

Endogenous Gas Gangrene after Laparoscopic Cholecystectomy

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ABSTRACT

Clostridial gas gangrene of the abdominal wall is rare, and it is usually associated with organ perforation, immunosuppression or gastrointestinal malignancies. In this paper, we present a case of fulminant, endogenous gas gangrene in a 58-year old diabetic female with arterial hypertension and atherosclerosis, following uneventful laparoscopic cholecystectomy. She developed gas gangrene of the abdominal wall 12-hours after cholecystectomy and died 24-hours after the onset of the first symptoms, in spite of treatment.

Keywords: Cholecystectomy, clostridial infection, non-traumatic gas gangrene

Gangrena Gaseosa Endógena Tras la Colectomía Laparoscópica

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RESUMEN

La gangrena gaseosa clostridial de la pared abdominal es rara, y normalmente se halla asociada con la perforación de órganos, inmunosupresión o malignidades gastrointestinales. En este trabajo, se presenta un caso de gangrena gaseosa endógena fulminante en una mujer diabética de 58 años con hipertensión arterial y aterosclerosis, tras una colectomía laparoscópica sin incidentes. Doce horas después de la colectomía, la mujer desarrolló una gangrena gaseosa de la pared abdominal, y murió 24 horas después del comienzo de los primeros síntomas, a pesar del tratamiento.

Palabras claves: Colectomía, infección clostridial, gangrena gaseosa no traumática

West Indian Med J 2011; 60 (1): 96

INTRODUCTION

Endogenous gas developed in the absence of trauma is associated mostly with conditions causing immunodeficiency, such as gastrointestinal malignancy, haematological disease, diabetes mellitus and atherosclerosis (1, 2). Early diagnosis is difficult and the mortality rate still remains high, between 50–93% (1). The phenomenon of postoperative gas gangrene is well described including several case reports,

both post laparoscopic cholecystectomy and open surgical approach (1, 3).

We present the case of a 58-year old diabetic and hypertensive woman who developed clostridial gas gangrene of the abdominal wall following an uncomplicated laparoscopic cholecystectomy. Despite intensive antibiotic therapy and repeated urgent operative treatment, no positive response was obtained.

CASE REPORT

A 58-year old female patient was admitted to the Department of digestive surgery for elective surgical treatment of symptomatic cholelithiasis. A year before, she suffered an episode of acute cholecystitis and during the last six months she had several attacks of non-inflammatory biliary colics. She had no features of common bile duct stones.

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The patient had a long standing history of arterial hypertension, which was regulated in a satisfactory manner. She was obese, with a body mass index of 32, and she suffered from Type 2 diabetes mellitus, which was regulated with insulin.

The laparoscopic cholecystectomy was uneventful and the patient was transferred to the surgical ward two hours after the procedure. Twelve hours after the surgery, she started to feel abdominal pain. The pain was sharp in nature and it was located in the right subcostal area towards the right flank. A sero-sanguinous exudate with a sickly, sweet odour was noted at the right lateral trocar wound. Antibiotic therapy with benzylpenicillin, metronidazole and gentamicin was immediately initiated, as a case of gas gangrene was suspected. She had urgent median laparotomy with inspection of all the abdominal viscera which seemed ordinary. In the subhepatic space, a small amount of serosal fluid was found. The right retroperitoneal space was full of air bubbles, as was the subperitoneal right lateral abdominal wall. All bubbles were wide open. The retroperitoneal space above the right kidney, from the diaphragm to the iliac vessels, was opened and drained. The abdominal wall was closed with running suture. During surgery, tissue and fluid samples were taken for microbiological examination. The gram stains contained a few white blood cells (WBCs) and Gram positive rods suggestive of *Clostridium spp.* Six hours after the second surgery, the patient developed signs of the abdominal compartment syndrome. Urgent re-laparotomy was indicated. After entering the abdomen, a lot of free malodorous air came out. A small amount of sero-sanguinous exudate was present in the abdominal cavity. All intra-abdominal organs seemed normal. All of the right retroperitoneal space and the inner surface of the right lateral abdominal wall were dark and crepitations were present. No sign of transmural spreading of the infection was noted. Multiple incisions in the retroperitoneal space were performed, yielding a thin sero-sanguinous exudate with a sickly, sweet odour. The abdominal cavity was closed with a temporary abdominal closer. The patient was transferred to the Intensive Care Unit but died 24-hours after the onset of the first symptoms, despite intensive postoperative treatment.

