Warfarin-Induced Skin Necrosis Following Routine Endoscopy in a Patient With Cirrhosis

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PRESENTATION

67-year-old male with ulcerative colitis, cirrhosis secondary to primary biliary cholangitis, and prior pulmonary embolism on warfarin

Underwent surveillance upper endoscopy and colonoscopy

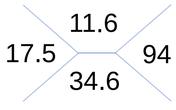
Warfarin was held 5 days prior to the procedures and resumed immediately thereafter

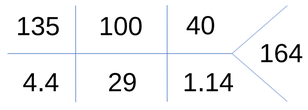
Two days later, he developed severe tenderness and bruising involving the skin of his abdomen, flank, and outer thighs

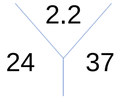
After no improvement with gabapentin, he ultimately returned to the emergency department for further evaluation

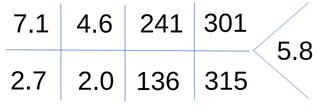
INITIAL WORKUP

Patient vitals Temp 37, HR 78, RR 20, BP 96/73, SpO2 100% on RA









Given vancomycin, ceftriaxone, 2 L NS and 60 mg IV furosemide prior to admission

EXAMINATION

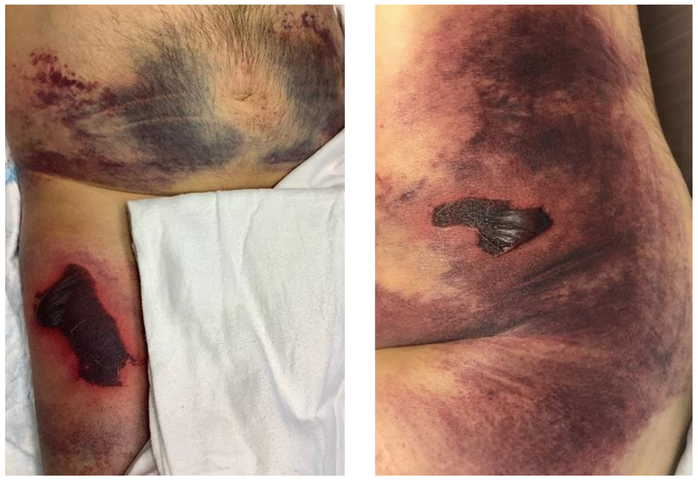


Figure 1: Images of the patient's abdomen and right thigh (left) and left hip (right). The skin is extensively involved with painful retiform purpura and areas of superficial necrosis with associated hemorrhagic bullae.

EVALUATION

Differential Diagnosis	Supportive Features	Inconsistent features
Sepsis	Hypotension, altered mentation, acute onset, leukocytosis	Afebrile, no improvement with antibiotics
DIC	Elevated d-dimer, low fibrinogen, prolonged PT/PTT	No clinical bleeding, no schistocytes on smear
TTP	Thrombocytopenia, AMS, rash	Normal haptoglobin/ADAMTS13
Vibrio vulnificus	Cirrhosis, severe rash	No exposure, no septic shock
Calciphylaxis	Widespread necrotic lesions	Acute onset, no ESRD
Vasculitis	Positive ANA, positive p-ANCA, CRP elevated, necrosis	Abrupt onset, no characteristic pattern of distribution
Cryoglobulinemia	Rash, Hx autoimmunity	Cryoglobulins negative

DIAGNOSIS

Confirmed protein C activity was low at 22% (normal 70-150%)

With characteristic appearance, temporal association with warfarin resumption, and absence of alternative cause, WISN was diagnosed without need for biopsy

Genetic testing for inherited protein C deficiency was negative, suggesting acquired deficiency likely due to liver disease

MANAGEMENT

Warfarin was discontinued, heparin was initiated, and protein C was repleted via administration of fresh frozen plasma

He initially improved and ultimately resumed warfarin on discharge after bridging with heparin

Unfortunately, his necrotic rash recurred, and he ultimately underwent excisional biopsy which confirmed WISN

Warfarin was discontinued thereafter in favor of lifelong enoxaparin

DISCUSSION

Warfarin skin necrosis is a rare but devastating complication

Most cases occur in patients with pre-existing inherited protein C deficiency, but deficiency can be acquired

Despite our patient's risk factors (liver disease and active UC) and characteristic presentation, diagnosis of WISN was delayed

Prompt recognition and treatment (discontinuation of warfarin, IV heparin, vitamin K and FFP) is crucial for tissue preservation and avoidance of morbid surgical intervention