SVOA Neurology

ISSN: 2753-9180

ScienceVolks Open Access

Case Report

Conversion Disorder Mimicking Transverse Myelitis in An Adolescent Lebanese Patient

Chadi Al ALAM, MD[1*], Rima Hazzouri, MD[2] and Razan Saleh[3]

[1]*Pediatrics and Pediatric Neurology, American Center for Psychiatry and Neurology, ACPN, Abu Dhabi, UAE*

[2]*Pediatrics and Neonatology, Al Mouwasat hospital, Riyadh, KSA*

[3]*Occupational therapist, American Center for Psychiatry and Neurology, ACPN, Abu Dhabi, UAE*

***Corresponding Author:** Chadi Al ALAM, MD, Pediatrics and Pediatric Neurology, American Center for Psychiatry and Neurology, ACPN, Abu Dhabi, UAE*

***Received:** May 10, 2022 **Published:** May 25, 2022*

Abstract

We present the case of a 13-year-old female Lebanese adolescent patient who initially presented to the emergency room for sudden onset of left sided weakness and dysarthria; All the investigations turned out to be negative; She was treated urgently as transverse myelitis because of the severity of this disease, with motor and sensory involvement and urinary retention; The previous history was hidden by the parents, and this was a reason to mislead the diagnosis. She developed during the hospitalisation a pseudo-seizure, and then we diagnosed her with conversion disorder and received the proper management with a multidisciplinary team.

***Keywords:** paroxysmal non-epileptic events, conversion disorder, transverse myelitis.*

Introduction

Paroxysmal non-epileptic events (PNEs) are characterized by episodes clinically similar to epileptic events but without the electrographic epileptic discharges in the brain. Psychogenic PNEs, are associated with psychological factors; Conversion disorder (CD) also called functional neurological symptom disorder is one of these events that we are going to discuss in this paper. There is a wide range of differential diagnosis, but transverse myelitis is one of the most serious and dangerous one and it is not listed in the literature as differential diagnosis in conversion disorder. Conversion reaction is underestimated, and we need to be more aware of this condition to provide the best care for the patients.

Case Presentation

In this study, we present the case of a 13-year-old Lebanese female patient who initially presented to the emergency room for sudden onset of left sided weakness, and dysarthria.

The patient is a full-term infant born to non-consanguineous parents. She was delivered at the hospital via spontaneous vaginal delivery with a birth weight of 3350 g and head circumference of 35 cm. There were no complications during pregnancy or delivery. Her developmental history negative: all the milestones were normal for age. Family history is positive for partial focal epilepsy in her 7-year-old brother.

She was not hospitalized before, and this is the first time she has such a weakness.

On her physical exam, she was somnolent, cooperative, normal facial tone, dysarthric, right sided motor power 5/5 and DTR +2; On the left side, she had severe weakness 1/5 motor power and DTR +2 only; she had improper sensitivity on the left leg, and paraesthesia with distended bladder requiring foley insertion.

Urgent CT brain was done in the ER to rule out any haemorrhagic stroke or arteriovenous malformation (AVM) and turned out to be negative.

Magnetic resonance imaging (MRI) brain and whole spine, and MRI brain stroke protocol, with magnetic resonance venography and angiography were done to R/O ischemic stroke, or arterial or venous malformation, or brain or cervical mass.

Lumbar puncture (LP) was done to check for any elevated proteins or WBC in the cerebro-spinal fluids to diagnose an autoimmune aetiologies or transverse myelitis, but the LP turned out to be negative.

Electromyography (EMG) and nerve conduction velocity (NCV) both were negative.

Despite the negative work up of all the imaging and the LP, we decided to treat her as transverse myelitis, because we have an acute motor weakness and sensory involvement and urinary retention, and because imaging and LP can still be negative in the early phase of transverse myelitis.

We started her on methylprednisolone sodium succinate 30mg/kg/day for 3 days.

On day 1 of steroids, she had a minimal motor power improvement of 2/5. But she was not able to speak completely.

On day 2, her motor power on the left side is decreased to 1/5, with complete mutism, and no urinary or bowel movement retention or incontinence.

On day 3, she had a sudden episode of shaking all over her bodies, not defined as clonic nor tonic movements.

EEG was performed with induction, and we were able to induce the same shacking episode with no electrographic correlation.

We stopped steroids, and we diagnosed the case as a conversion disorder, a classification of paroxysmal non-epileptic events or pseudo seizures.

We conducted a multidisciplinary team with the parents, and they informed us that they are having divorce and they are not handling well this issue with their children, and the mutism and the weakness were happened more than 3 times for the last 3 months and lasted for 5 days each.

The case was transferred under the care of psychiatry team and was followed by psychotherapist without medication.

