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ANTERIOR SPINAL CORD SYNDROME AFTER INITIATION OF TREATMENT WITH ATENOLOL

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□ **Abstract—Anterior spinal cord syndrome is a rare condition with a variety of precipitating factors. Patients typically complain of weakness or paralysis of the extremities, often accompanied by pain, but frequently without a history of trauma. A 48-year-old man presented to the emergency department complaining of neck pain and inability to move his legs in the absence of trauma. Several hours prior he had seen his private physician and was given a dose of atenolol for elevated blood pressure. He had not previously been on medications for hypertension. His neurological examination revealed bilateral paralysis of the lower extremities. In the upper extremities he had weakness and sensory loss at the level of C6. Rectal tone was decreased and without sensation. Cervical and thoracic spine magnetic resonance imaging showed spondylotic disc disease, with disc herniation at C6–7 causing severe spinal canal stenosis. Despite i.v. methylprednisolone, pressors, and a prolonged intensive care unit course, the patient was discharged 5 weeks later with continued neurological deficits. Anterior spinal cord syndrome results from compression of the anterior spinal artery and often occurs in the absence of traumatic injury. The recognition, management, and prognosis of this condition are discussed. © 2010 Elsevier Inc.**

□ **Keywords—anterior spinal cord syndrome; spinal cord infarction; magnetic resonance imaging; antihypertensive agent; atenolol**

INTRODUCTION

Spinal cord infarction from anterior cord syndrome is a rare condition with a variety of precipitating factors. Patients typically complain of weakness or paralysis of the extremities, often accompanied by pain, but without recent trauma. Proprioception and vibratory sense are preserved. Despite prompt diagnosis and management of the condition, the prognosis for neurological recovery is often poor.

CASE REPORT

A 48-year-old man presented to the Emergency Department (ED) complaining of inability to move his legs. The patient was employed as a chef and began to feel numbness in his legs and hands while working, approximately 1 h before arrival. Shortly thereafter, he stated that his legs began to feel weak. This weakness quickly progressed until he was unable to stand.

The patient also reported neck pain. He stated that he had been seen 6 days prior in another ED for this pain. At that time he had no weakness or paresthesias. Cervical spine X-ray studies revealed no abnormalities. He was discharged home with a recommendation to follow-up with his primary care provider for further evaluation of his neck pain. He also was informed that his blood pressure was elevated and that he should discuss this with his physician.

On the day of presentation, the patient had visited his primary care provider and was told he had a "pinched nerve" in his neck. His blood pressure was 190/110 mm Hg, and he was given a 50-mg atenolol tablet to take

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immediately, and a prescription for ibuprofen and a 6-month supply of atenolol. His presenting symptoms began 2 h later.

He denied recent injury or illness, bowel or bladder incontinence, headache, shortness of breath, abdominal or chest pain, and fever. His past surgical history was significant for a nephrectomy secondary to a living-donor transplant for his daughter 4 years earlier. His medical history was otherwise unremarkable, with the exception of the newly diagnosed hypertension. He smoked two packs of cigarettes and drank a pint of gin daily. He denied any intravenous or illicit drug use.

Initial vital signs were blood pressure 110/60 mm Hg, pulse 56 beats/min, respiratory rate 16 breaths/min, temperature 35.9°C (96.7°F), and oxygen saturation of 97% on room air. A bedside glucose reading was 103 mg/dL. The patient was lethargic but reacted to loud verbal stimulus. His neurological examination revealed bilateral paralysis of the lower extremities. In the upper extremities he had 0/5 strength in the biceps and brachioradialis muscles, 3/5 in the wrist extensors, and 4+/5 strength in the triceps (1). He had loss of light touch and pin prick sensation at the level of C6. Reflexes were absent in the lower extremities, biceps, and brachioradialis. Triceps reflexes were preserved. Vibratory and positional senses were preserved throughout. Rectal tone was decreased and without sensation. All cranial nerves were intact. He had no vertebral point tenderness. The remainder of his examination was unremarkable.

