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Fatal *Capnocytophaga canimorsus* (DF-2) septicaemia

A case report

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A 45-year-old man died 2 months after being bitten on the hand by a dog. He developed the rare but characteristic clinical picture of fulminant septicaemia and peripheral gangrene caused by a Gram-negative bacillus, *Capnocytophaga canimorsus,* previously known as dysgonic fermenter type 2 (DF-2), which is an occasional commensal in the oral flora of dogs and cats. This disease must be anticipated and dog bites appropriately managed to avoid the mortality associated with infection by this micro-organism. Initial treatment includes appropriate prophylactic antibiotics and debridement, while early exchange transfusion and emergency amputation may be of value in fulminant cases.

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Dysgonic fermenter type 2 (DF-2) was an earlier name given to a slow-growing Gram-negative bacillus transmitted by dog bite and producing fulminant septicaemia with marked peripheral ischaemia and a high mortality rate.[1] This organism has recently been renamed *Capnocytophaga canimorsus,*[2] and severe infection may be more common than previously reported. The disease, first described by Butler[3] in 1977, is characterised by the history of a dog bite followed by the clinical features of a malar rash, peripheral ecchymosis, symmetrical gangrene, disseminated intravascular coagulopathy and acute tubular necrosis with renal failure. The organism must be identified on a peripheral blood smear, since it is difficult to culture using routine culture methods.[4] This case highlights the potentially catastrophic result of an apparently insignificant dog bite and delayed medical intervention.

Case report

A 45-year-old previously healthy man, resident in Cape Town for only 1 month, was admitted to Groote Schuur Hospital in multiple organ failure. He had a history of heavy smoking and ethanol consumption for many years. One week before admission he had sustained a seemingly insignificant dog bite to the 5th digit and hypothenar eminence of his right hand. He had not sought medical attention for this injury and it was not the stated reason for his presentation to hospital. The puncture wounds on the hand were not obviously septic and no other septic focus was identified. He reported mild myalgia and fever for a few days, but his condition had deteriorated acutely during the 24 hours before admission. He had experienced profuse watery diarrhoea with approximately 15 bowel actions during the day before admission, associated with nausea, vomiting, abdominal discomfort, fever and dizziness. In addition he had noted numbness of the fingers and toes, a petechial rash on the legs, and haematuria.

On presentation the patient was ill, shocked, clinically dehydrated, deeply jaundiced, and had peripheral ischaemia. There was also evidence of recent spontaneous bleeding with petechial lesions on the forearms and thighs. He had obvious vascular compromise of the nose, fingers and toes, manifest by a well-demarcated bluish discoloration, hypothermia, a prolonged capillary filling time and decreased sensation with areas of hyperaesthesia. Most severely affected was the 5th digit of the right hand, with early gangrene. He had tachycardia and was hyperventilating in response to metabolic acidosis. Initial arterial blood gas analysis revealed pH 7,4, partial arterial oxygen tension (Pa_{O_2}) 20,4 kPa on a fractional inspired oxygen concentration (Fi_{O_2}) of 0,4, partial arterial carbon dioxide tension (Pa_{CO_2}) 2,6 kPa, standard bicarbonate 16,4 mmol/l and base excess –10,2. The lungs were clinically and radiographically normal. The abdomen was tender without evidence of peritonism. Palpation revealed a 6 cm firm, tender, non-pulsatile hepatomegaly with a 2 cm splenomegaly. The total bilirubin value was 215 µmol/l, conjugated bilirubin 68 µmol/l, alkaline phosphatase 108 IU/l, aspartate aminotransferase 1 141 IU/l and alanine aminotransferase 339 IU/l. Urinary catheterisation produced 10 ml of heavily bloodstained urine. The serum urea value

was 29,5 mmol/l and the creatinine value 593 µmol/l. Apart from distal sensory deficits related to the areas of vascular compromise, the central nervous system was intact. The haemoglobin concentration was 15,4 g/dl, the white cell count 16,3 x10^9/l and the platelet count 35 x10^9/l, and he had disseminated intravascular coagulopathy.

Initial management included crystalloid fluid and blood product resuscitation, which resulted in correction of the acidosis and the coagulopathy, and intravenous ceftriaxone 1 g. However, a peripheral blood smear taken at the time of admission to the respiratory intensive care unit revealed intracellular Gram-negative bacilli suggestive of *C. canimorsus*, and treatment with penicillin 2,5 million units 6-hourly and rifampicin 600 mg daily was commenced. The organism was subsequently isolated from blood cultures taken at the same time as the peripheral blood smear using the Bactec system and its identity confirmed. The patient was fully heparinised to limit anterograde coagulation, but during the next 48 hours the peripheral vascular compromise progressed with proximal extension of the ischaemic areas in the right hand, nose and feet (particularly the soles of the feet).

