-30 μmol/L]), and transferrin saturation of 12% (normal, 15%-45%). Results of the lipid profile, vitamin B_{12}, folate, virology, thyroid function, immunology, and heavy metals testing were grossly normal. The patient returned a positive antinuclear antibody with titer 1:160 and a speckled pattern; however, following this result, subsequent testing returned a negative extractable nuclear antigen test result.

Discussion

This case study explored the complex presentation of an otherwise well male who presented with acute onset of left lower limb weakness. The differential diagnosis considered in this case included stroke, compressive myelopathy such as cauda equina, or lumbar foraminal stenosis. The patient had a normal cranial nerve examination and isolated lower limb weakness. These presentations of symptoms made stroke diagnosis doubtful but also the most acute pathology to exclude which lead to prompt discussion with telestroke team.[6] Urinary incontinence and saddle anesthesia are common symptoms of cauda equina; however, it is uncommon for this to present without low back pain and intact anal tone.[7] Additionally, spinal cord infarction and Guillain-Barré syndrome were considered. Guillain-Barré syndrome usually presents with bilateral sensorimotor deficits and bladder dysfunction; although rare, there are reported cases of asymmetrical onset of sensorimotor deficits.[8] Other differential diagnoses were inclusive of dural arteriovenous fistula, epidural hematoma, metabolic, toxic, and neoplastic.

ATM is classified as an acute inflammatory necrosis or demyelination involving the spinal cord.9 Its focal pathological changes involve inflammation or swelling of myelin, proliferation of lymphocytes, axonal degeneration, rapid increase of perivascular inflammatory cells, and demyelination.[9] Typically, patients who present with ATM will present with a sudden onset of back pain, muscle weakness, sensory alteration, and possible urinary or bowel symptoms with or without dysfunction.[5] Commonly, ATM is classified into subtypes inclusive of acute partial, acute complete, and longitudinally extensive transverse myelitis.[2] Acute partial transverse myelitis is dysfunction of the spinal cord that is asymmetrical and mild, usually showing lesions on 1 to 2 vertebral segments on MRI.[2] Complete ATM is more severe, causing symmetrical dysfunction of the spinal cord, which can cause complete or near-complete neurologic dysfunction.[2] Longitudinally extensive transverse myelitis can be complete or incomplete spinal dysfunction, which will have a corresponding lesion on MRI involving 3 or more vertebral segments.[2]

ATM is commonly caused by other specific inflammatory and noninflammatory conditions.[2] There is no single specific etiology identifiable with this disease process; however, there are conditions attributable (Tables 1 and 2). If there is no cause identified, it is classified as idiopathic.[2]

John did not have any of the previously discussed comorbidities that have been directly linked as a causative factor of ATM; however, the inflammatory process and consequences of type 2 diabetes as a comorbidity must be considered. Patients with diabetes have an increased risk of infection because it impairs immune response to invading pathogens.[11] Diabetes acts on the polyol

Table 1
Inflammatory Causes for Acute Transverse Myelitis

CNS demyelinating disorders	Multiple sclerosis, neuromyelitis optica spectrum disorder, myelin oligodendrocyte glycoprotein antibody-associated disease, and acute disseminated encephalomyelitis[2-4]
Autoimmune disorders	Rheumatoid arthritis, antiphospholipid syndrome, SLE, Sjogren disease, sarcoidosis, ankylosing spondylitis, Behcet disease, and systemic sclerosis[2-4]
Infections	Enterovirus, HIV, human T-lymphotropic virus 1, measles, rubella, HSV, EBV, VZV, West Nile virus, arbovirus, tickborne infections, syphilis, and SARS-CoV-2[2-4,10]
Other	Idiopathic, paraneoplastic syndromes, vaccinations, and astrocytopathy[2-4]

CNS = central nervous system; EBV = Epstein-Barr virus; HSV = herpes simplex virus; SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2; SLE = systemic lupus erythematosus; VZV = varicella-zoster virus.

pathway, causing a downregulation of glutathione, leading to endothelial cell damage and depletion of nitric oxide, negatively impacting nerve vasculature.[11] It forms advanced glycation end products, which can cause irreversible death of Schwann cells and increase inflammatory cytokine release, resulting in a proinflammatory state.[11] Diabetes causes oxidative stress, causing reactive free radical release and leading to microvasculature damage of the nervous system.[11] It must be contemplated that the increased risk of infection along with the microvasculature and inflammatory consequences of diabetes on the nervous system could increase the likelihood of developing ATM in addition to prolonging recovery duration.

