





## Spinal epidural hematoma presenting with hypercapnic respiratory failure in diabetic ketoacidosis

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### ABSTRACT

Spinal epidural hematoma (SEH) is a rare but potentially devastating condition that may lead to severe neurological deficits if not recognized early. Diagnosis may be challenging when SEH occurs in patients with concurrent metabolic or infectious disorders. We report a case of SEH in a 70-year-old male patient who presented with diabetic ketoacidosis (DKA) and pneumosepsis. The patient was admitted to the intensive care unit and received treatment for DKA and infection. After metabolic stabilization, sedation was discontinued and the patient was extubated. Shortly after extubation, he developed acute respiratory failure accompanied by tetraplegia. Emergency contrast-enhanced cervicothoracic magnetic resonance imaging revealed an epidural hematoma extending from the C1-2 level to T6, causing significant spinal cord compression. Surgical intervention was not performed because of delayed diagnosis and lesion localization. This case highlights the importance of detailed neurological evaluation in patients with complex clinical presentations. Central causes such as SEH should be considered in cases of unexplained respiratory failure or sudden neurological deterioration.

**Keywords:** Spinal epidural hematoma, diabetic ketoacidosis, hypercapnia, tetraplegia

### INTRODUCTION

Spinal epidural hematoma (SEH) is a rare but potentially devastating clinical entity with high morbidity.<sup>1</sup> It is estimated to occur in approximately 1 per 100,000 to 1 per 1,000,000 individuals per year in the general population.<sup>2</sup> SEH typically presents with sudden onset neck or back pain and may rapidly progress to neurological deficits such as motor weakness or paralysis.<sup>3,4</sup> Delayed diagnosis may lead to irreversible outcomes including permanent tetraplegia or death.<sup>4</sup> When SEH occurs concurrently with metabolic conditions such as diabetic ketoacidosis (DKA) and infectious processes such as pneumonia, diagnostic recognition becomes even more challenging.

This case of cervicothoracic SEH in a patient hospitalized for DKA and pneumosepsis illustrates the difficulty of timely diagnosis in patients with overlapping metabolic and infectious disorders.

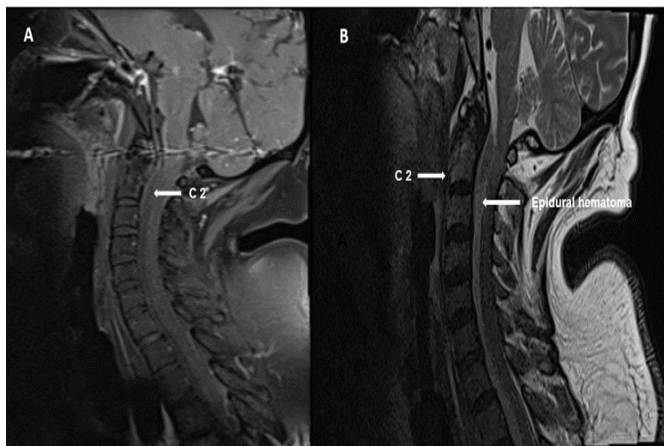
### CASE

Written informed consent was obtained from the patient for publication. A 70-year-old male with a history of

hypertension, diabetes mellitus, and benign prostatic hyperplasia presented to the emergency department with generalized weakness and deterioration in overall condition, reporting persistent neck and back pain for one week without a history of trauma. The patient was conscious, oriented, and cooperative, with moderately impaired general condition.

Initial laboratory tests revealed hyperglycemia (blood glucose 385 mg/dl), positive urinary ketones (++) , elevated C-reactive protein (365 mg/L), procalcitonin (1.7 ng/ml), and leukocytosis (white blood cell count 37,000/ $\mu$ L). Arterial blood gas analysis showed combined metabolic and respiratory acidosis (pH 7.1, pCO<sub>2</sub> 56 mmHg, HCO<sub>3</sub> 14 mmol/L). Peripheral oxygen saturation on room air was below 90%, accompanied by respiratory distress. Thoracic computed tomography demonstrated areas of consolidation, consistent with pneumosepsis. The patient was admitted to the intensive care unit (ICU) for management of concurrent DKA and pneumosepsis. Within approximately one hour, respiratory distress progressed, accompanied by carbon dioxide retention (pCO<sub>2</sub> 85-86 mmHg) and impaired

