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Research Article

Epidural Hematoma in a Patient with Undiagnosed Vitamin K Deficiency: A Case Report

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Abstract

A 72 year old man presented for colostomy repair prompting epidural catheter placement for pain management. A prolonged activated partial thromboplastin time (aPTT) coupled with degenerative spine disease were noted prior to placement. Postoperatively, he developed shortness of breath and leg weakness. This impelled a computed tomography (CT) scan for pulmonary embolism (PE) evaluation, which revealed an epidural hematoma. Stable neurologic findings prompted conservative management. In conclusion, 1) prolongation in aPTT should prompt consideration before neuraxial procedures, 2) vitamin K deficiency is a risk factor for epidural hematoma, and 3) hematoma management should be dictated by progression of neurologic findings.

Keywords: Epidural Hematoma; Vitamin K Deficiency; activated partial thromboplastin time

Glossary of Terms

aPTT: activated partial thromboplastin time

ASRA: American Society of Regional Anesthesia and Pain Medicine

CT: computed tomography

MRI: magnetic resonance imaging

PE: pulmonary embolism

PT: prothrombin time

Introduction

Epidural hematoma is a collection of blood within the epidural space and is caused by the rupture of veins within the Batson vertebral venous plexus [1]. This is one of most dreaded complications that can occur from accessing the posterior epidural compartment via needle or catheter. The incidence of clinically significant hematomas after epidural block or catheter insertion are estimated to be 1 in 150,000 [2]. However, the true incidence of epidural hematomas (symptomatic or asymptomatic) is unknown. Symptomatic epidural hematomas commonly present with new onset motor weakness of lower extremity accompanied by sharp back and/or leg pain [2]. Worsening neurologic deficits generally require urgent neurosurgical decompression. In contrast, improving deficits can be managed via close monitoring [3]. Although clinical practice can vary between institutions, the American Society of Regional Anesthesia and Pain Medicine (ASRA) recommends neuraxial procedures be avoided in patients with underlying coagulopathies [4].

An isolated prolonged activated partial thromboplastin time (aPTT) is a commonly encountered coagulopathy with a long list of differential diagnoses including Lupus Anticoagulant, heparin use, von Willebrand Disease, vitamin K deficiency, or other coagulation factor deficiencies [5]. Of the various etiologies, the most common cause is the presence of lupus anticoagulant (>50%), but this confers no increased risk of bleeding.[4] Vitamin K deficiency is one of the reversible causes of prolonged aPTT that causes an increased risk of bleeding [4].

Vitamin K is an essential cofactor to multiple hepatic enzymes required for the activation of coagulation factors II, VII, IX, and X. Vitamin K cannot be synthesized by the body, and therefore must be ingested, packaged into micelles, and absorbed by mucosal epithelial cells of the small intestine. Consequently, any conditions that impair fat absorption have the potential to cause vitamin K deficiency. Common examples leading to deficiency include dietary restriction, malabsorption, major surgery, antibiotic use, and hepatic disfunction [6]. Procedures involving the terminal ileum can impair resorption of bile acids and result in depletion of the bile acid pool, impeding micelle formation and vitamin K transport into the intestinal mucosal cells [7]. Though the majority is ingested, a fraction of vitamin K is produced by bacterial species of the GI tract. It is well established that antibiotic use may contribute to vitamin K deficiency via eradication of gut flora [8, 9]

Case Description

Mr. X was a frail 72 year old man presenting for colo-vesicular-fistula/colostomy repair. His history was significant for chronic obstructive lung disease and advanced thoracic spondylosis with radiographic evidence of thoracic scoliosis and multiple-level compression fractures with spine rotation (figure 1). He was severely physically deconditioned and had been wheelchair-bound for the prior 6 months secondary to chronic hip pain.

On the day of his operation, he had a aPTT of 41 seconds (beyond the upper normal limit of 35 seconds). In light of his previous uneventful abdominal surgeries, normal prothrombin time (PT) and platelets, the team deemed it safe to proceed with a thoracic epidural catheter at the T8/9 level. The epidural placement was reported without technical difficulty, and his operation was uneventful.

