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Case Report

Atypical case of Acenocoumarol induced skin necrosis: A rare case Report

[1]Dr Jude Rodrigues, [2]Dr. Samiksha Shyam Naik Talaulikar*, [3]Fatima Rodrigues, [4]Dr.Dattaprasad Samant

[1]Professor and Head, Department of General surgery, Goa Medical College Bambolim
[2]Junior resident, Department of General surgery,Goa Medical College Bambolim
[3]MBBS , Goa Medical College Bambolim
[4]Lecturer, Department of General surgery,Goa Medical College Bambolim
Corresponding author*

Abstract

Acenocoumarol induced skin necrosis is a rare complication. usually occurs after few days after initiation of treatment. As such Clinical diagnosis is difficult. Delayed onset skin necrosis itself is a rare complication. The role of detailed history, clinical examination and laboratory investigation is commendable in paving the way for appropriate diagnosis and for appropriate management.

Key words: Acenocoumarol ,Skin necrosis, occlusive Vasculopathy

Introduction

Acenocoumarol is a frequently used oral anticoagulant that is approved by the US Food and Drug Administration (FDA) for the treatment and prevention of various medical conditions. Acenocoumarol inhibits the activation of vitamin K–dependent clotting factors II, VII, IX, and X and the anticoagulant proteins C and S.[1-3] Acenocoumarol induced skin necrosis is a rare (0.01%-0.1%) but serious complication that usually occurs within the first several days following its initiation, although case reports have described this phenomenon later in therapy as well.[2-5]

Case report

A 47 years old female presented to emergency department with 15 days history of pain in gluteal region after examination diagnosis was made as gluteal necrotic patches for which debridement was done. History of discontinuation of Acenocoumarol for 7 days prior to surgery .15 days later patient developed necrotic patches over abdomen in right iliac fossa. Following which patient developed redness and pain over left upper limb, both thighs and both legs followed by development of necrotic patches. soft tissue ultrasound was suggestive of diffuse edema with raised echogenicity in subcutaneous plane. History of fever during this process. patient is a known case of bilateral deep vein thrombosis since 11 years on tablet Acitrome 3 mg OD orally. She also complained of weakness of both upper and lower limb. She is also a known case of hypertension since 10 years on Tab Olmesartan 40mg once daily.

On investigations hb :8.4, Tc: 9450 PT:14.4,INR:1.4,ANA ,ANCAantiphospholipid antibody panel, factor V Leiden mutation was negative. CRP was Positive. Renal and Liver function tests were normal. Procalcitonin level was increased. Blood culture was sterile .skin biopsy was done suggestive of occlusive vasculopathy(Non

inflammatory purpura),protein S levels were decreased whereas protein C levels were normal. Bilateral upper and lower limb DVT doppler was normal.

On admission patient was started on intravenous antibiotics according to culture sensitivity, acitrome was stopped immediately, FFPs ,inj vitamin K 10 mg intravenously were given to the patient .Rivaroxaban 15 mg twice daily for 21 days followed by 20 mg once daily was started for the patient.

Excision of Necrotic patches was done over abdomen and gluteal region for which daily dressing were done, Vac therapy was applied over wounds. Supportive measures including limb physiotherapy, nutritional support was given to the patient. With treatment patients spikes gradually decreased over a period of 1 month and ulcers are in healing stage.

