Case Report

Malabsorption mayhem: A Vitamin K story in trauma care

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ABSTRACT

A young adult male presented with persistent vomiting and a history of recent trauma and abdominal surgery. He was found to have significant coagulopathy, evidenced by marked prolongation of prothrombin time and activated partial thromboplastin time. Laboratory and clinical evaluation revealed multifactorial Vitamin K deficiency, likely due to malnutrition, malabsorption, and ongoing physiological stress. Prompt administration of Vitamin K resulted in rapid correction of coagulation parameters, facilitating safe surgical intervention without excessive intraoperative bleeding. Despite aggressive surgical and supportive management, the patient's course was complicated by recurrent infections and ultimately fatal septic shock. This case underscores the importance of early recognition of Vitamin K deficiency in trauma and surgical patients, especially those with risk factors for malnutrition or gastrointestinal compromise. Timely correction of coagulopathy is vital to minimize perioperative risks and improve clinical outcomes.

Key words: Blunt trauma abdomen, Coagulation dysfunction, Coagulation factors, Malabsorption, Traumatic injury, Vitamin K-dependent

Vitamin K deficiency is often overlooked but can have profound clinical consequences, particularly in the setting of acute illness or trauma. As a fat-soluble vitamin, Vitamin K is essential for the hepatic synthesis of coagulation factors II, VII, IX, and X, as well as for bone and cardiovascular health. Deficiency may result from inadequate dietary intake, malabsorption, prolonged antibiotic use, or chronic liver disease, leading to bleeding diathesis and impaired surgical outcomes. In adults, classic manifestations include an elevated prothrombin time (PT) that corrects with Vitamin K supplementation.

We report a rare case of post-traumatic diaphragmatic hernia presenting with persistent vomiting and coagulopathy secondary to Vitamin K deficiency, which was resolved with timely supplementation and enabled safe surgical intervention [1-3].

CASE PRESENTATION

A male in his late 20s presented to the outpatient department with a 5-day history of postprandial vomiting of undigested food. He reported frequent intake of oral rehydration solution (ORS). He denied hematemesis, melena, abdominal pain, fever, or chest pain. His medical history was significant for laparoscopic repair of a left diaphragmatic tear and internal fixation of a humeral shaft fracture following a road traffic accident 2 months prior.

Clinical examination revealed undernutrition, mild tachypnea (20 breaths/min), and decreased air entry in the left hemithorax. Bedside ultrasonography (E-FAST) was negative.

On admission, laboratory investigations revealed the following: Hemogram: Hemoglobin was low (11.7 g/dL), normal total leukocyte count (5800 cells/mm^3), and platelet count (233,000 cells/mm^3).

Biochemical analysis showed reduced albumin (2.5 g/dL) and total protein (6.3 g/dL), normal liver enzymes (serum glutamic pyruvic transaminase – 45 U/L, serum glutamic oxaloacetic transaminase – 38 U/L, alkaline phosphatase: 147 U/L), and bilirubin levels (direct – 0.86 mg/dL, total – 1.2 mg/dL). Renal function tests were within normal limits (creatinine – 0.9 mg/dL, blood urea nitrogen – 40). Random blood glucose was 109 mg/dL, with electrolytes in the normal range (sodium: 135 mmol/lt, potassium: 3.5 mmol/lt).

Conventional coagulation parameters showed prolongation of activated partial thromboplastin time (aPTT) (46.8 s) and PT (83.2 s, international normalized ratio: 7.26 s), mildly elevated plasma fibrinogen level

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(654 mg/dL), and d-dimer on the higher side of normal range (0.6 µg/dL). A mixing study was performed with a PT of 83.2 s, which was corrected to 32 s. An assay of clotting factors revealed reduced activity of Vitamin K-dependent factors II, VII, IX, and X. The patient had no history of bleeding disorders or use of oral anticoagulants. Furthermore, his liver function tests were within normal limits. Thus, a diagnosis of Vitamin K deficiency was made. After intravenous Vitamin K administration, coagulation parameters normalized: PT 14.6 s (INR 1.09) and aPTT 31.9 s. Point-of-care viscoelastic testing using rotational thromboelastometry (ROTEM) demonstrated global coagulopathy and highlighted ROTEM's sensitivity in detecting abnormalities in both extrinsic and intrinsic pathways during Vitamin K deficiency (Fig. 1).

Contrast-enhanced computed tomography (CT) of the abdomen revealed a 4 cm defect at the cardia extending to the gastroesophageal junction with moderate herniation of abdominal contents through the diaphragm. Following resuscitation and correction of coagulopathy with intravenous Vitamin K, the patient underwent emergency exploratory laparotomy. Intraoperatively, an 8 × 8 cm defect in the left hemidiaphragm was found, with herniation of the omentum and stomach into the left thoracic cavity. The stomach appeared ischemic. Contents were reduced, and splenectomy was performed due to inseparable adhesions and compromised vascularity. Two gastric perforations were identified: a 3 × 4 cm defect on the greater curvature and a 5 × 1 cm laceration at the gastroesophageal junction, which were repaired primarily in two layers. The diaphragmatic defect was closed with non-absorbable sutures. A feeding jejunostomy and decompressive gastrostomy were created for post-operative management.

