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CASE REPORT

Hardware failure and spinal pseudoarthrosis causing autonomic dysreflexia: a report of two cases

RS Li Pi Shan[1], AG Linassi[1], AK Dzus[2] and A Woo[2]

[1]*Department of Physical Medicine & Rehabilitation, University of Saskatchewan, Saskatoon, Saskatchewan, Canada and* [2]*Department of Surgery, Division of Orthopaedic Surgery, University of Saskatchewan, Saskatoon, Saskatchewan, Canada*

Study design: Case report.
Objectives: To describe two cases of hardware failure with pseudoarthrosis causing autonomic dysreflexia. The first patient was a 24-year-old woman with T3 ASIA (American Spinal Injury Association)-A paraplegia who developed complete failure and breakage of the Luque rods at the T11-12 region. The second woman was a 36-year-old T5 ASIA-A complete paraplegic who fractured her Harrington rods at T12 and L1 bilaterally.
Setting: Saskatoon City Hospital, Saskatchewan, Canada.
Methods and results: Both patients underwent operation for surgical fixation. In both cases, stabilization and fusion of the spinal deformity abolished the autonomic dysreflexia.
Conclusion: Owing to the failure of spine-stabilizing hardware, sitting upright may cause an afferent stimulus that triggers the onset and worsening of symptoms associated with autonomic dysreflexia. Therefore, in contrast to current acute treatment regimes, lying down may be preferred to sitting upright (to decrease blood pressure) as a means to relieve the afferent stimulus. Surgical correction and hardware replacement alleviated the symptoms in these two patients.
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Keywords: autonomic dysreflexia; spinal cord injury; pseudoarthrosis

Introduction

Autonomic dysreflexia (AD) is a syndrome of symptoms that presents with an increase in blood pressure in patients with spinal cord lesions typically above T6. It is the result of a variety of somatic and visceral stimuli that triggers widespread reflex sympathetic activity resulting in vasoconstriction below the neurological lesion. Elevated blood pressure is the cardinal finding in AD and is defined as being 20–40 mm Hg systolic pressure above normal resting blood pressure.[1]

The presenting symptoms include generalized pounding headache, anxiety, nausea and visual disturbances. Signs include sweating, flushed skin, piloerection, nasal congestion above the level of the lesion and cold pale skin below. AD may also present asymptomatically.

Although urological and visceral causes are the most common cause of AD,[1] musculoskeletal causes of AD have been reported and include sacral fractures,[2] hip dislocation,[3] Charcot spinal arthropathy[4] and heterotopic ossification.[5]

Correspondence: Dr RS Li Pi Shan, Department of Physical Medicine & Rehabilitation, University of Saskatchewan, 701 Queen Street, Saskatoon, Saskatchewan, Canada S7K 0M7.
E-mail: Rodney.lipishan@saskatoonhealthregion.ca
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Case 1

A 24-year-old woman with a complete T3 ASIA (American Spinal Injury Association)-A paraplegia, caused by a motor vehicle accident at age 4, developed scoliosis and was treated by Luque rod hardware and fusion in 1995. She was admitted to hospital in November 2004 with AD that progressively worsened over 6 weeks. Her symptoms included muscle spasms, sweats, blurred vision, labile temperature, nausea, severe pounding headache, photophobia and phonophobia. Blood pressure increased from 90/40 to 125/75. Heart rate was 105–125 and regular. The patient described a grinding sensation when turning and her symptoms were aggravated when sitting upright. A spine X-ray revealed complete fractures of the Luque rods at the T11/12 spinal level (Figure 1a). Increased activity at T11 was seen on bone scan, but both white blood cell scan and blood cultures were normal. Urinalysis, electrocardiography, electroencephalography and ultrasound of the kidneys were also normal. A pheochromocytoma workup was normal. By the end of December, her blood pressure was intermittently elevated to 163/105. She was prescribed a thoracolumbar brace, which somewhat improved her symptoms when upright and was scheduled for a thoracolumbar pseudoarthrosis repair. As she developed a grade 4 sacrococcygeal pressure ulcer, spinal