Our patient showed significant improvement and she left the hospital in 4 days, and back to normal. She was followed regularly with psychiatry team and since the last 2 years, she was free of attacks.

Discussion

Transverse myelitis

Transverse myelitis (TM) is an acquired focal inflammatory disorder affecting the spinal cord, presenting as rapid or sudden onset of weakness, involving motor system, associated with sensory deficits, and bladder or bowel dysfunction. It can occur independently secondary to an infection, or as part of other autoimmune neuro-inflammatory disorders [1], such as multiple sclerosis, acute disseminated encephalomyelitis, acute flaccid myelitis and neuromyelitis Optica spectrum disorder known as Devic syndrome or disease. TM occurs at any level of the spinal cord but affects commonly the thoracic region. It causes bilateral deficiencies below the level affected, and it may only cause partial or focal involvement. [2] The incidence of transverse myelitis is estimated 1 to 8 new cases per 1 million people per year. [3] There is no difference in occurrence between Euro/American-born and Afro/Asian-born populations. [3]TM affects men and women equally. Women with multiple sclerosis tend to be more affected. [4] It can affect patients at any ages, but it presents more at the ages 10, 20, and above 40. [5] As per one case series, 64% of cases were idiopathic (primary TM) in nature, and the rest, 36% were associated with a disease (secondary TM). Other reports include idiopathic TM accounting for 15 to 30% of cases. [6] To make the diagnoses, a compressive cord lesion must be excluded first by MRI. This is followed by a confirmation of inflammation either by a gadolinium-enhanced MRI or LP. [7]

The first-line therapy for the treatment of transverse myelitis is intravenous glucocorticoids. High-dose intravenous glucocorticoids should be initiated as soon as possible. No delay in the treatment is accepted. Potential regimens would include methylprednisolone or dexamethasone for 3 to 5 days. If no response on steroids, then plasma exchange may be efficacious for acute central nervous system demyelinating disease. [8.9]

Most patients with idiopathic TM should at least have a partial recovery. It should begin within 1 to 3 months and should continue to improve with rehabilitation therapy. [10]

Conversion Disorder

PNEs are characterized by episodes clinically like those of epileptic events, without the electrographic epileptic discharges in the brain. They involve motor, sensory and emotional changes, with or without impairment of consciousness. PNEs represents a rate of 15% (12%–16%) of patients referred to epilepsy clinic. [11.12] A review of 887 paediatric patients treated in an epilepsy monitoring unit, assessed by clinical history and video-electroencephalography, found a 15.9% rate of non-epileptic events. [11] Another study showed that 30-35% of children diagnosed with epilepsy are misdiagnosed. [13,14] Even for specialists, the diagnosis is sometimes difficult and requires a lot of attention and expertise. Almost 25% of paroxysmal events are misdiagnosed with specialist in neurology and neuropsychologist and psychiatrists. [15] Inadequate clinical history, incorrect interpretation of test results, consideration of traditional but unreliable warning signs, are among the factors that lead to a misdiagnosis. [16]

Non-epileptic events are classified as physiological or psychogenic. Physiological PNEs are associated with organic causes and are classified into hypoxic–ischemic phenomena, migraine-associated disorders, sleep disorders, and movement disorders.

However, Psychogenic PNEs, are associated with psychological factors. This includes conversion disorder also called functional neurological symptom disorder, with panic disorder as a differential diagnosis. [17,18, 19]. In CD, there are one or more symptoms of motor function or sensory changes that are not well explained by other mental disorders or medical conditions. [19] This is a great mimicker of seizures or strokes, where we have focal motor and sensory deficit. These events are prevalent in 1% - 9% in children and adolescence with suspected epilepsy. [20] Psychogenic events have been reported in children from 5 years of age; the frequency of this type of event increases with age, with psychogenic events being the most common non-epileptic events in adolescence. [21]

Approximately 10% of patients with psychogenic PNE also have epilepsy. [12,22]. Factitious disorder and simulation disorder are on top of the differential diagnosis. [19] When the patient falsifies sign and symptoms or inducing injury or disease this is called factitious disorder. However, Simulation involves purposely and consciously the production of signs and symptoms for personal gain, like simulating a seizure to stay home.

The approach for CD consists of a multidisciplinary approach including psychiatrist, psychologist, and behavioural therapist with psychoeducation, and medications sometimes.

Conclusion

Paroxysmal non-epileptic events are sometimes misdiagnosed and can be a great mimicker of neurological diseases or disorders that requires very fast treatment like transverse myelitis. Psychological conditions are still considered as taboo almost everywhere. Conversion disorder is one of the PNEs; It is still underestimated, and we should be more aware of this condition to prevent unnecessary tests and to provide faster approach and management. The earlier we diagnose, the better the outcome.