At the autopsy, massive myonecrosis of the retroperitoneum and abdominal wall was confirmed, but no macroscopic organ perforations or fistulae were present. Neither malignant cell changes, nor signs of other abdominal or gastrointestinal malignancies were found. Extensive atherosclerosis was detected in the carotid arteries, the abdominal aorta and both lower limb arterial vessels.

Clostridium perfringens was recovered from the tissue, from the fluid aspirate, as well as from blood cultures. Anaerobic cultures revealed typical colonies with a double zone of haemolysis on blood agar plates. The final identification was achieved using an API 20A microbiological system (bioMérieux, France).

DISCUSSION

Gas gangrene is a condition of rapidly developing and spreading infection mediated by toxins, which are released by the bacteria *Clostridium spp.* Most cases are reported to be due to *Clostridium septicum* (4, 5), although several reports (3, 6) and review articles demonstrated that *Clostridium perfringens* also plays a role in endogenous gas gangrene. Non-traumatic gas gangrene is associated mostly with immunodeficiency-related conditions, such as gastrointestinal malignancy, haematological disease, diabetes mellitus and atherosclerosis (1, 2). This form of the disease is reported to have a very high mortality, up to 90% (7). The possible pathophysiological pathway could be the infection of normal tissue – with no history of trauma – with *Clostridium perfringens* and its toxins in an anaerobic abdominal environment (8). Among the other conditions, two major reasons could be responsible for the extremely high mortality: the absence of early symptoms and a “central” (at trunk) site of manifestation. These two points, which are somewhat related, have been reported to have major impact on survival (9). At the early stage, non-traumatic gas gangrene produces symptoms that are minimal or nonspecific, before the fulminant toxæmia progresses. Post procedural – post cholecystectomy gas gangrene was previously well described (10).

In a paper published in 2004 (11), we reported a case of a fulminant non-traumatic gas gangrene in a previously healthy male, with no risk factors for gas gangrene development. Contrary to that, the patient described in this recent case had several risk factors: obesity, arterial hypertension and Type 2 diabetes mellitus regulated with insulin. Twelve hours after laparoscopic surgery, she developed fulminant clostridial myonecrosis with a fatal outcome. According to the fulminant clinical features and course of manifestation, we believe that metastatic endogenous gas gangrene was present. Considering the major symptoms and findings at the initial surgery, we assume that the infection started in the lateral abdominal wall upon the achievement of anaerobic conditions, due to the generalised atherosclerotic alterations of vessels and the associated diabetes mellitus, which causes perfusion damages in the affected areas. Although an intra-abdominal pressure of up to 13 mmHg does not induce haemodynamic changes by pressuring the big abdominal blood vessels, the extension and pressure on muscles during the laparoscopic procedure, in addition to the already altered blood vessels, contributed to anaerobic conditions in musculature and clostridial infection in this particular case. The odour and the wound exudates, along with haemodynamic instability, appeared very quickly. Characteristic lesions and the presence of Gram-positive bacilli with rare WBCs provide strong presumptive evidence of a clostridial gas gangrene. Unfortunately, despite an early and correct diagnosis aided by microbiological findings, and intense antibiotic and surgical treatment, the patient died.

In summary, laparoscopic surgery is associated with better preservation of the immune system and decreased incidence of infectious complications. However, the surgeon should be aware that carbon dioxide pneumoperitoneum affects the peritoneal response to injury, at least in patients with predisposing risk factors and may give rise to a metastatic endogenous infection with gas forming organisms. Unfortunately, clostridial gas gangrene still has a poor prognosis.

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