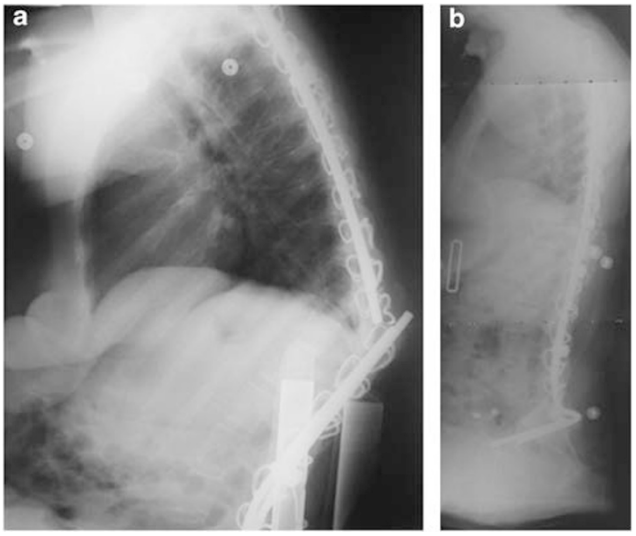

Figure 1 Radiographs of case 1 before (**a**) and after (**b**) surgical repair.

surgical repair was delayed until after she underwent pressure ulcer debridement and flap. In June 2005, she underwent pseudoarthrosis repair by anterior and posterior fusion with hardware revision and bone graft. Post-operative recovery was complicated by a wound infection that required irrigation, debridement and antibiotics. The symptoms of AD resolved after discharge with the blood pressure returning to baseline values immediately after surgery. Post-operatively, her blood pressures ranged between 90/59 and 112/75 with no other accompanying symptoms of AD. Follow-up X-ray showed acceptable spine alignment (Figure 1b).

Case 2

A 36-year-old female sustained a T5 ASIA-A complete paraplegia as a result of a motor vehicle accident 30 years ago and was treated by Harrington rod fixation.

She began having symptoms of AD, which rapidly worsened in January 2005. It was precipitated by sitting upright and relieved by lying down. It presented as cold sweats, fever and tachycardia. Blood pressure was elevated at 137/75. A spine X-ray showed fractured Harrington rods at T12 and L1 bilaterally with significant rotoscoliosis (Figure 2a). Surgery was deferred, as she was 4 weeks pregnant.

After childbirth, an empirical course of antibiotics did not relieve her symptoms and blood work did not reveal any infection. Bone scan showed low-grade hyperemia; however a white blood cell scan was negative. Blood cultures and V/Q scan were also negative.

The Harrington rods were removed, and internal fixation was obtained with pedicle screws from T6 to L5 with bone bank graft and local bone graft. Her pain improved and the fever subsided for 2 months. However, the AD continued,

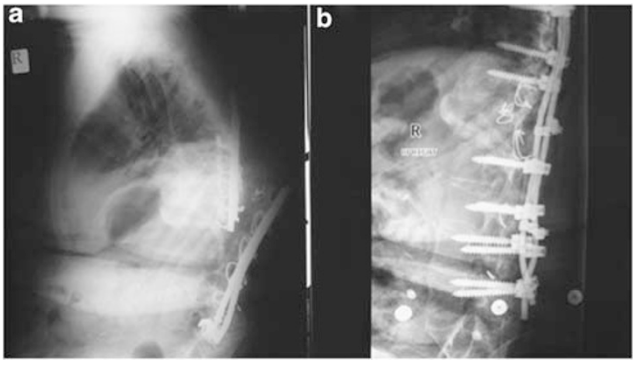

Figure 2 Radiographs of case 2 before (**a**) and after (**b**) surgical repair.

and her symptoms returned after this. Blood cultures, blood work and urinalysis were negative. X-rays carried out on 14 September 2006, showed failure of the proximal screws.

The patient underwent a second operation on 2 October 2006, involving T3 to L5 fusion with femoral head allograft and bone morphogenic protein. She was also prescribed a 6-week course of antibiotics and bed rest. The AD, fever, sweats and pain subsided. A year later, X-rays showed a healing stable spine (Figure 2b).

Results and discussion

We report hardware failure with pseudoarthrosis as an earlier unknown cause of AD in post-traumatic spinal cord injured patients. In most cases of AD, accepted acute treatment regimes include sitting the patient upright to decrease the elevated blood pressure. In these two cases, the patients were placed in the supine position to alleviate the afferent stimulus. Definitive resolution of symptoms was achieved by operative restabilization and not by bracing alone.

Failure of orthopedic surgical hardware with pseudoarthrosis as a cause of AD should be considered and investigated. Sitting as an initiating/aggravating factor, back discomfort and change in spinal alignment may alert the clinician to this possibility.

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