On post-operative day one, roughly 18 hours after epidural placement, he was able to participate in physical therapy. Following physical therapy, around 32 hours after epidural placement, he progressively felt cramping pain in between his scapulae, chest muscle tightness, shortness of breath, weakness bilaterally in his arms and legs. He did not report these symptoms at that time.

Mr. X's symptoms continued to worsen, causing him to arouse in the middle of night. These symptoms peaked approximately 36 hours after epidural placement. Eventually, the Acute Pain Service was notified and the 1/16th% bupivacaine epidural infusate was stopped. On evaluation, he had significant respiratory distress and tachycardia. He reported improving symptoms, but still complained of weakness with diffuse back and chest cramping. Exam revealed no tenderness to palpation over the epidural insertion site, intact muscle strength bilaterally in the upper extremities, and 3/5 muscle strength bilaterally in the lower extremities. His ongoing respiratory distress prompted the surgical team to begin a workup for pulmonary embolism (PE). A computed tomography (CT) scan was obtained, but initial read did not reveal a PE. However, upon further study, the CT was found to reveal a high density fluid collection in the spinal canal from C7 to T9 (figure 2), which was consistent with blood products (figure 3). Thus, the diagnosis of epidural hematoma was established and the epidural catheter was removed in preparation for possible magnetic resonance imaging (MRI) or surgical intervention (his aPTT was 45 at that time).

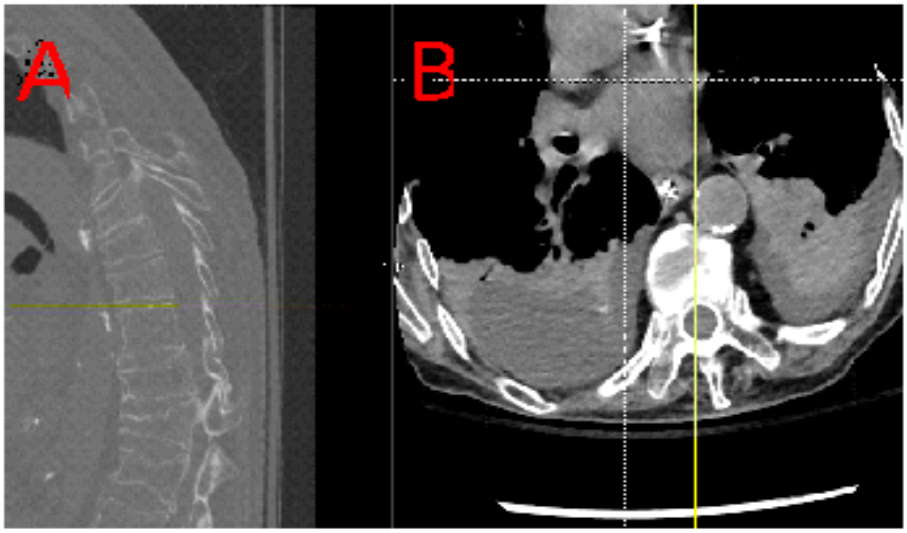

Figure 1: *Pre-operative baseline thoracic computed tomography (CT) scan. Panel A. Sagittal view of thoracic spine demonstrates significant kyphosis and scoliosis, with chronic compression fracture of T8-T10. Panel B. Axial view at T6-T7 disc space demonstrates significant spinal rotation to the left.*