consciousness, necessitating endotracheal intubation. Neurological assessment was not feasible due to sedation and analgesia initiated after intubation. Arterial blood gas analysis demonstrated combined metabolic and respiratory acidosis, and treatment for DKA was initiated according to endocrinology recommendations. At the 16<sup>th</sup> hour of ICU follow-up, blood glucose levels normalized and ketonemia resolved, allowing sedation to be discontinued. Extubation was performed at the 20<sup>th</sup> hour; however, within minutes, the patient developed acute respiratory failure and complete motor paralysis, necessitating immediate re-intubation. Emergency cervical magnetic resonance imaging (MRI) identified a fluid-containing collection at the C1-2 level, and contrast-enhanced cervicothoracic MRI demonstrated an epidural hemorrhagic collection extending from C1-2 to T6, causing anterior spinal cord compression (Figure). Neurosurgical consultation concluded that surgical intervention was not feasible due to craniospinal localization and delayed diagnosis.



**Figure.** Contrast-enhanced cervicothoracic magnetic resonance imaging demonstrating an epidural hematoma extending from the C1-2 level to the upper thoracic spine, causing anterior compression of the spinal cord. (A) Upper cervical level demonstrating the epidural hematoma. (B) Spinal cord compression caused by the epidural hematoma

Mechanical ventilation was maintained for six days, after which tracheostomy and percutaneous endoscopic gastrostomy were performed in the same session. Following improvement of pneumonia, the patient was transferred to a palliative care unit on the 25<sup>th</sup> day of hospitalization with home-type mechanical ventilation support.

## DISCUSSION

SEH typically presents with acute neck or back pain and may progress rapidly to motor deficits or paralysis.<sup>3-5</sup> In this case, neck and back pain was the initial symptom, providing an important clue for spinal epidural pathology. However, the simultaneous presence of DKA and pneumonia contributed to a delay in diagnosis.

In DKA, compensatory hyperventilation and resultant hypocapnia are typically expected as a physiological response to metabolic acidosis.<sup>6,7</sup> In contrast, this patient exhibited hypercapnia (pCO<sub>2</sub> 56 mmHg), which could not be explained by DKA alone and suggested involvement of the respiratory muscles. High cervical spinal cord compression can impair respiratory function by affecting the phrenic nerve, which

primarily originates from the C3-C5 segments and is essential for diaphragmatic function.<sup>8</sup> Although the hematoma was located at the C1-2 level, its mass effect extended along the cervical spinal cord, disrupting diaphragmatic and intercostal muscle innervation and resulting in hypercapnic respiratory failure.

The exact mechanism underlying spontaneous SEH remains unclear. Reported risk factors include anticoagulant or antiplatelet therapy, hypertension, anemia, diabetes, hypothyroidism, pregnancy, and activities such as deep diving.<sup>10</sup> In our patient, there was no history of trauma or use of anticoagulant medication, and no evidence of coagulopathy was identified. Nevertheless, the presence of diabetes and metabolic disturbances associated with DKA may have contributed to vascular fragility and spontaneous hemorrhage.

There are currently no established guidelines for the management of spontaneous SEH. Early surgical decompression is generally preferred, but conservative management may be considered in selected patients under close neurological monitoring.<sup>5</sup> Optimal neurological outcomes are associated with decompression performed within the first 24 hours after symptom onset.<sup>9</sup> In this case, however, the craniocervical location of the lesion, delayed diagnosis, and high surgical risk precluded operative intervention.

Clinicians should maintain a high index of suspicion for spinal pathology in patients with multifactorial conditions who develop unexplained hypercapnia or acute neurological deterioration.

## CONCLUSION

This case highlights the importance of thorough history taking and neurological examination in diagnosing SEH. Unexplained hypercapnia or sudden neurological deterioration in patients with multifactorial conditions should prompt consideration of spinal pathology.

## ETHICAL DECLARATIONS

### Informed Consent

Written informed consent was obtained from the patient(s) included in this report. Signed consent forms are retained by the authors and are available upon request.

### Peer Review Process

This report underwent external peer review.

### Conflict of Interest

The authors declare no conflicts of interest.

### Financial Disclosure

This case report did not receive any financial support.

### Author Contributions

Concept: PDA, ACS; Design: PDA, GK, ACS; Control: ACS, EK; Resources: PDA, GK; Materials: PDA, GK; Data Collection and/or Processing: PDA, GK, EK; Analysis and/or Interpretation:

PDA, ACS, EK; Literature Review: PDA, GK, ACS; Article Writing: PDA, GK, EK, ACS; Critical Review: ACS, EK.

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