Because the mechanism of his paralysis was unclear at this time, the patient received 30 mg/kg of methylprednisolone over 45 min initiated in the ED, followed by an infusion of 5.4 mg/kg/h given over the next 23 h. Before magnetic resonance imaging (MRI), the patient was fiberoptically intubated by anesthesiology while awake for prophylactic airway protection using bilateral glossopharyngeal and superior laryngeal nerve blocks. After MRI, the patient's blood pressure dropped to 70/40 mm Hg. The patient had received no medication for sedation during MRI or intubation. We believed that spinal shock was compounding the beta-blocker-induced hypotension. Intravenous fluids were administered and a phenylephrine infusion was started.

Cervical and thoracic spine MRI showed spondylotic disc disease, with disc herniation at C6–7 causing severe spinal canal stenosis (Figures 1, 2).

He was diagnosed with anterior cord syndrome secondary to spinal stenosis and disc herniation causing compression of the vascular supply and was admitted to the medical intensive care unit. Over the course of his hospital stay he developed upper gastrointestinal bleeding secondary to stress gastritis, a lower extremity deep

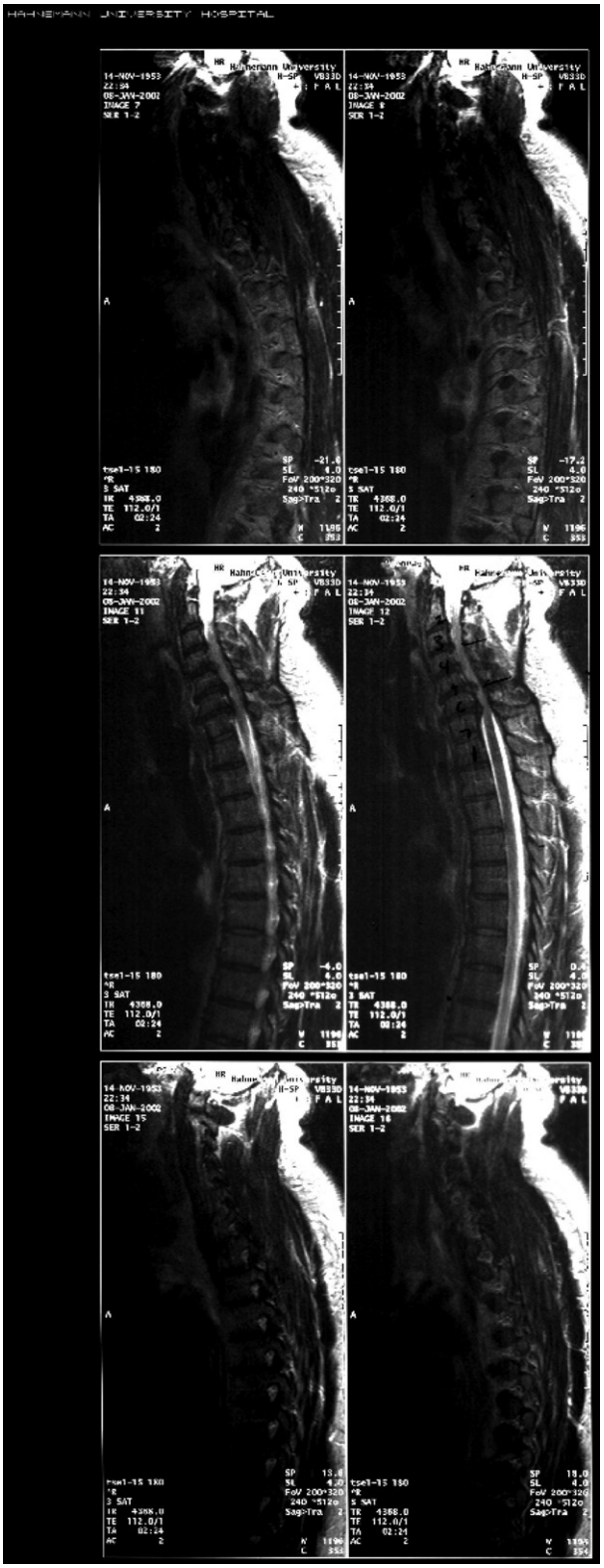

Figure 1.