Asplenic fulminant sepsis secondary to a dog bite complicated by toxic epidermal necrolysis/Stevens-Johnson syndrome

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Abstract
We report a case of asplenic fulminant sepsis in Australia following a dog bite which was complicated by toxic epidermal necrolysis/Stevens-Johnson syndrome (TENS/SJS). Capnocytophaga canimorsus, the infective organism, is a rare cause of sepsicaemia: a high degree of suspicion of this unusual organism and its early aggressive management is paramount. The diagnostic and management difficulties of TENS/SJS in the context of a patient with fulminant sepsis, DIC and on inotropes are also highlighted.

Case report
A 60-year-old woman, with a history of splenectomy secondary to idiopathic thrombocytopenic purpura, presented to the emergency department in an Australian country hospital in septic shock 3 days following a minor dog bite to her left calf.

After fluid resuscitation, administration of antibiotics (cephazolin 2g, IV, metronidazole 500mg, IV, gentamicin 5mg/kg, IV) and metaraminol (2.5mg), she was transferred to a regional Intensive Care Unit (ICU), where she developed multi-organ failure and disseminated intravascular coagulation (DIC). She was intubated, commenced on haemofiltration, flucloxacillin (IV, 2g, 6-hourly) and ceftriaxone (IV, 2g, 12 hourly).

A progressive purpura was noted over her upper and lower limbs and she had cold extremities. After 24 hours, the Infectious Diseases Department was consulted, with commencement of meropenam (IV, 1g, 8-hourly) and lincomycin (IV, 600mg, 8-hourly) given the suspicion of Capnocytophaga canimorsus (C. canimorsus) as the causative organism. Activated protein C and intravenous immunoglobulin (IVIG) were administered.

Over the next 48 hours, there was haemodynamic improvement and inotropic support was ceased. However, dry gangrene developed at the finger tips and toes with ischaemic demarcation present on both soles of her feet. The purpuric rash on her limbs became more generalised with formation of flaccid bullae and desquamation.

With the clinical presentation, morphology/Gram stain of punch biopsies taken from the bite wound, and blood cultures being positive with anaerobic Gram-negative bacilli—C. canimorsus was determined as the causative organism.
On day 6, the bite wound was debrided (Figure 1) and the patient was extubated. A transfer was organised to a tertiary referral centre on day 11.

**Figure 1. Debrided bite wound on left leg**

![Image of debrided bite wound on left leg](image1)

On arrival, the patient presented with ongoing asplenic sepsis and necrosis of her fingers, toes and distal lower extremities. There were widespread areas of erythematous to violaceous macules and patches with flaccid bullae and erosions over the upper and lower limbs as well as the peri-areolar areas with positive Nikolsky’s sign (Figures 2 and 3)—the total body surface area affected was 36%. Extensive mucositis and ulceration of the lips and oropharynx was noted with exposed hard palate. No other mucous membrane involvement was found.

**Figure 2. Right arm**

![Image of right arm](image2)

**Figure 3. Right leg**

![Image of right leg](image3)
Tracheostomy was performed and the wounds were cleaned and dressed with Jelonet™ (paraffin gauze) in theatre. The diagnosis of toxic epidermal necrolysis/Stevens-Johnson syndrome (TENS/SJS) (later confirmed by skin biopsy), on a background of asplenic fulminant sepsis, was made. TENS/SJS was managed with IVIG (2mg per kg in three divided doses).

In view of meropenem and lincomycin being commenced 2 days prior to rash onset, they were thought to be the potential causative agents. They were subsequently ceased as were all other non-essential medications. Moxifloxacin (IV, 400mg, daily) and vancomycin (IV, 1g, BD) were commenced instead.

Over the next week there was systemic improvement of the patient as well as the desquamated areas of skin and mucositis. There was demarcation of necrotic distal extremities and of areas of full thickness skin loss more proximally on the thighs and buttocks.

There was concern that the patient’s necrotic lower limbs posed an ongoing risk of further sepsis and so, lower limb amputations (right below knee and left above knee) were performed once the patient’s condition had been optimised.

Two days post amputations, the patient suffered severe neurological injury following a cardiorespiratory arrest. The cause was presumed to be ongoing sepsis and the decision to palliate the patient was made by the treating units and family. The patient died 1 day later.

Discussion

Whilst *C. canimorsus* is an unusual cause of septicaemia (estimated incidence of 0.5 cases/million per year)\(^1\), over 100 cases of human infections have been reported - predominantly in immunocompromised patients.\(^2\) It usually causes systemic infections (94%) and seldom localised infections (6%).\(^2\) Systemic infections range from mild to fulminating disease.

Rash (macular/maculopapular or purpuric) and gangrene are common. The organism is a commensal of saliva in dogs and cats, which can be transmitted to humans by bite (54%), scratch (8.5%) or mere exposure (27%).\(^3\) It is a slow-growing (2–7 days), fastidious Gram-negative bacterium which is difficult to isolate, and including a clinical history of suspicion for the organism is imperative to aid microbiological diagnosis.\(^3,4\) Despite being susceptible to a wide range of antibiotics (including beta lactams, tetracycline and clindamycin), the mortality rate is 30%.\(^2\) It is paramount that patients at high risk of *C. canimorsus* infection have prompt debridement and antimicrobial treatment.\(^5,6\) Immediate treatment may favourably influence the potentially fulminating course of systemic infection and patients with an increased susceptibility should be well-educated about this.

It is challenging to diagnose TENS/SJS in the context of fulminant sepsis and associated DIC in a patient requiring inotropic support. These conditions may all result in skin changes.
DIC causes a purpuric rash and high doses of inotropes (such as metaraminol) can cause distal extremity ischaemia and necrosis secondary to peripheral vasoconstriction. Mucositis and epidermal sloughing, however, are hallmarks of TENS/SJS (which were present in the reported patient) and should strongly suggest this diagnosis.

Another point in differentiating the skin changes of TENS/SJS and DIC is skin lesions in TENS/SJS tend to appear first on the trunk, spreading to the neck, face and proximal upper extremities—the distal portions of the upper and lower limbs are relatively spared.7,8

In comparison, although the purpura and petechiae in DIC can spread in a centripetal fashion or become generalised, they are usually confined to the extremities.9 TENS/SJS are severe idiosyncratic reactions most commonly triggered by medications and early identification and withdrawal of the offending agents improves prognosis significantly.10

This case emphasises the importance of a high degree of suspicion of this unusual organism (C. canimorsus) causing fulminant sepsis in asplenic and other immunocompromised patients, and the importance of its early aggressive management. In addition, it highlights the diagnostic challenge of TENS/SJS in the context of a patient with fulminant sepsis, DIC and on inotropes and the management difficulties that this poses.

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