Autonomic Dysreflexia-Like Syndrome in a T12 Paraplegic During Thoracic Spine Surgery

Samuel N. Blacker, MD,*† Carlton Q. Brown, MD,* and Nicki S. Tarant, DO*†

A 19-year-old African American man with a T12 spinal cord lesion underwent a T4–L5 thoracolumbar spinal fusion. Intraoperatively, his arterial blood pressure acutely increased from 110/60 to 260/130 mm Hg without a change in heart rate. The patient did not have pheochromocytoma, carcinoid syndrome, or thyroid storm. This presentation differs from autonomic dysreflexia because the spinal cord lesion was well below T6, hypertension was elicited with somatic stimulation above the lesion, and the response required aggressive pharmacologic management. This presentation is consistent with similar cases that support a central autonomic process. (Anesth Analg 2010;111:1290–2)

Autonomic dysreflexia (hyperreflexia) has traditionally been associated with spinal cord lesions at or above T6. We report a case of severe intraoperative hypertension with unchanged heart rate, which differs from autonomic dysreflexia because our patient had a spinal cord lesion at T12, the response was elicited with somatic stimulation above the lesion, and required aggressive pharmacologic management.

CASE DESCRIPTION

A 19-year-old African American man with a remote T12 spinal cord transection presented for a T4–L5 thoracolumbar spinal fusion. The patient sustained gunshot wounds to his elbow, leg, and back with a T12 spinal cord transaction 14 months previously. He had progressive thoracolumbar angulation because his injury created difficulty with positioning in his wheelchair and with physical therapy. He had bilateral paraparesis, but he could ambulate with a walker and leg braces, and he used a condom catheter for incontinence.

The patient had undergone 4 operations after his initial injury without reported problems. Outpatient arterial blood pressures were reported to range from 110 to 120 mm Hg systolic and 50 to 60 mm Hg diastolic, with normal sinus heart rates of 60 to 70 beats per minute (bpm). The patient had no drug allergies and denied alcohol, tobacco, or illicit drug use. His physical examination was largely unremarkable except for his paresis and scars from previous operations. He weighed 72 kg.

In the holding area, a peripheral IV line was placed, midazolam 2 mg was given, and the patient was taken to the operating room. Standard ASA monitors were applied and the patient was administered oxygen. After ensuring stable vital signs, induction ensued with midazolam 3 mg, fentanyl 500 μg, and inhaled sevoflurane. After loss of lid reflexes, the patient was mask ventilated, administered 70 mg rocuronium, and was easily intubated. An arterial line and second peripheral IV line were placed after induction. The patient was turned prone without event. His anesthetic level was maintained with 0.8% to 0.9% end-tidal isoflurane along with a fraction of inspired oxygen of approximately 50%, resulting in systolic blood pressures of approximately 100 mm Hg and heart rates ranging near 60 bpm. Of note, his systolic blood pressure did increase transiently to 140 mm Hg on 1 noninvasive reading during intubation, but promptly returned to a range of 100 to 110 mm Hg without any intervention.

During the first hour of surgery, the paraspinous muscles were detached from the lumbar spine. The patient's arterial blood pressure remained in the 100 to 110 mm Hg systolic range, with heart rates of approximately 60 bpm. One hour after initial lumbar incision, the surgeons moved to the midthoracic spine while an arterial blood sample was simultaneously being drawn. The first blood pressure reading from the arterial line, after drawing the blood gas, was noted as 260/130 mm Hg and was quickly confirmed with a noninvasive cuff. The arterial tracing was of high fidelity without signs of underdampening and the transducer height was unchanged and appropriately zeroed. The blood pressure was noted to have increased very quickly and the surgeons reported a concomitant, abrupt increase in bleeding in the surgical field. The heart rate remained stable between 60 and 70 bpm. The surgeons temporarily stopped operating to allow for better hemodynamic control. Nitroglycerin (250 μg) was administered IV over the next 10 minutes in divided doses. An additional 200 μg fentanyl was also administered. Isoflurane was increased to a maximum end-tidal value of 2.3%. The heart rate eventually increased to the low 90s, but only after 150 μg of IV nitroglycerin and increased isoflurane. This was presumed to be a reflex increase in heart rate from vasodilatation, and 10 mg of labetalol was administered for blood pressure and heart rate control. The peak blood pressure during this event was 273/151 mm Hg, with maximum mean arterial blood pressure of 181 mm Hg.