Progressive deterioration of respiratory function necessitated intubation and mechanical ventilatory support. An infiltrate developed in the mid-zone of the left lung, associated with an episode of haemoptysis, and alveolar haemorrhage was diagnosed. Subsequently diffuse bilateral opacification of the lung fields with reduced pulmonary compliance and hypoxaemia suggested development of the adult respiratory distress syndrome. A pulmonary artery catheter was placed and serial haemodynamic measurements were made over a 4-day period, during which time cardiopulmonary function remained essentially unchanged. The central venous pressure was 10 mmHg, pulmonary capillary wedge pressure 8 mmHg, mean arterial blood pressure 100 mmHg, mean pulmonary artery pressure 30 mmHg, cardiac output 7 l/min with an index of 4 l/min/m^2, systemic vascular resistance 1 096 dynes/cm^3 and pulmonary vascular resistance 200 dynes/cm^3 on insertion.

Peripheral blood smears taken 2 days after initiation of therapy revealed persistently high organism numbers despite appropriate antibiotics, and the patient was therefore given an exchange transfusion of 10 units of packed red blood cells, 10 units of fresh-frozen plasma and 12 units of platelets (6 000 ml of blood was venesected). The procedure was repeated after 36 hours and only thereafter were peripheral blood smears free of the organisms.

The vascular compromise progressed to gangrene in the patient's right hand, nose and toes. Technetium-99 m pyrophosphate scintigraphy (Fig. 1) confirmed the limits of perfusion of the digits. The gangrenous skin on the tip of the nose sloughed off spontaneously, but wet gangrene developed in the right hand and transmetacarpal amputation of all the fingers except the thumb was performed. Although the gangrenous toes were initially managed conservatively, they were all amputated after 6 weeks, at which time clear demarcation had occurred. All these wound sites healed after surgery. The patient developed three consecutive significant nosocomial infections during 8 weeks in the intensive care unit, namely a resistant *Staphylococcus aureus* septicaemia which responded to vancomycin, a *Candida albicans* fungaemia which responded to amphotericin and ketoconazole, and an *Acinetobacter anitratus* pneumonia which responded to ofloxacin. He remained in anuric renal failure for 6 weeks and required daily haemodialysis. Although he subsequently began to pass urine, he remained in high-output renal failure and required regular dialysis. The liver failure resolved spontaneously with gradual normalisation of liver functions. Mild coagulopathy persisted for 3 weeks and required daily administration of blood products before resolution.

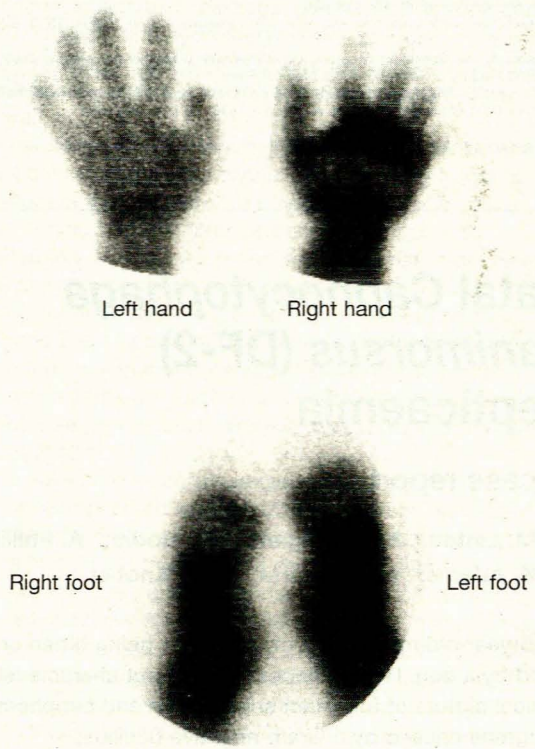


Fig. 1. ^{99m}Tc pyrophosphate scintigraphy of hands and feet showing the limits of perfusion of digits.

Seven weeks after admission the patient developed abdominal symptoms and intolerance of nasogastric feeds. He became more obtunded and was found to have right-sided hemiplegia. A computed tomography (CT) scan of the head confirmed an infarct in the left frontoparietal area. At the same time he became clinically septicaemic, and a CT scan of the abdomen (Fig. 2) revealed homogeneous enlargement of the liver and enlargement of the spleen, with areas in the spleen suspicious of abscesses or infarcts. A ^{99m}Tc colloid scan (Fig. 3) showed that functional tissue in the grossly enlarged spleen was limited to small islands only. An infarcted spleen was surgically removed, at which time the other abdominal organs appeared normal. The patient's condition stabilised after the laparotomy, but some 10 days later he had not regained bowel function and began to develop marked ascites. It was believed that he had probably developed a portal vein thrombosis. In view of his extremely poor prognosis, further investigations and surgical intervention were deemed inappropriate. He died just over 8 weeks after admission.

