Unusual presentation of more common disease/injury

Acute onset of tetraparesis, areflexia and respiratory distress in an otherwise healthy male

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Summary

The patient is a previously healthy Eastern-African man in his late 20s, with unknown vaccination status who presented to a US Army Combat Hospital in Iraq with acute upper and lower extremity weakness progressing to respiratory distress requiring intubation over the course of 12 h. His only antecedent symptoms were weakness and nausea. Laboratories including complete blood count, C reactive protein, cerebrospinal fluid analysis, venous blood gas, rapid HIV and chemistry were normal. Non-contrast head CT was normal. The patient was extubated after 24 h but continued to exhibit 2/5 strength in bilateral upper and lower extremities with absence of deep-tendon reflexes. At 48 h of illness it was discovered that the prior to his presentation the patient had a significant life stressor which had since been resolved. Upon discovery that this stressor had been resolved, the patient's symptoms improved over the subsequent 4–6 h and was discharged after 24 h without further complication.

BACKGROUND

Progressive systemic weakness leading to respiratory failure carries a broad differential diagnosis including several infectious, autoimmune and toxic aetiologies. Conversion disorder can cause similar symptoms although hemiplegia is more commonly reported and respiratory distress requiring intubation is rare. Additionally absence of deep-tendon reflexes is not typically associated with conversion disorder. This case highlights the importance of gathering psychosocial information from several sources when evaluating a patient with acute symptoms and no clear medical cause after radiographic and laboratory evaluation to help assess for possible psychiatric trigger of the patient's symptoms.

CASE PRESENTATION

The patient was a previously healthy East-African male in his late 20s, working as a security guard on a Forward Operating Base in Iraq. He presented to a clinic at the Forward Operating Base with feelings of weakness and nausea. Initial concerns were for heat-related illness and the patient was given intravenous fluids and kept under observation. Over the subsequent 12 h, the patient developed progressively worsening weakness of upper and lower extremities, decreased level of consciousness and tachypnoea with apnoeic spells lasting up to 30 s. At that time the patient was endotracheally intubated with 30 mg etomidate and 10 mg vecuronium and evacuated to a level III US Combat Support Hospital (CSH) via helicopter.

Upon arrival at the CSH, the patient was haemodynamically stable in normal sinus rhythm with heart rate of 70 beats per minute, afebrile, receiving mechanical ventilation with 40% FiO₂ on minimal ventilator settings with pulse oximetry of 100%. On initial examination the patient was noted to have equally reactive pupils, clear and equal breath sounds, normal heart rate with no murmurs, strong periph-

eral pulses with normal, warm extremities, a soft abdomen with bowel sounds present and a normal skin exam with no rashes, insect bites or tics noted. His initial Glasgow Coma Scale was 5T. His initial exam was remarkable for myoclonic jerking of his bilateral upper and lower extremities without purposeful movement as well as fasciculation of his quadriceps while femoral venous access was obtained. Two separate examiners were unable to elicit upper or lower extremity deep-tendon reflexes and a Babinski was performed which was downgoing.

The patient had no known medical or surgical history, no known allergies to medications and was taking no prescription or over the counter medications. His living quarters were searched and no supplements or illegal substances were discovered. The patient worked as a security guard on a guard tower and there were no environmental or other hazardous exposures reported recently by his co-workers or supervisors. No co-workers or associates were noted to have similar symptoms. The patient had been working in Iraq for more than 1 year.

Initial radiographic and laboratory investigations were obtained as noted below and were all within reference ranges. At this time the patient was started on midazolam and fentanyl infusions for pain control and sedation and given a second dose of 10 mg of vecuronium for respiratory dyssynchrony with mechanical ventilation.

The patient was admitted to the intensive care unit (ICU) for continued mechanical ventilation and observation. Sedation was decreased overnight and the patient was successfully extubated hospital day 2 to room air with no further episodes of tachypnoea or apnoea. The patient had a good cough and gag reflex, was able to talk and move his head but still displayed profound weakness of both the upper and lower extremities of which he did not seem overly concerned. His neurologic examination at this point demonstrated normal cranial nerves (II–XII), intact

sensation, vibration and proprioception in all extremities and two out of five motor strength in upper and lower extremities – bilateral. He was noted to be areflexic in the upper and lower extremities by two separate physicians.

During the course of his admission to the ICU, it was noted by the nursing staff that the patient was able to move his extremities while sleeping. At this time further contact was made with referring providers who related that they had discovered the patient was dealing with significant, ongoing stressful events which were scheduled to be concluded the week of his presentation. On ICU day 3 confirmation was obtained that the proximate stress inducing event had been resolved in a favourable manner for our patient. Physical exam was repeated at this time and continued to exhibit intact cranial nerves, 2/5 upper and lower extremity strength bilaterally and absent upper and lower extremity deep-tendon reflexes. After examination the patient was made aware that his stressor had been resolved. Several hours after this notification the patient had recovered 5/5 strength in his upper and lower extremities as well as 2+ reflexes in his bilateral upper and lower extremities.

INVESTIGATIONS

Non-contrast CT of the brain, no acute abnormalities.

Chest x-ray, no acute cardiopulmonary disease with an endotracheal tube in an appropriate position.

Lumbar puncture with cerebrospinal fluid sampling: glucose 62 mg/dl, protein 28 mg/dl, 1 leucocyte per hpf, 0 red blood cells per hpf, gram stain negative, cultures negative, opening pressure not recorded.

Urine drug screen, negative.

