ISSN: 2456-6659
International Journal of Advances in Medical Sciences
(A Monthly Scientific Journal of Kiban Research Publications)
www.kibanresearchpublications.com

CASE STUDY

# Purpura Fulminans Secondary to Rickettsial Infection

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**Abstract:** A 60 year old male presenting with purpuric lesions over both upper and lower limbs and consumption coagulopathy following rickettsial infection. It was diagnosed as purpura fulminans secondary to rickettsia infection with disseminated intravascular coagulation and treated with replacement of platelets and coagulation factors along with antibiotics and Doxycycline.

**Keywords**: *Rickettsial infection, Purpura fulminans, Coagulopathy.*

**Article Received: 30 Nov. 2018** **Revised: 15 Dec. 2018** **Accepted: 25 Dec. 2018**

## Introduction

Purpura fulminans is an acute life threatening disorder characterized by cutaneous haemorrhagic manifestations and necrosis caused by disseminated intravascular coagulation and dermal vascular thrombosis. It was first described by Guelliot in 1884 in a patient with Varicella zoster. Since then, it has been shown to be associated mainly with bacterial mainly meningococcal and viral infections [1]. The occurrence of antiphospholipid antibodies, disseminated intravascular coagulation and antibodies to protein C and S is thought to play a role in its pathogenesis [2, 3].

## Case Report

A 60 year old male presented to the emergency room with fever of eight days duration associated with swelling of both lower limbs. On examination he had multiple purpuric patches over the upper and lower extremities as shown in Fig.1. Vitals were normal and bilateral inguinal lymphadenopathy was present.

The patient was shifted to intensive care unit and started on inotropic support with antibiotics and supportive treatment. The patient developed acute onset breathlessness the next day of admission after he tried to get down from the bed against the advice of the staff. The patient developed altered sensorium on the fifth day of admission.

After his arterial blood gases showed metabolic acidosis correction with soda bicarbonate was given.

His haemogram showed leukocytosis with predominance of neutrophils and thrombocytopenia. Blood culture was sent before the administration of antibiotics followed by serial cultures every 8 hours which were sterile. Dengue duo test for NS1 antigen and dengue IgM and IgG antibodies were negative. Weil Felix test showed a titer of 1:160 for OX2. The patient was started on Doxycycline and platelet infusion was given.

His D-dimer was raised with positive troponin T, echocardiography showed akinesia of the anterior septal and apical regions with distal free wall hypokinesia and an ejection fraction of 32 %. The patient was started on fibrinolytic therapy with a bolus dose of 250000 units of streptokinase followed by an infusion of 50000 units every hour for 24 hours. The patient developed blebs over the skin lesions on the seventh day with no improvement in symptoms.

The patient was started on Vancomycin with continued Doxycycline for the next three days. The patient showed improvement in sensorium after ten days with gradual fading of the skin lesions. His coagulation profile was normal on the 15th day after which he shifted out of intensive care.

He was discharged after 20 days of admission and was keeping good health on follow up after a month.

## Discussion

Purpura fulminans is an acute often fatal thrombotic disorder which manifests as purpuric lesions over the body with extensive bruising and discoloration of the skin. It is also associated with disseminated intravascular coagulation. The two most common causes are meningococcal and varicella. Gram-negative bacilli and staphylococci have also been reported. Leptospira, rickettsia and other viral conditions are known to rarely cause purpura fulminans [4]. The three forms of this disease are classified by the triggering mechanisms.

- Neonatal purpura fulminans
- Idiopathic purpura fulminans and
- Acute infectious purpura fulminans.

In inflammatory conditions it is the activation of complement and coagulation pathways by the endotoxins and signaling by inflammatory cytokines or endothelial dysfunction and vasculitis caused by the offending organisms itself. Purpura fulminans presents with circumscribed ecchymotic lesions and symmetrical gangrene of the extremities with consumption coagulopathy regardless of the causative agent [5].

The initial appearance of purpura fulminans lesions is of well-demarcated erythematous macules that progress rapidly to develop irregular central area of blue-black haemorrhagic necrosis. Advancing areas of central necrosis are typically surrounded by a thin border of erythema that fades into adjacent uninvolved skin.

Haemorrhage into the necrotic dermis causes purpura fulminans lesions to become painful, dark and raised, sometimes with vesicle or bulla formation [6].

In our case the patient tested positive for rickettsia with high titers for OX2 antibodies. It is rare for rickettsial fever to present with purpura fulminans and the petechial lesions typically seen may be masked or totally absent. Hence it is important to keep a broad range of differential diagnosis and in cases where preceding history of fever is not clear causes other than bacterial should be sought and investigated.


**Fig.1 Purpuric lesion over lower extremity**

## Conclusion

Hence it is important to keep a broad range of differential diagnosis and in cases where preceding history of fever is not clear causes other than bacterial should be sought and investigated. Purpura fulminans is usually associated with gram negative organisms but rarely other infections like dengue, leptospira and rickettsia can also it. In patients presenting with purpuric lesions a thorough investigation for atypical organisms must be done.

## Acknowledgements

Author acknowledges the immense co-operation received by the patient and the help received from the scholars whose articles are cited and included in references of this manuscript. The authors are also grateful to authors / editors / publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed.

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