



Case Report- Acute Infectious Purpura Fulminans Due to Klebsiella Pneumoniae

Authors

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Abstract

Purpura fulminans (PF) is a rare but life threatening disorder of acute onset characterised by cutaneous haemorrhage and necrosis caused by disseminated intravascular coagulation and dermal vascular thrombosis.

Our case is a 19 yrs old female, with no previous co morbidities, high grade fever, loose stool, cough with non purulent mucoid expectoration not associated with hemoptysis and lower limb purpuric rashes.

Introduction

Purpura fulminans (PF) - characterised by acute onset of progressive cutaneous haemorrhage and necrosis due to dermal vascular thrombosis and disseminated intravascular coagulation.

Rare, potentially disabling and life threatening disorder

Predominantly in three clinical settings:

- (1) Inherited or acquired abnormalities of protein C or other coagulation systems.
- (2) Acute infectious PF.
- (3) Idiopathic.

Typically, acute infectious purpura fulminans occurs:

- Young children below 2 years of age.
- Usually in the setting of meningococcaemia due to elaboration of endotoxin.

Here, we report a case of acute infectious purpura fulminans with a few atypical features, such as presentation in adolescence, and the development of lesions during the course of sepsis due to Klebsiella pneumonia

Case Report

A 19 yrs old female residing in delhi, with complaints of high grade fever with loose stool multiple episode for 4 days.

This was associated with cough with non purulent mucoid expectorant without hemoptysis.

Followed by this patient started developing non palpable ecchymotic purpuric rashes over the lower limbs which increased in size and number over a period of 2 days.

There was no history of vomiting, pain abdomen, haematuria, epistaxis, gum bleeding, hematemesis or malena.

The patient was not a known case of any bleeding disorder. There was no history of photosensitivity, oral ulceration, or joint pains.

Examination

At the time of admission, the patient was conscious and fully oriented.

She was febrile with temperature of 103.6°F , her pulse rate was 96/min, and blood pressure was 90/60 mm Hg.

There was no pallor, icterus, or lymphadenopathy. Systemic examination of the patient was essentially normal.

Examination of the lower extremities revealed large areas of well circumscribed non palpable ecchymotic purpuric rashes over the ankles, planter and dorsal surface of both the feet.

On the day of admission



On the day of discharge



Investigation

Hb g/dl	TLC /mm3	DLC %	PLT /mm3	S.Na/S.K mg/dl	Ur/Cr mg/dl	S.Bil/D/InD mg/dl	OT/PT U/L	ALP U/L	Urine protein
9	14800	P83,L15,E1,M1	68000	139/ 2.4	30/0.3	2.9/1.9/1	124/42	147	Trace

Other investigations showed CRP positive, prolonged PT (control – 12.5 sec, patient – 13.4 sec), prolonged PTT (control – 32.5 sec, patient – 41.1), INR (patient 1.22).

Sputum cultures revealed a positive growth for Klebsiella pneumoniae sensitive to meropenam and netilmicin.

Blood sugar, ECG & X-ray chest were normal.

Blood culture was negative and doppler lower limb was normal.

The results of NS1Ag, Dengue serology (IgM, IgG), IgM chikungunya, malarial parasite, rheumatoid factor, ANCA, ANA, HIV, HBsAg, Anti HCV antibody, were negative.

Management

Patient was put on treatment with IV fluids, broad spectrum antibiotic- ceftriaxone.

Latter on culture based antibiotics - meropenam and netilmicin.

She was also given platelet transfusion.

Discussion

Purpura fulminans (PF) is a very severe but rare acute thrombohaemorrhagic illness of infants and young children.^[1]

It occurs in three clinical settings:

i) In the neonatal period, as a manifestation of inherited, homozygous protein C, or, rarely protein S deficiency.

ii) Idiopathic PF, occurs approximately 7-10 days after a relatively benign antecedent infection usually of the skin, such as varicella, scarlet fever, rubella, measles, streptococcal tonsillopharyngitis, etc.

iii) Acute infectious purpura fulminans, occurs in conjunction with acute infectious illness, particularly sepsis with endotoxin (lipopolysaccharide) producing gram-negative bacteria.^[2]

Sepsis-induced purpura fulminans is a rare but life-threatening disorder.

Meningococcal sepsis is the most common cause. Other organisms which have been reported to cause acute infectious purpura fulminans are

Streptococcus pneumoniae, Haemophilus influenzae Type B, Streptococcus agalactiae (group B streptococcus), Rickettsia rickettsii, Streptococcus pyogenes (group A Streptococcus), Staphylococcus aureus, Klebsiella pneumoniae, E. coli, Proteus mirabilis, Enterobacter spp., Neisseria catarrhalis, Haemophilus aegypticus, Capnocytophaga canimorsus.

Approximately 60 to 70% cases of acute purpura fulminans have been reported amongst children below 2 years of age,^[3] while our patient was 19 years old.

Besides this, acute infectious purpura fulminans due to Klebsiella pneumoniae has been reported rarely.

PF involve dysfunction of haemostasis with a shift from a quiescent state favouring anticoagulation to a disease state of overwhelming procoagulation.^[1]

Lesions of PF are similar regardless of the precipitating condition.

Features of PPF include disseminated intravascular coagulopathy, symmetrical necrotic purpura and/or ecchymoses and symmetrical peripheral gangrene.^[4]

Digital and/ or limb amputations and end-organ failure may also occur.

Development of systemic consumptive coagulopathy is a defining feature of PF, which distinguishes it from other forms of skin necrosis.^[5]

Early antibiotic administration and intensive care management according to the recommendations of severe sepsis and shock is crucial for patient's survival.

Highlights the need for high index of suspicion of this clinical entity

Differentiating it from other condition such as collagen vascular diseases, TTP, and other conditions associated with peripheral gangrene.

References

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