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OLD SCARS, NEW BLEEDS: REVISITING COAGULOPATHY IN QUIESCENT CROHN'S DISEASE

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ABSTRACT

Background: Vitamin K serves as a critical coenzyme in the γ-carboxylation of clotting factors II, VII, IX, and X, underscoring its essential role in the coagulation cascade. Deficiency of this fat-soluble vitamin is frequently observed in individuals with chronic gastrointestinal disorders, particularly those affecting nutrient absorption. If left untreated, long-term vitamin K deficiency can lead to serious complications, including an increased risk of bleeding, intracranial haemorrhage, and impaired bone mineralization. Given the potential severity of these outcomes, prompt recognition and treatment are imperative.

Case report: An 87-year-old male with a history of Crohn's disease and bowel resection 30 years ago, in sustained remission, presented with cutaneous purpura and elevated prothrombin time (PT) and activated partial thromboplastin time (aPTT). Laboratory evaluation revealed low serum vitamin K levels and decreased activity of vitamin K-dependent coagulation factors, which were corrected with vitamin K supplementation.

Conclusion: This case highlights that acquired vitamin K deficiency should be considered a potential cause of coagulopathy, even in patients with quiescent Crohn's disease and no active gastrointestinal symptoms. Our patient developed clinically significant bleeding decades after bowel resection, emphasizing that long-term nutritional consequences of gastrointestinal surgery may persist silently. Vigilance for vitamin K and other micronutrient deficiencies is essential in this population. Early recognition and supplementation can reverse coagulopathy, prevent haemorrhagic complications, and improve outcomes through regular monitoring and timely intervention.

KEYWORDS

Crohn's disease remission, vitamin K deficiency, coagulopathy, nutritional malabsorption, inflammatory bowel disease

LEARNING POINTS

- An elderly patient with quiescent Crohn's disease and prior bowel resection developed severe vitamin K deficiency–related coagulopathy three decades after remission.
- Selective depression of vitamin K–dependent clotting factors (II, VII, X) with normal factor V and elevated factor VIII was identified, and coagulopathy resolved rapidly with vitamin K supplementation.
- This case underscores the importance of long-term monitoring for fat-soluble vitamin deficiencies in patients with inflammatory bowel disease with a surgical history, even in prolonged remission.

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INTRODUCTION

Vitamin K is a fat-soluble vitamin essential for the activation of several coagulation factors, including factors II, VII, IX, and X, as well as for the regulation of anticoagulant proteins C and S through γ-carboxylation[1]. Deficiency may arise from inadequate dietary intake of vitamin K-rich foods, disorders affecting fat absorption—such as celiac disease and inflammatory bowel disease (IBD), prolonged use of antibiotics that disrupt intestinal microbiota responsible for endogenous vitamin K synthesis, or medications like anticoagulants that impair vitamin K metabolism[2]. Clinically, vitamin K deficiency often presents with spontaneous bleeding, cutaneous purpura, and haemorrhagic events involving the gastrointestinal tract or intracranial space. A high index of suspicion is warranted in the presence of such symptoms along with prolonged prothrombin time (PT) and/or activated partial thromboplastin time (aPTT). Diagnosis is confirmed by detecting reduced serum vitamin K levels, which are typically reversible with supplementation. The estimated prevalence of vitamin K deficiency among individuals with IBD ranges from 43.7 to 50%[3]. Here, we report the case of an 87-year-old male with a history of Crohn's disease in remission for 30 years, who presented with vitamin K deficiency.

CASE DESCRIPTION

An 87-year-old male with a history of Crohn's disease, status post bowel resection 30 years ago and in sustained remission, presented to the emergency department with spontaneous bruising of the bilateral lower extremities. The bruising developed following 5–6 hours of physical exertion while repeatedly climbing a ladder to repair garage lighting. He denied any history of falls, direct trauma, or overt bleeding. There was no personal or family history of bleeding disorders, including epistaxis, hematuria, gastrointestinal bleeding, hemarthrosis, or excessive bleeding following surgery or dental procedures. The patient also denied use of anticoagulants, antiplatelet agents, herbal supplements, antibiotics, alcohol, or any dietary restrictions.

On arrival, his vital signs were as follows: heart rate 72 bpm, respiratory rate 18 breaths per minute, blood pressure 142/63 mmHg, and afebrile 36.6°C. Physical examination revealed ecchymosis over the dorsal aspects of both feet, as well as a large ecchymotic area measuring 12 × 8 cm on the medial aspect of the right thigh. Initial laboratory results are shown in *Table 1*. Coagulation studies repeated over the next 48 hours showed a rapid correction after administration of vitamin K *(Table 2)*. Results of subsequent coagulation factor assays are shown in *Table 3*.

The patient's complete blood count and comprehensive metabolic panel were unremarkable. However, due to elevated PT and aPTT in the setting of superficial bleeding, coagulopathy was suspected. Hematology was consulted, and an extensive workup was initiated, including assessment of vitamin K levels, coagulation factor levels, mixing studies, and screening for nutritional deficiencies.