There is no effective cure for ATM, and treatment focuses on symptom reduction caused by spinal cord inflammation.[2] Frequently, patients have some neurologic recovery without medical intervention.[4] One of the most important aspects of management in ATM is determining the disease-specific etiology, and this requires comprehensive history taking and vast investigations, which leads to successful management.[7] Importantly, an infectious cause of ATM or a spinal dural arteriovenous fistula must be excluded because immunotherapy treatment can exacerbate these conditions.[7] ATM is a complicated disease process mainly due to its expanding list of complex etiologies, which most recently has linked cases to the severe acute respiratory syndrome coronavirus 2 virus and its vaccine.[2-4]

Management of ATM

In patients with ATM, first-line pharmacotherapy includes a short course of high-dose intravenous glucocorticoid, usually methylprednisolone or dexamethasone.[2,4] Additionally, patients who are unresponsive to corticosteroids can be administered plasma exchange or intravenous immunoglobulin as second-line rescue therapies. There is evidence that suggests plasma exchange and high-dose intravenous methylprednisolone compared with methylprednisolone alone reduced the likelihood of a relapse in patients with ATM.[2] There has been recent research into extended-release dalfampridine, which increases postsynaptic action potentials in the spinal cord, and this evidence suggests that dalfampridine improves walking speed in patients with ATM.[2] Additionally, certain relapsing immune-mediated myelopathies require ongoing immunotherapy to prevent relapse.[2]

Additional pharmacologic and nonpharmacologic management of the neurologic sequelae must also be considered when caring for a patient with ATM. Primarily, the focus of treatment is to reduce spinal cord inflammation to improve the neurologic outcomes.[10] Patients with ATM have poorer mobility, therefore, venous thromboembolism prophylaxis must be addressed.[10] Often, constipation and urinary symptoms are common sequelae of neurologic deficits that further complicate treatment, leading to urinary tract infections.[10] Involvement of a comprehensive multidisciplinary team is crucial to promoting physical therapy to improve return of function, optimize recovery, and reduce neurologic symptoms.[10] Additionally, neuropathic pain is common, and often tricyclic antidepressants, gabapentinoids, and selective serotonin reuptake inhibitors, in addition to serotonin and noradrenaline reuptake inhibitors, are being used to treat neuropathic pain.[10]

John was treated as having an idiopathic transverse myelitis. Treatment with steroids was considered, but neurologic symptoms improved without intervention. Physiotherapy aided in rehabilitation, mobility, and restrengthening exercises. After approximately 2 weeks at the accepting hospital, the patient returned to the rural facility with regular inpatient physiotherapy involvement and was discharged a week later. The patient made a partial recovery as John still experiences intermittent episodes of mild weakness in his left leg. Approximately 25% of all patients with idiopathic transverse myelitis experience a relapse, with young age at onset, female sex, low vitamin D levels, and longitudinally extensive transverse myelitis increasing the risk of relapse.[12] Additionally, it must be considered that if this patient does develop diabetic neuropathy due to poorly controlled type 2 diabetes, it may increase the risk of misdiagnosis of a relapse of ATM and/or delay a diabetic neuropathy diagnosis.

Medical Comanagement

The rural center had the ability to perform CT of the spine to provisionally assess for possible compressive myelopathies. This patient was comanaged by a medical officer and a nurse practitioner (NP), who had joint responsibility for this patient's ongoing care. Comanagement allows physicians and NPs to share responsibilities, leading to optimization of complex care, increased completion of tasks, and, ultimately, better patient outcomes.[13] Common attributes that ensure successful comanagement of patients are a shared philosophy of care, effective communication, and mutual respect and trust.[8] The treating NP deemed it necessary for a CT of the whole spine for further diagnostic clarification. The NP escalated concerns and explained thought processes and possible differential diagnoses; however, the medical officer in charge disagreed, leading to investigations not being completed.

Table 2
Noninflammatory Causes for Acute Transverse Myelitis

Metabolic	Vitamin B_{12} deficiency, copper deficiency, and mitochondrial disorders[2,3,10]
Structural	Cervical spondylosis, disc herniation, spinal cord syrinx, Chiari malformation with cord compression, dorsal arachnoid web, and spinal cord herniation[2,3,10]
Other	Arteriovenous fistula, dural arteriovenous fistula, and spinal cord infarction[2,3,10]

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The recommendation for CT of the whole spine from the NP was clearly documented on a referral letter. There were no cost or insurance barriers to obstruct this type of imaging for this patient, and this incident was reported to the health service manager of the hospital. The patient did undergo CT of the whole spine on arrival at the referral facility, and this delay in imaging did not cause direct harm to the patient; however, if there were any findings needing neurosurgical intervention, the hospital did not have this specialty. Often, rural health care facilities encounter patients where the services that are needed cannot be facilitated by the treating hospital.

Complexities of Rural Health Care

Frequently, rural health care facilities provide a high level of medical care to a multitude of patients with input from specialist services from referral hospitals. Recurrently, rural clinicians are faced with the question, "Does this patient require services that their facility cannot provide?" Additionally, clinicians must keep in mind that patients in the rural health care setting have increased rates of coronary heart disease, lung cancer, diabetes, chronic kidney disease, and suicide, and a lower life expectancy compared with people in urban cities.[14] These rates are due to access to health care from geographical spread, low population density, limited availability of services, and additional time and costs to access the care needed.[14] Additionally, treating clinicians, particularly NPs, in rural health facilities need to have in-depth knowledge of their accepting larger hospitals. This knowledge includes the specialties they provide, bed capacity, clinical assessment equipment such as imaging machinery, and limitations of care. NPs also need to manage multiple considerations for patient care, including patient transfer procedures, resources, and costs of the service while providing gold-standard, evidence-based care. One of the primary reasons for transfer to the accepting hospital was that the initial hospital the patient presented to did not have MRI capability.