The early post-operative period was complicated by persistent pyrexia and respiratory distress. CT thorax confirmed left-sided pyopneumothorax and a recurrent diaphragmatic defect. On post-operative day 24, the patient underwent a combined left lateral thoracotomy and re-laparotomy. Approximately 500 mL of purulent material was evacuated from the empyema cavity, a new 3 × 1 cm diaphragmatic defect was repaired, and a left intercostal drain was placed. Despite aggressive surgical intervention, broad-spectrum antimicrobial therapy, and intensive care unit support, the patient deteriorated and developed severe sepsis progressing to refractory septic shock with multiorgan dysfunction. He died 4 weeks after the second operation.

DISCUSSION

Vitamin K deficiency, though uncommon in adults, can precipitate significant coagulopathy, especially in trauma and complex surgical contexts. Vitamin K is required for γ-carboxylation of coagulation factors II, VII, IX, and X, as well as proteins C and S, which are critical for hemostasis. Secondary deficiency in adults is typically due to poor dietary intake, gastrointestinal malabsorption, prolonged use of broad-spectrum antibiotics, or hepatobiliary disease. Trauma patients are particularly at risk due to acute blood loss, frequent transfusions, metabolic stress, and periods of inadequate enteral nutrition [4]. Similar mechanisms were noted by Nicoletto *et al*., who reported that poor intake, intestinal infection, and antibiotics aggravated the deficiency, and by Nakai *et al*., who reported that afferent loop obstruction was the cause [5,6].

In this case, chronic undernutrition, excessive reliance on ORS, and physiological stress from recent trauma likely contributed to Vitamin K deficiency. Excessive ORS use can also cause osmotic diarrhea due to transient sugar malabsorption and slight hypertonicity [7]. While PT is commonly used to assess

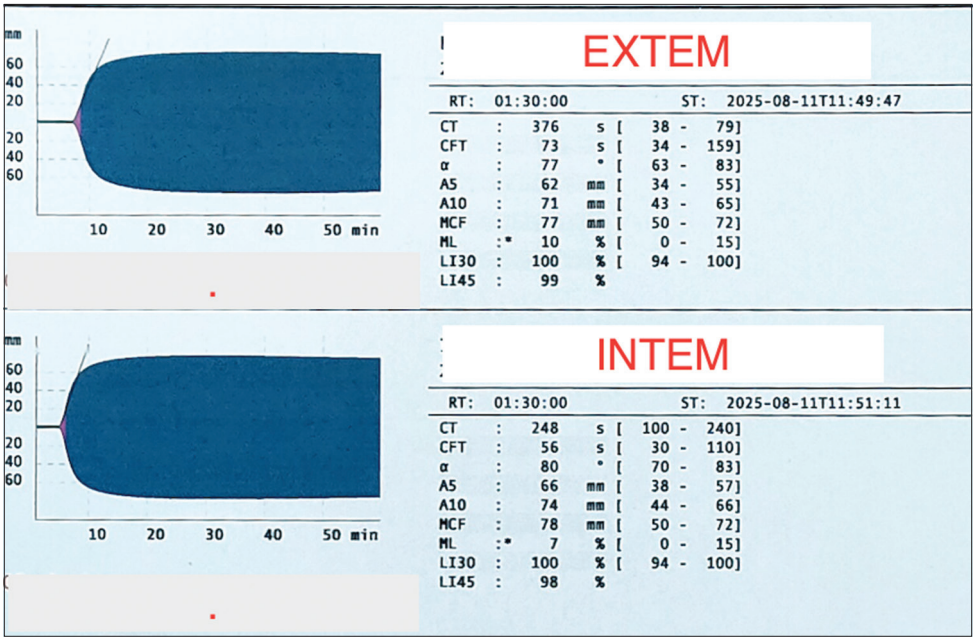

Figure 1: Rotational thromboelastometry tracing shows significant prolongation of clotting time, with EXTEM displaying a more pronounced delay than INTEM

Vitamin K status, it is relatively non-specific and may remain normal until prothrombin levels fall by 50%. Proteins induced by Vitamin K absence or antagonist-II (PIVKA-II), also known as des-γ-carboxy prothrombin (DCP), offer a more sensitive and specific measure of Vitamin K deficiency and are less affected by age-related variations [8,9]. In this patient, markedly prolonged PT and aPTT that normalized with Vitamin K confirmed the diagnosis [1,2].

A limitation of this report is the absence of PIVKA-II/DCP testing, which would have provided a more specific marker of Vitamin K deficiency. In addition, detailed dietary and antibiotic history before presentation was not available [4].

CONCLUSION

This case demonstrates that marked prolongation of PT and aPTT, with normalization after Vitamin K supplementation, is diagnostic for Vitamin K deficiency. Early recognition and correction were crucial for safe surgery and bleeding control. Clinicians should consider Vitamin K deficiency in trauma or malnourished patients with unexplained coagulopathy.

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