The patient's vital signs returned to the previous ranges after 15 minutes of interventions described, without pulmonary, ventilatory, renal, or acid base changes. The remainder of the 6-hour surgical procedure was uneventful.

From the *Department of Anesthesiology, National Naval Medical Center, Bethesda, Maryland; and †Department of Anesthesiology, Walter Reed Army Medical Center, Washington, DC.

Accepted for publication July 9, 2010.

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Army, Department of Defense, nor the U.S. Government.

Disclosure: The authors report no conflicts of interest.

Reprints will not be available from the author.

Address correspondence to Samuel N. Blacker, MD, Walter Reed Army Medical Center, 6900 Georgia Ave., NW, Washington, DC 20307. Address e-mail to Samuel.Blacker@gmail.com.

DOI: 10.1213/ANE.0b013e3181f334b8

Report Documentation Page

Form Approved
OMB No. 0704-0188

1. REPORT DATE	2. REPORT TYPE	3. DATES COVERED
JUL 2010		**00-00-2010 to 00-00-2010**

4. TITLE AND SUBTITLE	5a. CONTRACT NUMBER
Autonomic Dysreflexia-Like Syndrome in a T12 Paraplegic During Thoracic Spine Surgery	5b. GRANT NUMBER
	5c. PROGRAM ELEMENT NUMBER
6. AUTHOR(S)	5d. PROJECT NUMBER
	5e. TASK NUMBER
	5f. WORK UNIT NUMBER
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) **Walter Reed Army Medical Center,Department of Anesthesiology,6900 Georgia Ave., NW,Washington,DC,20307**	8. PERFORMING ORGANIZATION REPORT NUMBER
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)	10. SPONSOR/MONITOR'S ACRONYM(S)
	11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release; distribution unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT

15. SUBJECT TERMS

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			
unclassified	**unclassified**	**unclassified**	**Same as Report (SAR)**	**3**	

The patient was tracheally extubated in the operating room at the end of the case. His physical examination remained unchanged from preoperatively and no postoperative complications were evident. Cognitive and neurologic examinations were unchanged postoperatively.

DISCUSSION

Our case demonstrates a sudden, unexpected intraoperative hypertensive emergency in a patient with a known spinal cord lesion at the T12 level. This patient's lesion was anatomically quite specific, because a retained bullet fragment at T12 clearly marked the level of cord injury. This level of injury was consistent with his clinical neurologic functioning. The patient had no history of hypertension or any symptoms consistent with preoperative autonomic vasomotor instability.

The differential diagnosis of isolated, abrupt intraoperative hypertension includes pheochromocytoma, carcinoid syndrome, thyroid storm, syringe swap or iatrogenic infusion of pressors, light anesthesia with pain, and autonomic dysreflexia. None of these diagnoses fit well with the presentation of our patient. Secretory vasoactive neoplasms or other endogenous hormone secretion seem unlikely, because this hypertensive event did not recur during the remainder of the procedure and the patient had no prodromal hypertensive episodes, intraoperatively or otherwise.[1,2] Iatrogenic hypertension is unlikely, because the patient received no IV vasoactive medications proximate in time of the abrupt blood pressure change and all syringes were correctly labeled and accounted for. Intraoperative stimulation with light anesthesia and pain should cause endogenous catecholamine release and should have resulted in an increase in both blood pressure and heart rate; however, no substantial increase in heart rate was ever observed in our patient. An additional possible explanation for the observed hypertension could be direct surgical stimulation of the thoracic sympathetic chain. This is unlikely in our case, because the thoracic part of the procedure had just commenced. The surgeons were separating the paraspinous muscles from the spinous processes by blunt dissection at the time of the hypertension and they were not distracting the vertebral bodies. An increased heart rate would be expected with direct stimulation of the sympathetic ganglia; none was noted. Large amounts of medications were required to control the blood pressure, even after cessation of surgical stimulation. This suggests an underlying, persistent physiologic cause for the sustained and resistant hypertension.

