CASE REPORT

Infarction of the cervical spinal cord in a 20-year-old healthy man resulting in complete quadriplegia

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SUMMARY

How often are we faced with a tragic diagnosis in a young patient whose life is completely changed? Often in medicine the tragedy is short-lived: the patients never stabilise, they succumb early to their injuries or complications. We present the case of a young man in whom the exact cause of a spinal cord infarct has never been confirmed. As it transpires, regardless of the sequence of events and the time elapsed between injury and the onset of paralysis, his symptoms came with no warning, were life-threatening and terrifying. He could have had no time to understand what had happened and is now faced with what must seem an eternity to come to terms with a life of quadriplegia. He gives an account of his life for the last 7 years as he has remained at home, while life for his siblings has moved on and he watches from his bed. The triumph is his adjustment to his life now, the vigilance and care of his family and the dedication of the medical staff of a local clinic in a small village in the mountains in the north of Israel.

BACKGROUND

Spinal cord injury resulting in complete paralysis is unfortunately not rare. Notable cases include well-known public figures who have suffered acute trauma. For every individual to whom this has happened, however, life is completely changed forever and long-term care is an enormous challenge physically, emotionally and in terms of the financial burden on healthcare and support services.

CASE PRESENTATION

In 2008 a 20-year-old previously healthy man, an engineering student, presented to his general practitioner with headache and neck pain that began hours before his visit. During the consultation, while lying on the examination couch he complained of weakness and shortness of breath. He then suddenly stopped talking. The doctor immediately checked for a pulse, checked that the patient was still breathing and then attached ECG monitoring. He then checked the blood pressure and oxygen saturation (the clinic, a community clinic in a village in the north of Israel, has all the facilities for primary emergency care including monitoring, intubation and ventilation).

The pulse was 80 beats/min, blood pressure was 120/80 mm Hg and oxygen saturation 86% but, although conscious, he was unable to move his arms and legs and could not breathe. An immediate working diagnosis of acute onset flaccid paralysis requiring ventilation was made—the cause possibly related to the pain in his neck (spinal injury as he

was conscious but unable to breathe for himself). With bag and mask ventilation the oxygen saturation improved to 96%. A Philadelphia cervical collar was applied and urgent transfer to the local general hospital via ambulance was arranged. There was no immediate urinary or faecal incontinence but, on further examination, it was clear that joint and position sense were also lost.

Supplementary history from the family revealed that the patient had been injured in a car crash 8 months earlier and, although checked in the local emergency room at the time, he had been found to have no significant injuries. He had returned to work the next day with no subsequent complaint and had been well until the present day. The differential diagnosis was therefore of a late presentation of spinal cord injury above the level of C3, a cord lesion possibly unrelated to trauma, or drug intoxication.

INVESTIGATIONS

As soon as the ambulance arrived he was intubated. On arrival at the emergency room an urgent CT scan of the brain and cervical spine was performed. No abnormality was seen on the CT images and a lumbar puncture was therefore performed. The results were as follows: cerebrospinal fluid (CSF) white blood cells 19 cells/mm³, CSF red blood cells 0, CSF protein total 33 mg/dl (normal 15–45 mg/dl), glucose 72 mg/dl (normal 70–110 mg/dl). Drug levels were normal: carbamazepine <0.5 µg/ml, phenobarbitone <1.1 µg/ml, phenytoin <0.5, valproic acid <0.7 µg/ml, paracetamol <1 µg/ml and troponin 0.00. Full blood count and blood chemistry were also within normal limits.

The patient was then transferred to a level 1 trauma centre where he underwent MRI of the brain and spinal cord as the suspicion of a spinal cord lesion remained. MRI scan of the brain was normal but cervical spine imaging showed infarction of the spinal cord at the level of C2, in keeping with the patient's clinical signs (figure 1). CT angiography of the cerebral circulation was normal with no vascular malformation or aneurysm visible. Echocardiography (to exclude cardiac embolism) and the coagulation profile were also normal.

DIFFERENTIAL DIAGNOSIS

The patient remained in the intensive care unit and underwent tracheostomy 5 days later. It became apparent that neurological recovery would not be immediate and there was no improvement in his clinical signs. Complete motor paralysis from the level of C2 was confirmed.

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Figure 1 A 20-year-old man presented with sudden complete quadriplegia. MRI findings indicated infarction of the spinal cord in the area supplied by the anterior spinal artery. (A) T1-weighted (260/8 (TR/TE)) sagittal MR image on admission showing slight low signal intensity at the C2–C4 level. (B) T2-weighted (3940/110 (TR/TE)) sagittal MR image showing high signal intensity lesion at the anterior part of the spinal cord at the C2–C4 level. (C) Corresponding T1-weighted MR image after gadolinium administration showing no enhancement of the lesion. (D) Corresponding T2-weighted (3380/104 (TR/TE)) MR image showing rounded high signal intensity area at the anterior part of the spinal cord (owl's eye appearance).

