CASE REPORTS

Non-traumatic clostridial myositis: an unusual feature of brain death

J. P. THYS
M.D.

P. ECTORS
M.D.

P. NOEL
M.D.

The Medical Intensive Care Unit, University Hospital of St Pierre, Brussels, Belgium

Summary

In a case of brain death, a *Clostridium sordellii* and *Escherichia coli* septicaemia—of probable gastro-intestinal origin—developed and was associated with a diffuse clostridial myositis. Factors responsible for the initiation and development of this unusual clostridial infection are discussed.

Introduction

Two patterns of non-traumatic clostridial tissue infections can be distinguished. The first one, which is relatively common, consists of visceral anaerobic cellulitis, with or without contiguous spread to the adjacent muscles. In the latter case, primary intra-abdominal infection can spread directly to the abdominal wall or, along the retroperitoneal tissue planes including the psoas muscles, spread to the lower extremity (Mzabi, HIMAL and MacLean, 1975; Hitchcock and Bubrick, 1976). The second type of spontaneous clostridial tissue infection, which is quite unusual, is metastatic myositis due to a muscular seeding during a clostridial septicaemia arising from a site distant from the muscular lesion.

A case is reported of spontaneous gas gangrene with clostridial septicaemia occurring in a young patient with brain death due to an alcoholic intoxication.

Report of a case

One New Year's Eve, a usually sober 18-year-old drank in a short time 26 oz of whisky and an indefinite number of pints of beer: he was found unconscious on a chair approximately 15 min later. He was immediately sent to St Pierre's Hospital, Brussels, where a cardio-respiratory arrest was observed. Resuscitation manoeuvres restored BP to 90 mmHg within a few minutes, but the patient remained unconscious without any spontaneous respiration. Two hours after admission, the patient

was in deep coma with no reaction to painful stimuli; the pupils were dilated and fixed to light; the corneal responses were absent; he remained apnoeic and the rectal temperature was 34°C; the diuresis was 70 ml/hr but the patient became anuric a few hr later. The treatment consisted of respiratory maintenance, plasma expanders, alkalinization, intravenous frusemide and dexamethasone. No i.m. injection was given.

Twenty-four hours after admission, the clinical status remained unchanged: brain death was assessed by the absence of brain-stem function and isoelectric electroencephalogram. The stools were stained with fresh blood. The blood tests were as follows: urea 22.7 mmol/l; creatinine 707.2 µmol/l (at admission, 141.4 µmol/l); pH 7.20; lactate 6.8 µmol/l; potassium 6.7 mmol/l; SGOT 5160 i.u.; SGPT 1700 i.u.; creatine phosphokinase 20 000 i.u.; amylase 750 Street-Close u./dl (normal < 33 u./dl). The alcoholaeamia was 280 mg/dl approximately 4 hr after ingestion.

On the second day, fever and deep shock developed. A few hours later, generalized muscle oedema appeared, most pronounced in the left thigh and in the back where the skin presented cyanotic marblings. Gaseous crepitation was present in the same areas; an X-ray film (Fig. 1) revealed extensive soft tissue gas in the thigh. During a left quadriceps biopsy, foul-smelling gas escaped from the aponeurotomy; the muscle appeared dark with gelatinous oedema but was not visibly necrotic; *Clostridium sordellii* was isolated from this muscle sample as well as from the 2 blood cultures, one of which also revealed an *Escherichia coli*. The patient died 48 hr after admission. Post-mortem examination confirmed a diffuse oedematous myositis and acute necrotic-haemorrhagic pancreatitis.

Discussion

In this case, acute alcoholic intoxication induced a cardio-respiratory arrest and consequent brain death. A muscular oedematous swelling with

Correspondence: J. P. Thys, M.D., Department of Internal Medicine, University Hospital of St Pierre, rue Haute 322, B-1000 Brussels, Belgium.

crepitations, characteristic of gas gangrene, was observed: this diffuse myositis was attributed to a *C. sordellii* septicaemia assessed by blood and muscle cultures.

FIG. 1. X-ray of the left thigh demonstrating muscle oedema and intramuscular gas.

Clostridial septicaemias of non-traumatic origin are not rare but septic muscular involvement in this condition is quite unusual: Jendrzejewski *et al.* (1978) recently reported 2 cases and reviewed 8 other cases. In 7 of these 10 cases, ulcerative lesions of the gastrointestinal tract were present: most often, carcinomas were found but focal necrosis or inflamed areas of the intestinal mucosa associated with haematological or lymphoproliferative disorders were described (Congeni and Nankervis, 1976; Jendrzejewski *et al.*, 1978). Such breaks in the intestinal mucosa probably allowed normal faecal flora access to the blood stream. In the present patient, gastrointestinal lesions could be inferred

from the emission of bloody faeces; they could result from necrotico-haemorrhagic pancreatitis or from cerebral brain damage (Bogoch, 1974).

MacLennan (1962) suggested that slight local tissue injury might lead to the metastatic foci of clostridial myositis in the case of septicaemia. Previous muscle injury has been noted in the same site of spontaneous myositis, probably related to clostridial septicaemia (Valentine, 1957; Engeset *et al.*, 1973; Jendrzejewski *et al.*, 1978). In the present patient, muscular lesions—as shown by increased muscle enzymes and the very rapid rise of creatinine (Grossman *et al.*, 1974)—might have resulted from circulatory arrest and persistent shock with muscle anoxia and partial anaerobiosis: such conditions decrease oxydo-reduction potential in which *Clostridium* sp. could grow and produce myositis. The position of the patient when found unconscious could account for asymmetrical body compression and asymmetrical muscular involvement.

Systemic factors seem to be very important in the pathogenesis of spontaneous clostridial infections. Nine of the 10 patients reviewed by Jendrzejewski *et al.* (1978) presented debilitated states or malignancies. Cancer as well as cytostatic and immunosuppressive therapy are often cited as predisposing factors to *Clostridium* septicaemia (Smucker, Reed and Harding, 1960; Cabrera, Tsukada and Pickren, 1965; Wynne and Armstrong, 1972; Mzabi, *et al.*, 1975). In addition to the epithelial break due to the neoplasia or its treatment, the metabolic and immunological changes associated with neoplasia may contribute to create a local environment favourable to infection. Likewise, diabetes has been found related to non-metastatic spontaneous clostridial myositis (Soscia and Grace, 1965; Mzabi, *et al.*, 1975). Surprisingly, cancer chemotherapy and diabetes do not seem to be frequently associated with clostridial myositis arising at a site distant from a visceral lesion (Jendrzejewski *et al.*, 1978). None of these associated factors were present in this patient who was in good health until the terminal event.

C. sordellii usually associated with other *Clostridium* spp. has been shown to be an unusual causative micro-organism of gas gangrene in man; it has rarely been found as the unique pathogen in that disease: in such cases, muscular lesions with gelatinous oedema without obvious necrosis have been described previously (MacLennan, 1962; Browdie *et al.*, 1975).

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