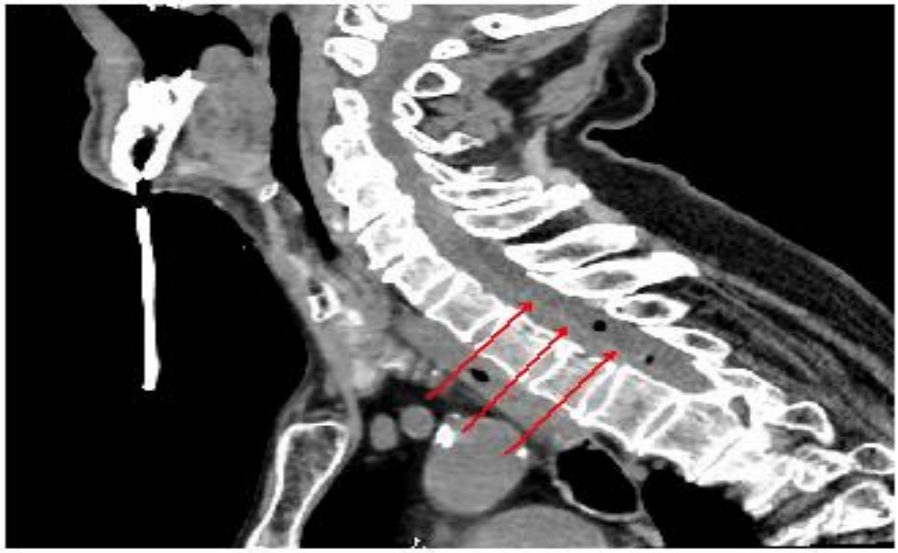

Figure 2: *CT scan of the thoracic spine with fluid collection extending throughout the dorsal surface of the cervical and thoracic spinal cord (red arrows).*

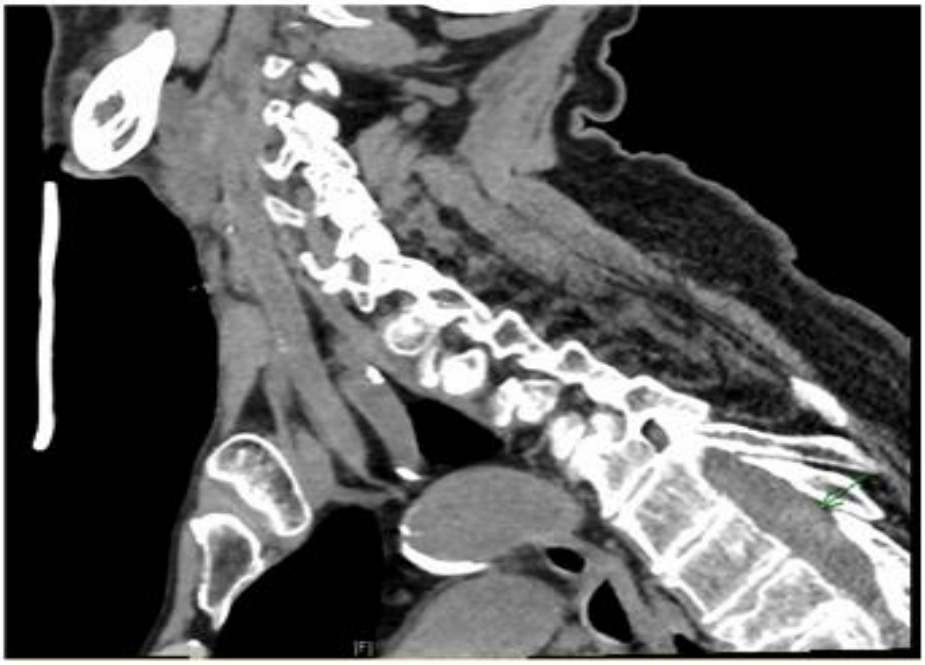

Figure 3: *CT of the thoracic spine with dense fluid collection consistent with blood around T8-9 (green arrow) with ventral displacement of the spinal cord.*

The patient's leg weakness, back and chest cramping continued to improve and neurosurgery supported ongoing conservative management with serial exams. Given respiratory distress and his inability to lie supine on the MRI table, an MRI of the thoracic spine was withheld. All subsequent neuro exams were consistently stable, and his symptoms continued to improve. A surveillance CT scan of the thoracic spine on postoperative day six demonstrated a posterior epidural fluid collection, measuring up to nine millimeters in thickness extending from T5 to T9. It was deemed to be essentially unchanged from the prior scan.

