Purpura fulminans 10 years after contaminated cocaine use

Sylvia Martinez-Cabriales, Jorge Ocampo-Garza, Laura Barbosa-Moreno, Sonia Chavez-Alvarez, Jorge Ocampo-Candiani

A 43-year-old man with a history of cocaine use 10 years previously presented to the emergency room in March, 2014, with disseminated dermatosis, fever, and 1 day of dyspnoea. 6 months previously, he had had a spider bite in the earlobe with a subsequent eschar. Physical examination showed multiple plaques consisting of violaceous, coalescent, and non-evanescent macules with an erythematous border, on his ears, trunk, arms, and legs, some with flaccid blisters that increased in number and size over the next 24 h (figure). He had normal leucocytes and a platelet count of 103×10^9 cells per L (normal range $150–300\times10^9$ cells per L), decreasing to $65·8\times10^9$ cells per L in 24 h. His haemoglobin concentration decreased from 12 g/L (normal range 12·2–18·1 g/L) to 9·7 g/L within 72 h. Blood urea nitrogen, serum creatinine, erythrocyte sedimentation, partial thromboplastin time, and serum fibrinogen were all raised at 24 h; prothrombin time was normal. CT scan and abdominal ultrasound showed lobar consolidation of the right lung and hepatosplenomegaly. We made a diagnosis of community-acquired pneumonia and suspected that his rash was purpura fulminans and vasculopathy associated with use of levamisole-contaminated cocaine. We took a skin biopsy sample and immediate Gram staining showed Gram-positive cocci. Histological examination revealed thrombogenic vasculopathy without vasculitis. We started empirical treatment with vancomycin 500 mg twice-daily for 48 h. Blood cultures grew *Streptococcus pneumonia*, and once *Staphylococcus aureus* was excluded we changed the antibiotics to ceftriaxone 1 g twice-daily and azithromycin 500 mg once-daily. Over the next 14 days his haemodynamic signs and blood tests normalised and he had complete re-epithelialisation of the skin lesions over the next 16 weeks. We followed up the patient for 8 months to November, 2014, without relapse.

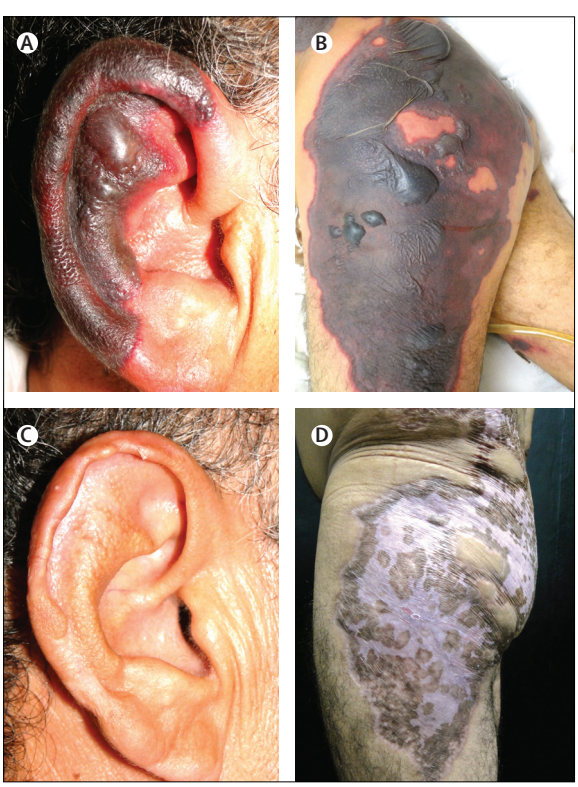

Figure: **Skin lesions associated with purpura fulminans**
Ear (A) and trunk (B) at initial presentation in March, 2014. Ear (C) and trunk (D) at 6 months follow-up in September, 2014, showing multiple areas of post-inflammatory hypopigmentation and hyperpigmentation.

Vasculopathy associated with levamisole-contaminated cocaine was our principle differential diagnosis owing to the history of cocaine use and the topography of the lesions. However, such vasculopathy has a latency period, with cutaneous necrosis occurring 5 years after persistent levamisole use. Furthermore, agranulocytosis was absent. Other differential diagnoses included thrombotic thrombocytopenic purpura, calciphylaxis, coumadin necrosis, necrotising fasciitis, and acute meningococcaemia. A spider bite can cause dermonecrosis of the affected area within 3 days without spreading to other areas, but this diagnosis was not consistent with our patient's clinical course.

Purpura fulminans was described in 1884 by Antoine-Octave Guelliot. The disease is rapidly progressive, with small vessel microthrombi and haemorrhagic skin infarction usually associated with disseminated intravascular coagulation. Cutaneous lesions begin as non-blanching, well-demarcated erythaematous macules that rapidly evolve into plaques with violaceous–black haemorrhagic necrosis and bullae formation. The most common cause is meningococcaemia, followed by pneumococcus infection, usually in asplenic patients. Purpura fulminans is a medical emergency that has high mortality (50%) secondary to multiple organ dysfunction, and long-term morbidity. Early diagnosis and aggressive antibiotic treatment must be aimed at the underlying infection, combined with supportive management and haemodynamic monitoring. Recognition of the pathology is essential because purpura fulminans often precedes the signs of shock, and rapid treatment can save the patient's life.

Contributors
All authors cared for the patient and contributed to research and writing of the report. Written consent to publication was obtained. We thank Sergio Lozano-Rodriguez for his help in editing the manuscript.

Lancet 2015; 386: e21

Published **Online**
May 29, 2015
http://dx.doi.org/10.1016/S0140-6736(15)60069-1

Department of Dermatology, University Hospital "Dr José Eleuterio González", Monterrey, Mexico
(S Martinez-Cabriales MD, J Ocampo-Garza MD, L Barbosa-Moreno MD, S Chavez-Alvarez MD, Prof J Ocampo-Candiani MD)

Correspondence to:
Prof Jorge Ocampo-Candiani, Department of Dermatology, University Hospital "Dr Jose Eleuterio González", Avenue Madero and Gonzalitos S/N 64460 Mitras Centro, Monterrey, Mexico
jocampo2000@yahoo.com.mx