

Class A ventral spinal epidural hematoma as a rare complication of hypertensive crisis

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ABSTRACT

Spinal epidural hematoma is a rare but severe medical condition that can cause neurological deficits and disability. In this case report, a 53-year-old man with poorly controlled hypertension and end-stage renal disease presented with hypertensive crisis and a one-week history of back pain, abdominal pain, and pain radiating down his legs. Despite rapid blood pressure control, the patient was found to have a 21 mm ventral epidural hematoma at T10 causing thoracic cord compression and paraplegia. The patient underwent surgical hematoma evacuation but remained paraplegic with minimal motor and sensory recovery. Spinal epidural hematoma is a complication of hypertensive crisis. Clinicians should be aware of the possible development of spinal epidural hematoma in patients with hypertensive crisis, even in cases with no obvious risk factors. Prompt diagnosis and treatment are crucial for preventing permanent neurological damage and improving patient outcomes. Further research is needed to better understand the underlying mechanisms and risk factors for spinal epidural hematoma in patients with hypertensive crisis.

Keywords: Hypertensive crisis, spinal epidural hematoma, paraplegia; neurosurgery

INTRODUCTION

Spinal epidural hematomas (SEH), a rare medical condition, can cause significant neurological deficits and disability by the collection of blood between the dura mater and the vertebral column, compressing the spinal cord and leading to back pain, weakness, numbness, and paralysis. Various factors, like trauma, vascular malformations, bleeding disorders, neoplasms, and medications,⁴ can cause SEH. Hypertension (HTN) and hypertensive crisis (HC) are potential risk factors for SEH.² Hypertensive crisis is a medical emergency with a rapid and severe increase in blood pressure, causing organ damage

and potential death without prompt treatment.⁵ Though SEH is uncommon in HC cases, it requires consideration in these cases.

The causes of hypertensive crisis include medication noncompliance, renal failure, preeclampsia/eclampsia, and drug abuse, and it presents with headache, visual disturbances, confusion, seizures, chest pain, and shortness of breath. Effective management involves aggressive blood pressure control with antihypertensive medications and management of underlying conditions.⁵ Early recognition and management of SEH and HC are vital for preventing permanent neurological damage and improving patient outcomes. Clinicians should know that SEH is an unusual HC complication, even without apparent risk factors like trauma or bleeding disorders. Rapid diagnosis and treatment can change patient outcomes; more studies on the underlying mechanisms and risk factors for SEH in HC patients.

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This case report highlights a rare SEH occurrence in a patient with HC and underscores its clinical significance.

CASE

A 53-year-old man with a medical history of hypertension and end-stage renal disease presented to the Emergency Department with a one-week history of dry cough, fatigue, headache, back pain, abdominal pain, and pain radiating down his legs. The patient reported that he is normally compliant with his medications and dialysis. However, he missed dialysis that morning due to a headache. He denied any recent trauma or injury. His home medications include azithromycin, clonidine, hydralazine, metoprolol tartrate, sevelamer, and Tessalon Perles. Upon examination, his blood pressure was

found to be 204/107 mmHg. The patient was started on IV hydralazine and metoprolol for blood pressure control and admitted to the Critical Care Unit for further management. His admission creatinine was 8.8 mg/dL; his blood urea nitrogen was 54 mg/DL. He had low hemoglobin (12.2 gm/dL) with low sodium (128 mEq/L) and high potassium (5.5 mEq/L). Other laboratory values were within normal limits; he had a negative urinalysis and urine toxicology. Coagulation tests were unavailable at this time at the time of admission.

Despite aggressive medical management with nicardipine, the patient remained somnolent and lethargic. Emergent dialysis was performed, his hypertension improved, and he was stable. A day later, his weakness progressed to complete paraplegia. A magnetic resonance imaging (MRI) of the thoracic spine revealed a 21 mm ventral epidural