Hematology was consulted, and following radiologic detection of the epidural hematoma, his coagulation labs were repeated. His PT was found to be prolonged to 16.4 seconds with a aPTT also prolonged to 47 seconds. A 1:1 mixing study revealed a complete correction of the PT, but an incomplete correction of the aPTT from 49 seconds to 37 seconds. This sample further prolonged to 41 seconds after incubation. The lack of immediate correction raised concern for an antibody-mediated phenomenon, likely an antiphospholipid antibody causing an in-vitro lupus anticoagulant effect. Lupus anticoagulant testing confirmed the presence of a lupus anticoagulant antibody. Given these findings, both hematology and laboratory medicine concluded that the patient's mildly perturbed coagulation parameters were likely related to a mild Vitamin K deficiency with a disproportionate prolongation in the aPTT from a clinically insignificant lupus anticoagulant antibody. This was further supported by improvement in both the PT and aPTT after administration of Vitamin K.

Discussion

This case revealed two important challenges faced by his care team regarding neuraxial techniques. The first was a decision faced by the regional anesthesia team on whether or not to place an epidural catheter in a patient with an isolated prolonged aPTT. Although this laboratory abnormality may signify an underlying coagulopathy, a careful hematologic work-up was probably warranted in this case to characterize and localize the correctable or non-correctable causes.

Hospitalized patients are at increased risk of developing vitamin deficiencies, as many of the common contributing causes of deficiency occur simultaneously during a hospital course. Mr. X's frailty on admission, and prior abdominal surgical history, calls into question his nutritional status and intestinal integrity. A subclinical vitamin K deficiency on admission would have been further exacerbated by the subsequent surgical procedure, multi-day hospitalization requiring dietary restriction, and perioperative antibiotic use. The developing prolongation of Mr. X's PT and aPTT over the duration of his hospitalization supports that the etiology was likely multifactorial.

Providers faced with this scenario should decide whether patients would benefit from Vitamin K administration prior to neuraxial procedures. A dose of intravenous Vitamin K (80-240 mg) is recommended for urgent reversal, and begins to correct aPTT within 2 hours [10, 11]. Clearly, such deficiencies would be best treated before a patient becomes an operative candidate. That is, coagulopathies that can be treated with nutrition or replacements before the day of surgery would be beneficial to all.

Although the benefit of placing an epidural for postoperative pain management was deemed by the providers to outweigh the risks, this case emphasizes that significant prolongation of aPTT should trigger caution before invasive procedures are attempted. ASRA guidelines (2018) for epidural catheter placement are to postpone the procedure until coagulation normalizes [4]. For example, it is recommended to hold intravenous heparin for 4-6 hours before placement, and to hold therapeutic subcutaneous heparin for 6 hours before placement. The recommendation emphasizes checking coagulation status beforehand, and to place an epidural only after aPTT has normalized [4].

Mr. X's postoperative course posed another challenging scenario for his care team. Not until 32 hours after epidural placement did Mr. X report

subjective weakness and diffuse stiffness. This contrasts the typically described presentation of an evolving focal deficit in an extremity, accompanied by localized back pain and saddle anesthesia [3]. It was not until a CT scan was ordered to rule out a pulmonary embolism that the epidural fluid collection was discovered. Importantly, this case highlights that management of an epidural hematoma should be dictated by the patient's symptoms and physical exam, not by radiographic findings alone.[12] His improving symptoms, and not his radiologic findings, guided the decision to defer surgical intervention, and he returned to his baseline without surgical intervention.

Financial Disclosures: None

Conflicts of Interest: None

Authors: Jonathan T Rawley: This author helped with background, case details and discussion. Jiang Wu: This author helped with case details, figures and discussion. Ryan J Jense: This author helped with background and discussion. Emma E Fulton: This author helped with the hematology portion of the discussion. Robert Diep: this author helped with the hematology portion of the discussion.

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