Classic autonomic dysreflexia does present with bradycardia, arrhythmias, and abrupt-onset hypertension up to 260 mm Hg systolic and diastolic blood pressures >170 mm Hg.[3] However, there are several significant differences. Intraoperative autonomic dysreflexia is usually described with spinal cord lesions above T6.[4] It is classically elicited by visceral not somatic surgical stimulation at an anatomic level below the cord lesion during a period of inadequate anesthesia.[5] This case is unusual in that the inciting surgical stimulation was somatic stimulation and above the cord lesion, even after initial surgical stimulation below the lesion did not result in any response. Likewise, the hypertensive response was prolonged despite cessation of surgery and required aggressive, persistent hemodynamic management. In contrast, previous clinical reports describe autonomic dysreflexia as self-limited or promptly responsive to cessation of surgical stimulation or increasing anesthetic depth.[6] Similarly, however, hyperreflexia cannot be reliably blocked unless at a depth of 2.2 minimum alveolar concentration.[7]

The hemodynamic response of autonomic dysreflexia is clinically well described but incompletely understood. It has been attributed to discontinuities in descending vasomotor pathways, baroreflex-mediated withdrawal of sympathetic drive, and blockage of afferent impulses within the dorsal and lateral columns.[8,9] The denervated terminal boutons of presynaptic sympathetic fibers become disorganized and multiply, resulting in inappropriate and exaggerated distal vasoregulatory reflexes.[10–12] *N*-methyl-D-aspartic acid and α-amino-3-hydroxyl-5-methyl-4-isoxazole-propionate overexpression is also thought to contribute to the inappropriate response.[13] The bradycardia has been attributed to high reflexive parasympathetic tone triggered by baroreceptor activity above the cord lesion.[14] Patients and animal models with autonomic dysreflexia often have high peripheral artery catecholamine sensitivity, but low resting catecholamine levels, resulting in low resting heart rate and blood pressure, which is similar to the preoperative presentation for this patient.[15,16]

Patients with low thoracic (below T6) or lumbar spinal cord lesions and paraplegia have been reported to exhibit catecholamine-induced hypertension and autonomic dysfunction of central cord origin. Roche et al.[17] reported a series of 5 African American male patients with low thoracic or lumbar traumatic paraplegia exhibiting severe nonoperative episodic hypertension and autonomic dysfunction. Using a clonidine suppression test, these patients were shown to have a central origin of their autonomic dysfunction. Other case reports suggest that autonomic dysreflexia is not limited to complete spinal cord lesions or injuries at T6 or above, as it has been reported with incomplete cord lesions from multiple sclerosis.[18]

Our patient's cord lesion was clearly at T12, which is largely below his postganglionic sympathetic fibers. Preganglionic sympathetic fibers reside within the intermediolateral column from T1 to L2. We believe the postinjury disorganization of presynaptic fibers below his lesion, combined with peripheral vascular catecholamine sensitivity, were sufficient to cause the described clinical response. The relatively low blood pressure before the event, the quick surge in blood pressure with brief somatic stimulation, sustained hypertension after withdrawal of the stimulation, and the unchanged heart rate with markedly increased blood pressure, fit with an exaggerated central catecholamine surge from a lack of sufficient descending inhibition and peripheral vascular catecholamine sensitivity.

This case demonstrates that a low thoracic spinal cord lesion can still result in a physiologically distinct autonomic dysreflexia-like phenomenon in the operating room. It is not clear how often patients with cord lesions exhibit this hemodynamic behavior. Burton et al.[19] stated that skin and muscle stimulation in rats do not elicit autonomic dysreflexia

and triggering is not consistent. Applicability of this principle to humans is unknown. Our patient certainly responded to muscle incision above the lesion, but only once.

Anesthesiologists must remain vigilant with any spinal cord–injured patient, as our case demonstrates. Historical symptoms of autonomic dysfunction may not help in assessing risk of adverse intraoperative hypertension, because no prodromal vasomotor event had occurred in our patient. We had placed an arterial line in this patient mainly for hemodynamic variability from potential rapid large surgical blood loss. The arterial line was fortuitous in the prompt diagnosis and treatment of the unexpected severe hypertension in this patient. We were fortunate that there were no postoperative complications. The increased blood pressure levels, had they been sustained, might have caused cerebral injury, hemorrhage, or even sudden death.[20,21]

This case suggests that patients with cord lesions below T6 may warrant aggressive invasive monitoring for the detection of abrupt hypertension. Preoperative planning should also include the availability and preparation of IV vasoactive medications.

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