**Clinical microbiological case: a necrotic skin lesion in a patient with renal failure**

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Please refer to the article on pages 538–539 of this issue to view the questions to which these answers refer.

**EVOLUTION**

The patient was transferred to the Intensive Care Unit (ICU) because of shock, respiratory failure and decreased consciousness level without focal signs.

The multiorgan failure persisted despite aggressive treatment (inotropic drugs, mechanical ventilation and broad-spectrum antibiotics: imipenem/cilastatin and vancomycin). *Escherichia coli* was recovered from blood cultures and *E. coli* and *Morganella morganii* were isolated from the fistula. Left-thigh skin biopsy showed microvascular thrombosis.

After 48 h in the ICU the patient died as a result of refractory multiorgan failure.

**DISCUSSION**

1. The differential diagnosis of the skin lesion presented by our patient should include thrombotic thrombocytopenic purpura and Schönlein–Henoch Purpura.

   The diagnosis of this patient was *Purpura fulminans* (PF) and multiorgan failure as a result of systemic infection which probably originated in the AVF caused by *E. coli* and *Morganella morganii*. Three categories are recognized:

   a) PF related to an acute infection (90%). The most common cause is *Neisseria meningitidis* [12], while *Streptococcus pyogenes* and *Enterobacteriaceae* occur less frequently [3]. PF has also been described after viral infections such as chickenpox.

   b) PF is secondary to a hereditary deficiency of protein C or protein S. This form usually appears in newborn babies and has a very high mortality.

   c) Idiopathic PF. This form is not very common and the lesions are limited to the skin and never appear in other organs.

2. Patients with PF usually have fever and may rapidly develop disseminated intravascular coagulopathy (DIC) and septic shock. The PF lesion is not different from any other purpuric skin process. However, it is rapidly progressive and has irregular central necrotic areas with a surrounding erythematous border and inflammatory process. The idiopathic variety is confined to the skin and generally affects both legs.

   The pathophysiological mechanism involves a procoagulant pathway and the anticoagulant endothelial cell activities. It may be induced by bacterial endotoxins and by the inflammatory cytokines which directly affect protein C and S [5,13,15]. The fibrinogen degradation products tend to increase, while the protein C, S levels, fibrinogen and the antithrombin III concentration decrease [6].

   3. *Purpura fulminans* has a high mortality (>90%), therefore an early diagnosis is of the utmost importance. The therapy is based in supportive measures including fluid replacement, inotropic drugs and, occasionally, mechanical ventilation support.

   Antibiotics are an essential tool and, initially, should offer broad-spectrum (Gram-negative and Gram-positive bacteria) coverage. Surgical evaluation may also be required with extensive resection of the necrotic areas. In extreme cases, major amputations may be needed. Other therapies to be considered include [3]:

   Heparin therapy; used to minimize the effects of distal thrombosis because it inhibits the vascular thrombosis and the consumption of coagulation factors. Idiopathic PF has a good response to heparin therapy [7].

   Protein C; it is a K-dependent vitamin protein with anticoagulant and anti-inflammatory properties [4]. The administration of fresh frozen plasma with high concentration of protein C is recommended mainly in cases with low serum concentrations [14,16].

   Recombinant tissue plasminogen activator (rtPA); it induces clot-specific fibrinolysis without any direct hemodynamic effect and with few bleeding complications [8].

   Topical nitroglycerin (TNG); the appearance of severe pain is characteristic of PF. This is secondary to prostaglandin release from poor tissue perfusion, with consequent sensitization of nerve fibers [10]. TNG is metabolized to nitric oxide which produces vasodilatation [10].

   Plasmapheresis; through plasmapheresis it is possible to remove bacterial endotoxins, cytokines and inflammatory mediators.

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REFERENCES