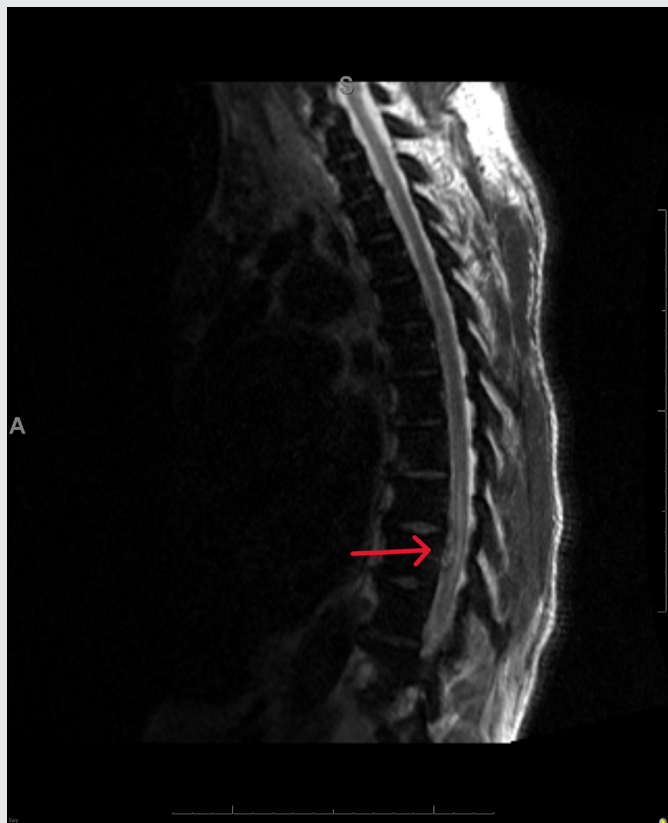


Figure 1. Sagittal T2 view, reviewing 21 mm hematoma at T10 (arrow) pre-op (day 0).

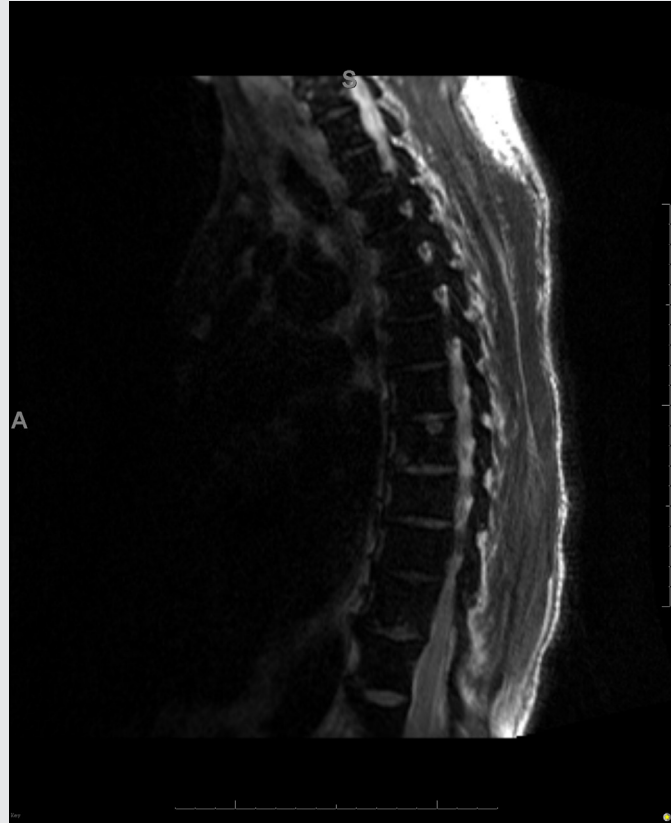


Figure 2. Sagittal T2 view of the distal cord, with no hemorrhage pre-op (day 0).

hematoma at T10 causing thoracic cord compression with associated cord edema from T9–T11 (Figures 1–2). Neurosurgery was consulted. The patient underwent emergent T9–T11 laminectomy and evacuation of the hematoma. Following the surgery, the patient remained paraplegic with minimal motor and sensory recovery status. No other symptoms were described at this time. Before this admission, he had been independent, ambulating and doing daily living activities with moderate physical activities.

He was then admitted to the inpatient rehabilitation unit for physical and occupational therapy with tapering dexamethasone. His rehabilitation course was complicated by *Clostridium difficile* enterocolitis and cerebrovascular accident with subsequent seizure. He was readmitted and intubated in the Intensive Care Unit. During the second admission, he had a repeat MRI T spine 9 days later showing