Discussion

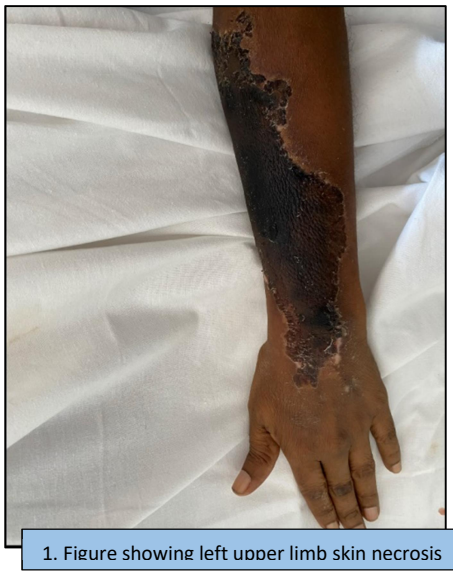
Acenocoumarol -induced skin necrosis, although rare (<0.1%), is a known complication.[4,5,2] Necrosis occurs more often in females and can occur in the limbs and adipose tissue, including the breast, buttocks, and penis, with an onset usually within several days of initiation of therapy[4-7] Usually including the breast, buttocks, and penis.[4-7] . in addition to the inhibition of the coagulation factors II, VII, IX, and X, Acenocoumarol also inhibits the anticoagulant proteins C and S.[7] This can predispose the patient to thrombosis and is worse in patients who already have protein C deficiency. Other less common predisposing factors include Protein S deficiency, Factor V Leiden mutation, Antithrombin III deficiency, Antiphospholipid antibody syndrome, Anticardiolipin antibody and Lupus anticoagulant.[1,8] Several reported cases have however occurred in patients without any of these deficiencies.[9,10,11]

Typically, there are multiple microthrombi in the capillaries and venules of the dermis and subcutaneous tissue which results in ischaemic necrosis of the skin.[11,12]

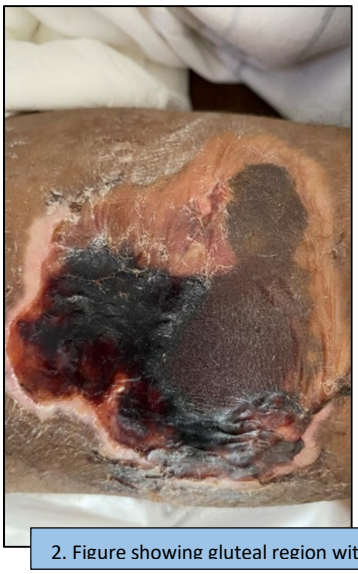
The mainstay of management is supportive. Treatment is aimed at withdrawing Acenocoumarol and reversing its effect on protein C with vitamin K and fresh frozen plasma. Heparin therapy is utilised to prevent further thrombosis in the postcapillary venules, and protein C concentrate is effective although high cost has limited its use. Patients may require local debridement and occasionally skin grafting or even amputation.[2] The main causes of death are deep tissue necrosis, sepsis syndrome secondary to wound infection and multi-organ failure.[13]In this case the diagnosis was delayed as there was delayed onset of symptoms which occurred after 11 years. fever occurred secondary to infected necrotic patches after debridement. Ultimately diagnosis was made after detailed history, examination, multiple laboratory tests and biopsy.

Conclusion

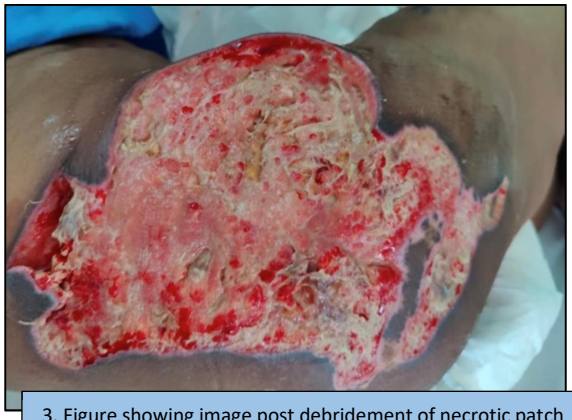
Skin necrosis due to Acenocoumarol is usually a rare complication in this case. It was seen to occur after many years following Acenocoumarol drug usage. The diagnosis is often confused with those of other skin conditions. It is crucial to take adequate history and physical examination also adequate laboratory studies helps us in early detection for adequate treatment and to avoid complications. This case should increase awareness that Acenocoumarol induced skin necrosis can affect patients who are restarting Acenocoumarol, despite of history of chronic Acenocoumarol use without any complication. There should be high level of suspicion for this rare yet potentially fatal reaction to Acenocoumarol.



1. Figure showing left upper limb skin necrosis



2. Figure showing gluteal region with necrotic patch



3. Figure showing image post debridement of necrotic patch

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