Laboratory test	Results	Reference range
Hb (g/dl)	9.8	14-18
WBC count (×10³/μl)	4.5	4.5-11
Platelets (×10³/μl)	166	150-400
PT (sec)	109.9	10.2-12.9
INR	>10.0	0.87-1.14
APTT (sec)	80	25-37
Thrombin time (sec)	16.1	10.3-16
D-dimer (ng/ml)	432	<500
Fibrinogen (mg/dl)	425	200-393

Abbreviations: Hb, hemoglobin; WBC, white blood cell; PT, prothrombin time; INR, international normalized ratio; APTT, activated partial thromboplastin time.

Table 1. Labs before vitamin K administration.

Laboratory test	Results
PT (sec)	15.8
INR	1.42
APTT (sec)	39

Abbreviations: PT, prothrombin time; INR, international normalized ratio; APTT, activated partial thromboplastin time.

Table 2. Coagulation studies after administration of Vitamin K.

Factor	Result	Reference range
Factor II (%)	32	70–150
Factor VII (%)	5	60–150
Factor X (%)	3	70–150
Factor V (%)	113	65-150
Factor VIII (%)	212.8	50-150
Factor IX (%)	84.5	65-150
Factor XI (%)	172	65-150
Factor XII (%)	134	50-150
Vitamin K (pg/ml)	<50	130-1500
Vitamin B12 (pg/ml)	159	239-931

Table 3. Results of subsequent coagulation factor assays.

The mixing study corrected both PT and aPTT, suggesting a deficiency of coagulation factors rather than the presence of an inhibitor. The patient received a single oral dose of vitamin K (2.5 mg). Repeat coagulation studies over the following 48 hours showed rapid normalization. Further hematologic evaluation revealed low vitamin K levels along with reduced

levels of factors II, VII, and X. He was also found to have low levels of vitamin B 12. His bruising and ecchymosis remained stable, with no further episodes of active bleeding. The patient was subsequently discharged with instructions to follow up with outpatient Hematology. Outpatient follow-up revealed elevated PT/aPTT (now asymptomatic), due to which he was placed on long-term maintenance Vitamin K supplementation.

DISCUSSION

This case highlights a rare yet clinically significant presentation of acquired vitamin K deficiency in an elderly patient with a long-standing history of quiescent Crohn's disease and prior bowel resection. Despite being in sustained remission for over three decades, lacking any overt gastrointestinal symptoms, the patient developed coagulopathy, evidenced by markedly prolonged PT and aPTT, and spontaneous cutaneous bruising. The underlying aetiology was traced to a profound deficiency in vitamin K, as confirmed by undetectable serum levels and selective depression of vitamin K-dependent coagulation factors (II, VII, and X), with preserved levels of other factors such as V and VIII.

While Crohn's disease is classically associated with active inflammation and symptomatic nutrient malabsorption, this case demonstrates that patients with a remote history of small bowel resection may continue to experience subclinical malabsorption. The ileum and proximal colon are significant sites for bile acid reabsorption, which is essential for the emulsification and absorption of fat-soluble vitamins, including vitamin K[4]. Surgical resection of these segments can compromise this process indefinitely, predisposing patients to latent deficiencies even in the absence of active disease[5].

Vitamin K deficiency should be suspected in patients presenting with unexplained coagulopathy, especially in the setting of malabsorptive conditions, prior gastrointestinal surgery, or broad-spectrum antibiotic use. In our case, the patient had no recent antibiotic exposure, dietary restrictions, or medication use that could interfere with vitamin K metabolism (e.g., warfarin, cephalosporins). The absence of inhibitors and rapid correction of PT/aPTT following oral vitamin K supported a diagnosis of simple deficiency rather than a consumptive or autoimmune process.

Additionally, the concurrent vitamin B12 deficiency further supports the hypothesis of chronic subclinical malabsorption. While pernicious anaemia was ruled out, the combination of deficiencies underscores the importance of nutritional surveillance in patients with a history of gastrointestinal surgery, regardless of symptom burden. This case also demonstrates the utility of factor assays in differentiating between coagulopathy due to vitamin K deficiency and other causes, such as liver disease or disseminated intravascular coagulation, particularly when liver function tests are normal, and fibrinogen levels are preserved or elevated.

Although rare, acquired vitamin K deficiency in Crohn's disease—especially in remission—is a clinically relevant phenomenon[6]. Without prompt recognition, it may lead to life-threatening haemorrhagic complications, particularly in vulnerable populations such as the elderly. Fortunately, the coagulopathy is rapidly reversible with vitamin K supplementation, and early intervention can prevent unnecessary invasive testing or empiric transfusion.

This case underscores the importance of considering acquired vitamin K deficiency as a potential cause of coagulopathy, even in patients with quiescent Crohn's disease and no recent gastrointestinal symptoms. Despite long-standing remission, our patient developed a clinically significant bleeding tendency due to subclinical fat-soluble vitamin malabsorption—likely secondary to prior bowel resection. This highlights the long-term nutritional consequences of gastrointestinal surgery, which may persist silently for decades. Clinicians should maintain vigilance for vitamin K and other micronutrient deficiencies in individuals with a history of IBD and surgical interventions, irrespective of current disease activity. Early identification and treatment with vitamin supplementation can reverse coagulopathy and prevent serious haemorrhagic complications. Regular monitoring of nutritional markers in this high-risk population may aid in timely diagnosis and improve patient outcomes.

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