Conflict of Interest

The authors declare no conflict of interest.

References

1. West TW, Hess C, Cree BA. Acute transverse myelitis: demyelinating, inflammatory, and infectious myelopathies. Semin Neurol. 2012 Apr;32(2):97-113.

2. Krishnan C, Kaplin AI, Pardo CA, Kerr DA, Keswani SC. Demyelinating disorders: update on transverse myelitis. Curr Neurol Neurosci Rep. 2006 May;6(3):236-43.

3. Berman M, Feldman S, Alter M, Zilber N, Kahana E. Acute transverse myelitis: incidence and etiologic considerations. Neurology. 1981 Aug;31(8):966-71.

4. Scott TF, Frohman EM, De Seze J, Gronseth GS, Weinshenker BG., Therapeutics and Technology Assessment Subcommittee of American Academy of Neurology. Evidence-based guideline: clinical evaluation and treatment of transverse myelitis: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology. 2011 Dec 13;77(24):2128-34.

5. ALTROCCHI PH. ACUTE TRANSVERSE MYELOPATHY. Arch Neurol. 1963 Aug; 9:111- 9.

6. Beh SC, Greenberg BM, Frohman T, Frohman EM. Transverse myelitis. Neurol Clin. 2013 Feb;31(1):79-138.

7. Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. Neurology. 2002 Aug 27;59(4):499-505.

8. Weinshenker BG, O'Brien PC, Petterson TM, Noseworthy JH, Lucchinetti CF, Dodick DW, Pineda AA, Stevens LN, Rodriguez M. A randomized trial of plasma exchange in acute central nervous system inflammatory demyelinating disease. Ann Neurol. 1999 Dec;46(6):878-86.

9. Cortese I, Chaudhry V, So YT, Cantor F, Cornblath DR, Rae-Grant A. Evidence-based guideline update: Plasmapheresis in neurologic disorders: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology. 2011 Jan 18;76(3):294-300.

10. Defresne P, Hollenberg H, Husson B, Tabarki B, Landrieu P, Huault G, Tardieu M, Sébire G. Acute transverse myelitis in children: clinical course and prognostic factors. J Child Neurol. 2003 Jun;18(6):401-6.

11. Park E, Lee J, Lee BL, Lee M, Lee J. Paroxysmal nonepileptic events in pediatric patients. Epilepsy Behav. 2015; 48:83-7.

12. Yılmaz U, Serdaroğlu, Gürkaş E, Hirfanoğlu T, Cansu A. Childhood paroxysmal nonepileptic events. Epilepsy Behav. 2013; 27(1):124-9.

13. Alving J, Hansen LK, Kibaek M, Buchholt J. The misdiagnosis of epilepsy in children admitted to a tertiary epilepsy centre with paroxysmal events. Arch Dis Child. 2006; 91(3):219-21.

14. Hindley D, Ali A, Robson C. Diagnoses made in a secondary care "fits, faints, and funny turns" clinic. Arch Dis Child. 2006; 91(3):214-8.

15. Ristic AJ, Mijovic K, Bukumiric Z, et al. Differential diagnosis of a paroxysmal neurological event: Do neurologists know how to clinically recognize it? Epilepsy Behav. 2017; 67:77-83.

16. Oto MX. The misdiagnosis of epilepsy: Appraising risks and managing uncertainty. Seizure. 2017; 44:143-6.

17. Luat AF, Kamat D, Sivaswamy L. Paroxysmal nonepileptic events in infancy, childhood and adolescence. Pediatr Ann. 2015; 44(2): e18-23.

18. Doss JL, Plioplys S. Pediatric psychogenic nonepileptic seizures. Child Adolesc Psychiatr Clin N Am. 2018; 27(1):53-61.

19. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5 ed.Washington, DC: American Psychiatric Association; 2014.

20. Reilly C, Menlove L, Fenton V, Das KB. Psychogenic nonepileptic seizures in children: A review. Epilepsia. 2013; 54 (10):1715-24.

21. Kotagal P, Costa M, Wyllie E, Wolgamuth B. Paroxysmal nonepileptic events in children and adolescents. Pediatrics. 2002; 110(4): e46.1-e46.5.

22. Duncan R, Oto M. Predictors of antecedent factors in psychogenic non- 102 Residência Pediátrica 2018;8(supl 1):93-102 epileptic attacks: multivariate analysis. Neurology. 2008;71(13):1000-5.

Citation: ALAM CA, Hazzouri R, Saleh R. "Conversion Disorder Mimicking Transverse Myelitis in An Adolescent Lebanese Patient". *SVOA Neurology* 2022, 3:3, 127-130.