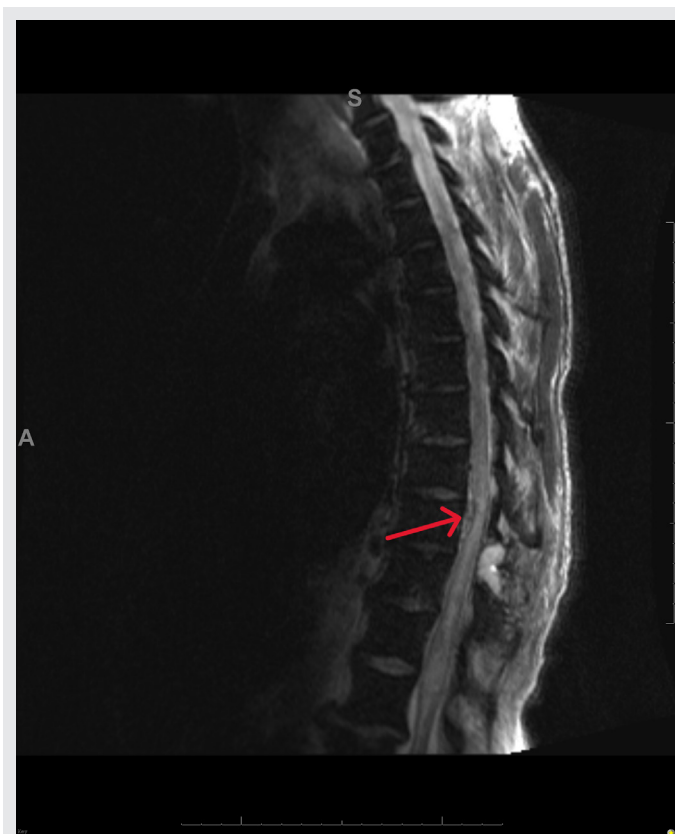


Figure 3. Sagittal T2 view of the hematoma with minimal change at T10 (arrow) post-op (day 18).



Figure 4. Sagittal T2 view of the distal cord with hemorrhage and signs of surgery (arrow) post-op (day 18).

some improved T spine cord edema now T9 and below, epidural fluid collection at T10, and an MRI L spine showed the persistent mass like enlargement of conus without clear evidence of discitis/osteomyelitis, spinal stenosis L2–4 (Figures 3–4). He was subsequently transferred to Dallas for ongoing treatment and rehabilitation.

DISCUSSION

Spinal epidural hematomas are incredibly rare, mostly appearing dorsally because of the spinal canal's anatomy. The dura mater attaches to the posterior longitudinal ligament, making the anterior epidural space rather small. As a result, ventral spinal epidural hematoma is an exceptionally uncommon occurrence.¹ The case presented here stands out as

unique and unusual, featuring a large ventral SEH, as clearly seen in Figure 1.

The exact cause of SEH in hypertensive crisis isn't fully understood, but there are a few proposed theories. One idea suggests that the rapid surge in blood pressure during HC could rupture small blood vessels in the epidural space, leading to hematoma formation.^{6,7} Another hypothesis is that hypertension may directly damage the spinal cord vessels, causing hemorrhage in the epidural space.⁶ Moreover, hypertension might enhance the permeability of the epidural vasculature, leading to blood extravasation.⁸ In this particular case, the patient's hypertensive crisis, coupled with end-stage renal disease and missed dialysis, likely caused the SEH.

End-stage renal disease is a known risk factor for bleeding, given its potential to cause platelet dysfunction, vascular calcifications, and anemia.⁹ Only three SEH cases in patients with renal disease undergoing hemodialysis have been reported.¹¹ Furthermore, patients with end-stage renal disease may require anticoagulation or antiplatelet therapy for concurrent cardiovascular conditions. Therefore, meticulous monitoring becomes essential to identify any signs of bleeding or hematoma formation, especially when these patients experience hypertension or hypertensive crisis.

Persistent SEH can occur due to several reasons. One possibility is rebleeding, which occurs when the hematoma reaccumulates after surgical evacuation.¹⁰ As noted in Figure 4, signs of hemorrhage are noted along the distal cord that were not present pre-operatively. This can occur due to inadequate evacuation or incomplete hemostasis during the initial surgery. Other factors, such as hypertension, antiplatelet or anticoagulant use, or bleeding disorders, may increase the risk of rebleeding. Figure 3 shows evidence of the hematoma, with slight change in size, indicating possible incomplete evacuation. The remaining clot can act as a source of inflammation and can continue to cause spinal cord compression.

The case presented here is a rare occurrence of ventral SEH in a patient with HC and end-stage renal

disease. The rarity of this case can be attributed to the fact that most SEH have a dorsal location and are caused by trauma or bleeding disorders. In addition, the presence of end-stage renal disease highlights the increased risk of bleeding in patients with comorbidities. Early recognition and management of SEH and its risk factors are crucial to prevent permanent neurological damage and improve patient outcomes.³ Further research is needed to better understand the underlying mechanisms and risk factors for SEH in patients with HC. This will likely depend on case series reports.

CONCLUSION

Spinal epidural hematomas, as an uncommon complication of HC, merits clinical attention. This case report highlights the rare occurrence of SEH in a patient with HC and end-stage renal disease, causing significant neurological deficits and disability. This case emphasizes the importance of prompt diagnosis and management of both SEH and HC to prevent permanent neurological damage and improve patient outcomes. Clinicians should be aware of the potential for SEH as an unusual complication of HC, even in cases with no obvious risk factors, such as trauma or bleeding disorders.

Permission: Verbal consent was obtained from patient for the case report.

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