

Warfarin Induced Skin Necrosis - A Case Report

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Abstract: Warfarin induced skin necrosis (WISN) is an uncommon catastrophic complication of oral anticoagulant therapy. A 30 years old lady had a history of venographically proven puerperal deep vein thrombosis developed a “painful bruise” on her left leg, thigh. The skin at the center of the area was supposed to break after necrosis occurred over the following 48 hours. Warfarin was discontinued. This case was diagnosed as an adverse drug reaction of warfarin causing skin necrosis. INR level should be monitored before starting treatment with warfarin and if any risk factor is stated alternative therapy for anticoagulation should be given.

Keywords: Warfarin, Skin necrosis, Bruise

1. Introduction

Warfarin is a widely used anticoagulant (blood thinner) used for treatment and prevention of thrombosis and thromboembolism. Warfarin induced skin necrosis (WISN) is a rare but severe complication of oral anticoagulant therapy in which skin and subcutaneous tissue necrosis occurs [1], typically seen in middle aged obese person. Although WISN typically occurs within first few days of initiation of warfarin therapy, but rarely delayed occurrence noted. High initial doses increases the risk of development [2].

2. Case Report

A 30 years old lady had a history of venographically proven puerperal deep vein thrombosis following which she was given anticoagulant with warfarin 5 mg daily for about 6 month. After 10 days she attended skin OPD a “painful bruise” (picture - 1) on her left leg, thigh. On examination, reddish purpuric spots were seen with scaling of skin. The skin at the center of the area started to break after necrosis occurred over the following 48 hours. Warfarin was discontinued, platelet count and INR were done, given injection vitamin K intravenously. The required surgical debridement was scheduled after 4 weeks, if necrosis isn't healed.



Picture 1

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3. Discussion

Warfarin acts by inhibiting the vitamin K dependant gamma - carboxylation of coagulation factors II, VII, IX, X and anticoagulant protein C and protein S. Very rarely (in about 0.01 to 0.2 percent cases) the resultant initial procoagulant state due to protein C and protein S deficiency in the first few days of starting warfarin leads to thrombosis in dermal capillaries, leading to skin necrosis. Concentration of anticoagulant protein C falls more rapidly than other vitamin K dependant procoagulant factors, which have longer half lives. Due to inactivity of vitamin K dependant protein C and protein S, a paradoxical hypercoagulable state is created in which micro thrombi are developed causing occlusion of cutaneous small vessels [1].

4. Conclusion

This case was diagnosed as an adverse drug reaction of warfarin causing skin necrosis. INR level should be monitored before starting treatment with warfarin. If required, warfarin therapy can be initiated at low dose. Since treatment is generally supportive, prompt and prudent evaluation of suspicious skin lesions is necessary to prevent the serious sequelae such as necrosis or amputation of affected part.

References

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