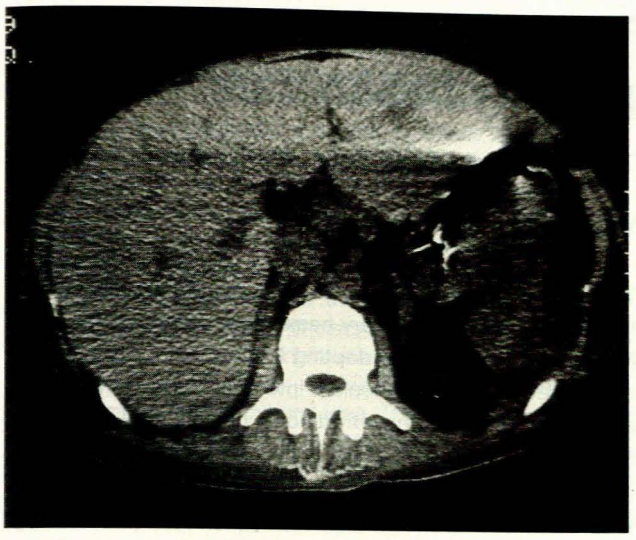
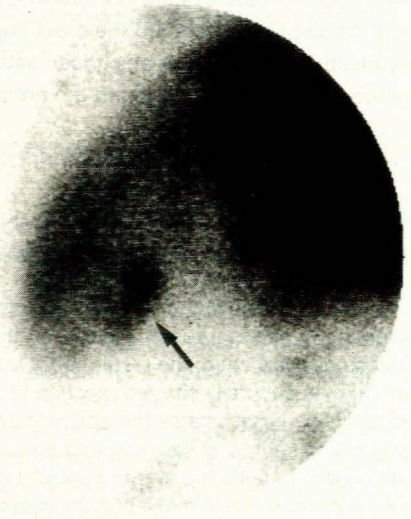


Fig. 2. Selected view of CT scan of abdomen showing enlarged liver and spleen with areas in the spleen suspicious of abscesses or infarcts.



Left posterior oblique

Fig. 3. ^{99m}Tc colloid scan of upper abdomen showing small islands (arrow) of functioning spleen posterior to large left lobe of liver.

At autopsy the amputation lines were almost healed and histological examination revealed no residual inflammation, tissue necrosis or vascular abnormalities. Bilateral small pleural effusions were present and the lungs were extremely heavy (right lung 1 280 g, left lung 1 100 g) with firm fleshy-grey cut surfaces. Microscopic organisation of alveolar exudate, interstitial fibrosis and proliferation of alveolar epithelium cells were consistent with the proliferative stage of diffuse alveolar damage as seen in the adult respiratory distress syndrome. Numerous cytomegalovirus (CMV) cells present in alveoli and alveolar epithelial cells indicated active CMV infection, suggesting an immunocompromised state. The heart was enlarged with a dilated right ventricle. Histological examination showed a few small areas of focal myocytolysis in the ventricular walls with early replacement fibrosis. The abdominal cavity was filled with 4,5 litres of odourless inflammatory exudate, shown to contain numerous polymorphs on cytological examination. An acute superficial ulcer was present in the prepyloric posterior wall of the stomach. There was no evidence of vascular occlusion of mesenteric vessels. Bowel loops contained copious lumps of inspissated bile and were covered with a thin fibropurulent exudate. A 15 cm long central loop of ileum, which appeared slightly darkened with a deeply bile-stained mucosa, showed denudation of the mucosal epithelium, serosal reaction and peritonitis. A similar area was found on the rectal surface. No bowel infarctions were present. The liver was congested with a nutmeg appearance and microscopic examination revealed severe bile congestion but no evidence of hepatitis or hepatocellular disease. The kidneys were large, pale and oedematous and histological examination revealed pigment and bile cylinders in tubules, but no residual evidence of acute tubular necrosis. The gall bladder, pancreas and adrenal, salivary, thyroid and parathyroid glands were normal with no evidence of infarction or CMV infection. Examination of the brain revealed a 5 cm left frontal cerebral infarction with central haemorrhage. Histological examination confirmed an organising haemorrhagic septic infarction, with numerous thick branching fungal hyphae within surrounding vessels, vessel walls and adjacent parenchyma, as well as in meningeal vessels with a localised suppurative meningo-encephalitic reaction. Death was therefore due to persistent multiple organ failure resulting from septicaemic complications following the dog bite and terminal opportunistic infections in the brain and lungs.

