

Disordini emorragici acquisiti: aspetti diagnostici e clinici

UNEXPLAINED BLEEDING IN A YOUNG WOMAN: A CASE REPORT OF SUPERWARFARIN TOXICITY

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Background. Superwarfarins are long-acting anticoagulant rodenticides, that share the same mechanisms of action as warfarin. They inhibit the vitamin K epoxide reductase complex, preventing the γ-carboxylation of vitamin K–dependent coagulation factors (II, VII, IX, and X) as well as proteins C and S. This results in severe coagulopathy and potentially life-threatening hemorrhage, which may persist for months and often requires prolonged high-dose vitamin K1 therapy.

Case Report. A 49 years old woman came to our attention due to the onset of epistaxis originating from a varix in the right nasal cavity and painful spontaneous bruising associated with functional joint impairment, which had developed over several months in the absence of trauma. She had no personal or family history of bleeding disorders. Initially, laboratory findings showed a haemoglobin (Hb) level of 13 g/dL and a platelet (PLT) count of 220000/mm3. Coagulation tests revealed a markedly prolonged prothrombin time (PT) beyond measurable limits (INR 20), and activated partial thromboplastin time (aPTT) of 60 seconds. Than the patient was admitted to our ward and coagulation factor assays showed reduced levels of FVll 7.7%, FX 13.8%, FIX 23%. Mixing studies corrected, C Protein 38%, S Protein 8,7%. High-dose intravenous vitamin K was promptly initiated. After one week of treatment, partial improvement of coagulation parameters was observed, PT 10s, INR 7,1, aPTT 63s, ratio 2,11. The patient was referred to a tertiary care center for further evaluation because of an inadequate response to high-dose vitamin K therapy. Laboratory findings remained consistent with vitamin K deficiency: PT 69.9 s, INR 6.55, aPTT 69.5 s, ratio 2.79, AT 133%, FVll 155%, S protein 21%, C protein 28%. Vitamin K supplementation was confirmed and on suspicion of poisoning with an anti–vitamin K substance, a sample is sent for toxicological analysis. The anticoagulant poisoning panel ultimately revealed high levels of Brodifacoum, Difenacoum, and Bromadiolone. Vitamin K therapy was therefore confirmed at high dose (10 mg/die per os). After five months despite the absence of major bleeding, a mild cutaneous bruising persisted. Coagulation parameters normalized (PT 10.1 s, INR 0.92, aPTT 24.8 s) and vitamin K was discontinued.

Conclusions. This case highlights the importance of through social and toxicological investigation in patients presenting with unexplained coagulopathy suggestive of severe vitamin K defciency, particularly to identify possible exposure to long-acting anticoagulant rodenticides.