Conclusion

In patients presenting with acute-onset weakness, sensory changes, and urinary or bowel symptoms, the clinical condition of ATM should be considered as a possible differential diagnosis. ATM is a rare neurologic condition that can be inflammatory and noninflammatory in nature. Its onset is rapid and can cause debilitating neurologic changes to a patient's normal physiologic function. ATM is complicated further by its extensive list of complex etiologies that frequently accompany its onset. Clinicians must be made aware that patients in the rural and remote setting have a lower life expectancy and increased risk of disease processes compared with patients in the urban setting. Clinicians in the rural setting have the responsibility to advocate for their patients to be transferred and receive specialist care at local referral hospitals. Additionally, NPs can sometimes encounter patients who can be complex in nature, where diagnostically they cannot request or perform investigations that they may require. The NP in this case advocated for the patient to receive appropriate investigations; however, the medical officer ultimately declined. It is important that we effectively communicate with our physician counterparts to ensure the best possible outcomes for our patients.

CRediT authorship contribution statement

Mitchell Copeman: Conceptualization, Methodology, Investigation, Data curation, Writing - original draft, Writing - review & editing, Project administration. **Nadia Hulsbos:** Data curation, Writing - original draft, writing - review & editing. **Jessica Levick:** Conceptualization, Methodology, Writing - original draft, Writing - review & editing, Supervision, Project administration.

Declaration of Competing Interest

The author Mitchell Copeman declares a conflict of interest as one of the treating clinicians in this case study.

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References

1. Annunziata P, Masi G, Cioni C, et al. Clinical, laboratory features, and prognostic factors in adult acute transverse myelitis: an Italian multicenter study. *Neurol Sci.* 2019;40(7):1383-1391. https://doi.org/10.1007/s10072-019-03830-6
2. Fiani B, Covarrubias C, Jarrah R. Neuroimmunology and novel methods of treatment for acute transverse myelitis. *Cureus.* 2021;9(13):17043. https://doi.org/10.7759/cureus.17043
3. Murphy OC, Barreras P, Villabona-Rueda AF, Mealy MA, Pardo CA. Identification of specific causes of myelopathy in a large cohort of patients initially diagnosed with transverse myelitis. *J Neurol Sci.* 2022;442:120425. https://doi.org/10.1016/j.jns.2022.120425
4. Abbatemarco JR, Galli JR, Sweeney ML, et al. Modern look at transverse myelitis and inflammatory myelopathy. *Neurol Neuroimmunol Neuroinflamm.* 2021;8(6):1071. https://doi.org/10.1212/nxi.0000000000001071
5. Ali A, Bareeqa SB, Riaz A, Ahmed SI, Shaikh MH, Ghauri MI. Assessment of clinical outcomes in patients presenting with transverse myelitis: a tertiary care experience from a developing country. *Cureus.* 2019;11(3):e4342. https://doi.org/10.7759/cureus.4342
6. Price HL, Campbell SG. Isolated lower limb weakness following hemorrhagic stroke: a case report. *Cureus.* 2023;15(5):38798. https://doi.org/10.7759/cureus.38798
7. Zeb J, Zaib J, Khan A, Farid M, Ambreen S, Shah SH. Characteristics and clinical features of cauda equina syndrome: insights from a study on 256 patients. *SICOT J.* 2023;9:22. https://doi.org/10.1051/sicotj/2023019
8. Leonhard SE, Mandarakas MR, Gondim FAA. Diagnosis and management of Guillain–Barré syndrome in ten steps. *Nat Rev Neurol.* 2019;15:671-683. https://doi.org/10.1038/s41582-019-0250-9
9. Wen X, Xu D, Yuan S, Zhang J. Transverse myelitis in systemic lupus erythematosus: a case report and systematic literature review. *Autoimmun Rev.* 2022;21(6):103103. https://doi.org/10.1016/j.autrev.2022.103103
10. Tisavipat N, Flanagan EP. Current perspectives on the diagnosis and management of acute transverse myelitis. *Expert Rev Neurother.* 2023;23(4):389-411. https://doi.org/10.1080/14737175.2023.2195095
11. Luna R, Talanki Manjunatha R, Bollu B, et al. A comprehensive review of neuronal changes in diabetics. *Cureus.* 2021;13(10):19142. https://doi.org/10.7759/cureus.19142
12. Lee EK, Kim S, Sohn E. Clinical characteristics and predictive factors of recurrent idiopathic transverse myelitis. *Front Neurol.* 2024;15:1416251. https://doi.org/10.3389/fneur.2024.1416251
13. Norful AA, Ye S, Van der-Biezen M, Poghosyan L. Nurse practitioner–physician comanagement of patients in primary care. *Policy Polit Nurs Pract.* 2018;19(3-4):82-90. https://doi.org/10.1177/1527154418815024
14. Australian Institute of Health and Welfare. Rural and remote health. Published 2024. Accessed February 28, 2024. https://www.aihw.gov.au/reports/rural-remote-australians/rural-and-remote-health

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