

Clinical Image

Iatrogenic Cutaneous Necrosis

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Clinical Image

43-year-old man, with no significant history, admitted to the internal medicine department for assessment of popliteal ilio femoro deep vein thrombosis of the left lower limb, no triggering factor was identified. The treatment of this thrombosis was based on enoxaparin and AVK overlap. Heparin was stopped before an INR in the therapeutic range. On Day 5 of the initiation of the AVK, the patient reports a notion of paresthesia with the appearance of very extensive bullous skin necrosis in the thigh and leg associated with vascular purpura (Figure 1). Imaging showed no tumor syndrome, immunological assessment was negative. The thrombophilia assessment finds a protein S deficiency. Healing was obtained by withdrawal from AVK and relayed by heparin at a curative dose (Figure 2).

Described for the first time in 1943 [1], cutaneous necroses with vitamin K antagonist's rare cutaneous complications and typically occur in patients with strong coagulation within 3 to 10 days of the initiation of treatment, they preferentially affect areas rich in adipocytes (breasts, thighs, buttocks, abdomen) [1]. Cutaneous necrosis induced by AVKs is linked to an imbalance between procoagulant and anticoagulant factors. It occurs in patients with congenital or acquired thrombophilia leading to an unbalanced hypercoagulable state. Most commonly, thrombophilia has been associated with the deficiency of protein C. It can also be linked to antithrombin III deficiency, protein C resistance, antiphospholipid syndrome or a protein S deficiency [2]. In our case, the patient, presented with deep vein thrombosis due to AVK, has been diagnosed with a protein S deficiency. The combined action of AVK therapy and protein S deficiency explain the atypical case of cutaneous ulcerations and necroses. The treatment was based on the immediate cessation of AVK, the administration of vitamin K and curative anticoagulation by heparin [3].

The recognition of the diagnosis is an emergency because the necrosis evolves very quickly by involving the vital prognosis with a mortality of 15% at 3 months [4].

References

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Figure 1: A) Bullous skin necrosis and B) Vascular purpura.



Figure 2: Healing of skin lesions.

Citation: Zaizaa M, Bahadi N, El Bougrini Z, Jamal O, Sahel N, Talamoussa B, et al. Iatrogenic Cutaneous Necrosis. *Ann Med Case Rep*. 2024;6(1):1045.

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Publisher Name: Medtext Publications LLC

Manuscript compiled: Jan 22nd, 2024

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