Discussion

C. canimorsus infection can result in severe sepsis following contact with a dog's saliva or a dog bite.[1] The offending organism has been shown to be a member of the oral flora of dogs and cats.[4,5] Infection by this organism, however, appears to be extremely rare given the high incidence of dog bites. A prevalence study showed a 3,3% carriage rate in the oral cavities of dogs in the Cape Peninsula.[4] The sepsis is clinically characterised by a malar rash, purpura/ecchymosis, relatively symmetrical peripheral gangrene, disseminated intravascular coagulopathy and glomerular necrosis with renal failure.[1] Additional features may include purulent meningitis or cerebral abscess, endocarditis, pulmonary infiltrates or adult respiratory distress syndrome, septic arthritis and myocardial infarction. Patients who are chronically disabled, immunocompromised, alcoholic or have had splenectomy are believed to be at greater risk of developing this disease after a dog bite. Certainly neglect of a dog bite without appropriate antibiotics would allow the organism to become established. The organism is known to be extremely difficult to culture and is exquisitely sensitive to many antibiotics including the penicillins, the cephalosporins, the tetracyclines, the macrolides and the quinolones. It is reported to be resistant to the aminoglycosides and the sulphonamides.

Because this organism is difficult to culture the definitive diagnosis is likely to be missed unless the intracellular bacillus is looked for on peripheral blood smears. Initial isolation may take several days and identification may not

be complete for up to 2 weeks.[4] A high index of suspicion is therefore needed in the correct clinical setting.

Treatment is debridement and penicillin unless contraindicated. Serial blood smears should be monitored until the organism is cleared from the bloodstream. In theory, exchange transfusion should assist in rapidly removing the baccilli load, and this was achieved in our patient. This form of therapy has not to our knowledge been reported previously. Alternatively, leucopheresis could be utilised. Plasma exchange would not clear the intracellular organisms.

Because the peripheral vascular compromise is a microvascular insult with anterograde vascular occlusion and disseminated coagulopathy, anticoagulant and thrombolytic therapy may be beneficial although the risks of haemorrhage from areas of infarct may be substantial. The haemodynamic features of *C. canimorsus* sepsis appear to be unlike other forms of severe Gram-negative sepsis in that the systemic blood pressure and systemic vascular resistance tend to be elevated in association with an increased cardiac output. This is in keeping with the pathophysiological process of microvascular insult and anterograde vascular occlusion in this disease. To our knowledge, these haemodynamic findings have not been reported previously.

Patients who survive the acute insult frequently require amputation of ischaemic digits. The timing of amputation will depend on the development of gangrene and its continuous effect on major organ dysfunction. Recognition of infarction of internal organs such as the spleen requires a high index of suspicion in this disease. Radionuclide scanning techniques assisted in the definition of areas of inadequate perfusion in our patient's limbs and internal organs and aided the decision to perform laparotomy when the usual clinical signs were confusing.

Since Butler *et al.*[3] first described this 'new disease' in 1977, approximately 50 cases have been reported in the English literature; since 1987, 6 cases have been identified at Groote Schuur Hospital.

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The role of culture in primary health care

Two case studies

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The purpose of this article is to show the importance of traditional healers in primary health care (PHC) services. Most countries, despite adopting PHC, have not incorporated traditional healers into this service. The article also illustrates how traditional healers fulfil three of Morrell's four PHC objectives, and how incorporating traditional healers into health services will fulfil the fourth objective.

The first contact between a black African patient and health care services usually takes place in the traditional healing system. Therefore health workers should realise that the traditional care system is important if PHC is to succeed. Traditional healers are the most important primary health care service in an African setting. This is highlighted by 2 cases described in the article.

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After the Alma-Ata conference of 1978, most countries adopted the primary health care (PHC) strategy as their universal health care system. PHC is the first and nearest contact between an individual and his health care system (whatever he conceives it to be). Health, on the other hand, is a process of adaptation, the result not of instinct but of an autonomous yet culturally shaped reaction to socially created realities.[1] It designates the ability to adapt to changing environments, to growing and ageing, to healing when damaged, to suffering and to peaceful expectation of death. A health care system is a cultural system just as language or religion is.

In every culture, illness, the response to it, individuals experiencing it and treating it, and the social institutions relating to it are all systematically interconnected. Health care in any culture includes patterns of belief about the causes of illness, norms governing the choice and evaluation of treatment, the socially defined and legitimised statuses of hierarchies such as chief or healer, institutions and power relationships. The health care system includes people's beliefs and patterns of behaviour. These behaviours and beliefs are governed by cultural beliefs. Lay people activate their health care by deciding when and whom to consult, whether or not to comply, when to switch between treatment alternatives, whether care is effective and whether they are satisfied with its quality. The prerequisite for the success of primary health care is the concept of community

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