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Case Report

IgA vasculitis induced by acenocoumarol[☆]

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ABSTRACT

We present the case of a 73-year-old man with IgA vasculitis after administration of acenocoumarol, confirmed by anatomopathological study. He had cutaneous, joint and renal involvement. With the reintroduction of the drug, the clinical manifestations worsened. They were completely resolved with its suspension, without additional maintenance treatment.

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Vasculitis IgA inducida por acenocoumarol

RESUMEN

Se presenta el caso de un varón de 73 años con vasculitis IgA tras la administración de acenocoumarol, confirmada mediante estudio anatomopatológico. El cuadro cursó con afectación cutánea, articular y renal. La clínica fue más florida con la reintroducción del fármaco y se resolvió por completo al suspenderlo, sin tratamiento de mantenimiento adicional.

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Introduction

Acenocoumarol is an anticoagulant agent derived from coumarine and widely used in the prevention and treatment of thromboembolic processes. Vasculitis induced by acenocoumarol is an adverse effect which has rarely been described in the literature.

Clinical observation

A 73-year-old man with auricular fibrillation presented with palpable purpura in lower limbs associated with arthritis of the knees 20 days after initiation of treatment with acenocoumarol,

and was therefore admitted to hospital. During his stay the acenocoumarol was replaced by heparin. Lab tests revealed a raised CRP level with normal serologies and immunology (rheumatoid factor, complement, ANA, ANCA, cryoglobulins) and haematuria. Synovial fluid of the knee with normal characteristics was obtained through arthrocentesis. Skin lesion biopsies were carried out with diagnosis of leukocytoclastic vasculitis with negative direct immunofluorescence (DIF). The medical symptoms were related to previous respiratory infection. Treatment with prednisone at a 5 mg dose per day was prescribed. The patient's condition improved and he was discharged from hospital with the reintroduction of acenocoumarol.

Two days after reintroduction, he again presented with new outbreaks of skin lesions, this time with the formation of haemorrhagic blisters (Fig. 1). Analysis and biopsies were repeated. Proteinuria associated with haematuria was observed with normal kidney function and the dematopathological analysis again showed leukocytoclastic vasculitis, this time with positive DIF for IgA and C3 in vascular walls. Due to all of the above IgA vasculitis induced by

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Fig. 1. Clinical image of the patient who presented with palpable purpura in lower limbs with formation of haemorrhagic blisters.

acenocumarol was suspected. The acenocumarol was discontinued and treatment with apixaban was begun. Three 125 mg intravenous shots of methylprednisolone was prescribed with a posterior dose of 15 mg/day prednisolone in descending order and discontinuation of the corticosteroid treatment within a period of 2 months.

Since the discontinuation with acenocumarol six months ago, the patient has not presented with any skin lesions and urinary lab test changes have been completely reversed.

Discussion

The adverse cutaneous effects of acenocumarol are equimosis, cholesterol embolism, skin necrosis, maculopapular eruptions and photosensitivity. Vasculitis is an adverse effect which has little documentation in the literature.¹ We present a case of IgA induced vasculitis (Schönlein–Henoch disease) secondary to acenocumarol. Causal diagnosis was based on the clear temporal relationship of clinical manifestations with the administration of the drug and its reintroduction, leading to its discontinuation.²

IgA vasculitis is caused by the deposition of immune complexes. Participation from different inductor antigens has been suggested, such as those from the upper respiratory airways and from some drugs. Clinical symptoms consist of palpable purpura,

polyarthralgias, mild glomerulonephritis with proteinuria, microscopic haematuria (as in the case we present) and abdominal pain. In adults initial discomfort in the intestines is less common.³

Diagnosis of IgA vasculitis is based on clinical signs and symptoms. Histopathological study of the skin is useful for confirming leukocytoclastic vasculitis with IgA deposit and C3 through immunofluorescence. Renal biopsy is rarely necessary for the diagnosis.³

To date 5 cases of vasculitis induced by acenocumarol have been reported in the literature^{1,4–7} and of them all, only one case is an IgA vasculitis. In these cases clinical symptoms present between 2 and 20 days after treatment initiation and the symptoms are resolved following suspension.

Conclusions

We present the second case of IgA vasculitis associated with acenocumarol. We should be aware of this adverse effect and recommend drug suspension when the temporal relationship between administration and initiation of medical symptoms are suggestive of this condition.

Conflict of interests

The authors have no conflict of interests to declare.

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