TREATMENT

A percutaneous endoscopic gastrostomy (PEG) tube was inserted for feeding. The patient was then transferred to a rehabilitation unit and ultimately discharged home on a mechanical ventilator.

OUTCOME AND FOLLOW-UP

At home he has his own room which has been specially built. He has a hospital bed with automatic controls and an air mattress. His family has been trained by the rehabilitation team to offer bedside care (PEG feeding, suction of the tracheostomy tube and turning). Their car has been adapted so that he can travel with them while on the ventilator. He usually goes somewhere by car everyday. All his medical care is provided by the medical staff of the community clinic. He receives home care from the local clinic which comprises tracheostomy care, ventilator checks and twice-weekly visits from the local doctor and nurse. He has no control over bowel or bladder function but generally has one bowel motion a day so, with a urinary catheter and appropriate incontinence pads, is almost always dry. The tracheostomy tube is changed once a month and the suprapubic catheter and gastrostomy tube every 3 months. He has twice-weekly physiotherapy, regular visits from his friends who congregate in his room around his bed and is able to watch a television perfectly positioned on the wall opposite.

In 2009 he was admitted to the local community hospital for investigation and intravenous antibiotics for sepsis found to be secondary to a urinary tract infection. Eventually, after recurrent

infections, a suprapubic catheter was inserted. In 2011 he was admitted with pneumonia which improved after a course of intravenous antibiotics and intensive physiotherapy. He has had no pressure sores and it is the dedication of his family and the support of the local health care services with the ability to provide ventilator care that has kept him alive. There has been no improvement in neurology since the onset of paralysis and a cause of the C2 infarct, other than the putative diagnosis of antecedent trauma, has never been found.

DISCUSSION

The diagnosis of spinal cord infarction secondary to antecedent trauma months earlier has never been established in this patient. While spinal cord infarction secondary to atheromatosis is well researched, and acute spinal injury resulting in direct trauma to the spinal cord, intervertebral discs or a fractured or dislocated vertebra and the compression or destruction of axons causing incomplete or complete paralysis is well understood, there are few instances in the literature of delayed neurological presentation months after spinal injury.

Although the diagnosis of paralysis was clear from the outset, proving the exact cause remains difficult. Possible mechanisms of injury include chronic ischaemia after injury, exacerbated by the arterial anatomy of the spinal cord that can render areas supplied by the anterior spinal artery hypoxic. Medical causes such as vasculitis, collagen or coagulation disorders and drugs (such as cocaine) might contribute to this, but the patient had

none of these risks factors as a predisposition. Spinal cord injury without vertebral fracture, dislocation or radiological abnormality has been described in children secondary to the inherent elasticity of the paediatric vertebral column subjected to deforming forces.¹ Delayed paralysis caused by ischaemia in mice with spinal cord injury after aortic cross-clamping² has been used to study paralysis in humans subjected to spinal cord ischaemia as a result of surgery (a disastrous complication often resulting in litigation).³ We know that damage to the spinal cord continues long after the initial trauma, but research indicates that this is within hours of the injury rather than days or months and may involve free radical damage and excitotoxicity (an excess of neurotransmitters). This has been the basis of research into the prevention of paralysis after injury.⁴

The only other cases of delayed onset paralysis after trauma found were those in whom symptoms developed a maximum of 24 h after the initial injury.⁵ The absence of pain or the evolution of neurological symptoms in the 8 months between injury and onset of paralysis suggests that the posterior spinal arteries or a transverse infarction of the spinal cord was unlikely. This is confirmed by radiology which suggests infarction of the anterior spinal artery. Lynch *et al*⁶ and Alblas *et al*⁷ have described the expected findings after infarction of the spinal cord. Our findings are similar, and although an acute infarct, we have assumed that the only rational cause for this was cervical spine trauma 8 months earlier.

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Learning points

- ▶ Neurological examination after spinal injury may be normal and patients may present late with symptoms.
- ▶ MRI will pick up lesions that CT does not. If the index of suspicion remains high or CT findings do not account for neurological signs, MRI is recommended.
- ▶ All doctors should know how to protect the airway and provide bag and mask ventilation until a definite airway is established.
- ▶ Patients with cord lesions above the C3 spinal level need ventilation.
- ▶ Neurological recovery may never occur and, in this case, provision for long-term care should be available. With the cooperation of the community medical team and the dedication of the family and the local hospital, patients may enjoy an appreciable quality of life and survive on artificial ventilation for years.

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