Complete blood count: white cell count $10.8 \times 10^3/\mu\text{l}$ with a normal differential, haemoglobin 15.1 g/dl, platelets $210 \times 10^3/\mu\text{l}$ venous blood gas, pH 7.48, pCO_2 33.3 mm Hg, PO_2 45 mm Hg.

Liver function tests: amylase 77 U/l, total protein 8.6 g/dl, albumin 4.6 g/dl, aspartate aminotransferase 30 U/l, alanine aminotransferase 18 U/l, lactate dehydrogenase 725 U/l, alkaline phosphatase 106 U/l, total bilirubin 1.2 mg/dl.

Lactic acid 2.7 mmol/l.

Chemistry: sodium 139 mmol/l, potassium 3.7 mmol/l, chloride 113 mmol/l, bicarbonate 22 mmol/l, BUN 7 mg/dl, creatinine 1 mg/dl, glucose 114 mg/dl, calcium 9.5 mg/dl, magnesium 1.9 mg/dl and phosphorus 2 mg/dl.

C reactive protein 1.

Thyroid-stimulating hormone 1 $\mu\text{UI/ml}$.

Creatine kinase 129 U/l.

Rapid plasma reagin, non-reactive.

Rapid HIV, negative.

DIFFERENTIAL DIAGNOSIS

Guillain-Barre syndrome, tic paralysis, bulbar polio, mass, toxin ingestion, occult spinal trauma, conversion paralysis.

OUTCOME AND FOLLOW-UP

The patient was diagnosed with conversion disorder and discharged after 24 h of observation with no further complications prior to returning to Uganda.

DISCUSSION

Conversion paralysis is defined as voluntary motor and/or sensory loss that suggest a neurological or other general medical condition associated with psychological stressor with symptoms that are not intentionally produced or feigned and cannot be fully explained by a general medical condition. Rates of conversion disorder are reported between 0.011% and 0.5% with 2–10-fold greater incidence in women.¹

Conversion paralysis can present with mono, para, hemi, tri or even tetraparesis as in our patient. The aetiology of this disorder is still unknown although it has been postulated to be caused by dysregulation of the striato-thalamocortical circuits through interaction with the limbic system to explain its association with strong emotional stimulus.² In a 30-patient case controlled study by Binzer *et al*, statistically significant risk factors for conversion motor disorders include; low education, presence of a personality disorder and high Hamilton Depression Score. Interestingly, in their study female sex was not a statistically significant predictor.³

In 1893, Joseph F F Babinski famously described the upgoing toe as a discriminator between organic paralysis and that caused by psychogenic factors.⁴ Since that time conversion disorder has continued to be a diagnosis of exclusion often leading to extensive, and sometimes invasive diagnostic testing.

The clinical presentation of conversion paralysis can vary greatly, with exam findings not consistent with known pathology. Case series and reports show a similar variability in the constellation of exam findings. Some cases demonstrate preservation of deep-tendon reflexes in lower extremity paresis with or without loss of bowel and bladder function.^{5–7} Other reports have demonstrate loss of deep-tendon reflexes with lower extremity paresis.⁸ A case series by Yugué *et al* reported varying neurologic exams but preservation of the bulbocavernosus reflex in all 14 patients.⁹ Conversion tetraparesis is rare, demonstrated in only 3 of 34 patients in a case series, all of whom had normal deep-tendon reflexes and normal respiration with full control of sphincters.³ Another case series reported only 2 of 79 patients with tetraparesis.¹⁰ There are no other reports of tetraparesis found in the literature. We are aware of only one other report demonstrating respiratory symptoms with conversion motor disorders, described as gasping respirations with 10–15 s apnoeic episodes that did not lead to any significant respiratory impairment.¹¹

There are several unique aspects to our clinical presentation. First, he presented with weakness and sensory deficits, which is common, but additionally he demonstrated respiratory distress to such a degree that he required intubation for stabilisation prior to air evacuation. While one other case has been reported with respiratory symptoms with conversion paralysis,¹¹ we are aware of no other reports of patients presenting with respiratory distress requiring intubation. As stated above tetraparesis is an uncommon presentation of conversion disorder. Additionally, our patient demonstrated loss of both upper and lower extremity deep-tendon reflexes but preservation of sphincter control which is consistent with the variable presentation of this disorder.

Bulbocavernosus reflex was not evaluated in our patient so its presence or absence is unknown. While we cannot rule out malingering in our patient, we feel this is highly unlikely given the invasive nature of interventions and respiratory distress with which he presented. He did also demonstrate rapid recovery following resolution of his proximate stressor which is more consistent with conversion disorder than malingering.

Our case demonstrates the importance of consideration of conversion disorder in a patient presenting with tetraparesis with no obvious medical explanation even with respiratory compromise necessitating intervention. Additionally, it highlights the importance of obtaining a psychosocial history as soon as possible after stabilisation to perhaps mitigate further expensive and potentially invasive testing in patients who may have a psychogenic aetiology of paralysis. It also supports the multidisciplinary approach recommended for evaluation of such patients as advocated by Smith *et al*⁶ and the importance of tools, such as the Spinal Injuries Center test⁹ to help clinically evaluate patients suspected of having conversion paralysis.

Learning points

- ▶ Conversion disorder can present with respiratory distress requiring intubation.
- ▶ Full psychosocial history from various sources should be sought in any patient presenting with neurologic symptoms with unclear cause.
- ▶ Respiratory distress and lack of deep-tendon reflexes do not rule out conversion paralysis.

Competing interests None.

Patient consent Not obtained.

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Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Hamele M, Mount CA. Acute onset of tetraparesis, areflexia and respiratory distress in an otherwise healthy male. *BMJ Case Reports* 2010; 10.1136/bcr.10.2010.3398, date of publication

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