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Severe Leptospirosis Presenting as Acalculous Cholecystitis

Colecistitis alitiásica como forma de presentación de una leptospirosis grave

Leptospirosis is a worldwide zoonosis caused by spirochetes, more common in humid climates and rural areas, although in recent years an increase in cases in urban areas in developing countries has been described.¹ Severe presentations (Weil disease) with acute renal failure, respiratory failure due to adult respiratory distress or pulmonary hemorrhage are variable in frequency, but could appear in approximately 10%–50% of cases.^{2–4} We present a case of severe leptospirosis and an uncommon clinical presentation, acute acalculous cholecystitis (AC).

A 55-year-old man, who worked in a farm, with a prior history of a lacunar stroke, came to the Emergency department of our Hospital with a 3-week history of myalgia and articular pains. In the last few days he had also presented abdominal pain radiated to the right shoulder, diarrhea and jaundice. On arrival, the patient was in mild distress, diaphoretic, jaundiced, with right conjunctival chemosis and intense pain in the right upper quadrant of the abdomen. He was in shock and in atrial fibrillation at 120 ppm. Blood tests revealed urea: 190 mg/dl (vn: 10–50), creatinine: 3 mg/dl (vn: 0.5–1.4), total bilirubin: 12.4 mg/dl (vn: 0.2–1.2), direct bilirubin: 12.1 mg/dl (vn: 0.1–0.3), ALT: 134 U/l (vn: 5–37), AST: 61 U/l (vn: 5–45), amylase: 157 U/l (vn: 20–115), CRP: 16.5 mg/dl (vn<0.5), neutrophilic leucocytosis: 27 100/mm³ and 84.20% respectively, thrombocytopenia: 57 000 platelets/mm³ and hyperfibrinogenemia: 1.291 mg/dl (nv: 150–400). An arterial gasometry reveals severe hypoxemia and hypocapnia. A chest X-ray showed a bilateral interstitial infiltrate and an abdominal CT scan revealed a gallbladder with thickened and oedematous walls without cholelithiasis or common bile duct dilatation. With a diagnosis of cholecystitis and possible cholangitis emergency surgery was performed. A cholecystectomy and live biopsy were performed. The patient was admitted in the Intensive Care Unit due to multiorgan failure, and mechanical ventilation, ionotropic support and anti-biotherapy with ceftriaxone and ciprofloxacin.

During the next few hours the patient deteriorated, presenting a leukemoid reaction in blood tests and increase in bilirubin levels. Surgical re-exploration was performed. A thrombosis of the portal vein was observed, and the patient died in the operating room.

Due to prior contact with animals, serology for *Mycoplasma*, *Chlamydia*, *Brucella* and *Coxiella* were performed, which were negative; however, using indirect immunofluorescence, IgG and IgM antibodies for *Leptospira interrogans* were positive at 1/512 (nv<1/256) and 1/2.560 (nv<1/40), respectively. Microscopic agglutination (MAT) showed a title of 1/100 to the *hardjo* serovariety, and negative titles to *bratislava*, *canicola*, *copenhageni*, *castellonis*, *grippityphosa*, *pomona* and *pyrogenes* serovarieties; polymerase chain reaction in a urine sample was positive for *Leptospira* spp. Histological examination of the gallbladder revealed an unspecific cholecystitis without detecting microbes and the liver biopsy revealed an infiltration of inflammatory cells, mostly neutrophils in the portal spaces.

An AC due to *Leptospira* can appear in isolation,^{5–7} or associated with pancreatitis, with high levels of amylase and lipase associated with radiological findings due to pancreatitis.^{8,9} The pathogenesis of AC is unknown, although as leptospirosis is considered a generalized vasculitis, a microvascular involvement could play an important role.

Occasionally the bacteria can be detected in the gallbladder,⁷ bile or intraabdominal fluid¹⁰ or by immunohistochemistry finding bacterial antigens in the gallbladder wall.⁷ Treatment is controversial; in some cases the AC can resolve with medical treatment, without surgery^{6,8}; however, there have been cases described of clinical deterioration after an initial stabilization and several days of hospitalization^{5,6,10} and a period of several days of intensive monitoring is recommended. In our case, due to the severity of the clinical presentation, emergency surgery was indicated, and in the reoperation an extensive thrombosis of the portal vein was found, that could have contributed to the fatal outcome.

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It is therefore important, in young patients with AC, to perform a directed clinical anamnesis on possible epidemiological risk of leptospirosis, in order to start an adequate antibiotic treatment and in occasions avoid surgery using a conservative approach.

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Intestinal Peritonitis due to Nontyphoidal *Salmonella*^{☆,☆☆}

Peritonitis intestinal por *Salmonella* no tífica

Salmonella are Gram-negative bacilli classified as enterobacteriae that are present worldwide and are a part of the intestinal flora of humans and other animals.¹ They are usually non-pathogenic, but in certain situations can become pathogenic and cause a wide variety of diseases in humans.

An example of the infections caused by *Salmonella* sp. are the extraintestinal manifestations, that are classified into 4 types: primary bacteriemia (PB), enteritis associated with bacteraemia or secondary bacteraemia (SB), focal digestive infection (FDI) and non-focal digestive infection (NFDI).²

Excluding the digestive tract, the most common locations of intraabdominal infection caused by these microorganisms are the spleen in the form of abscesses and the gallbladder as

acute cholecystitis. Other less frequent locations are liver abscesses, pancreas, adrenal glands and subphrenic abscesses. Peritonitis caused by *Salmonella* sp. is extremely rare.

The most important predisposing factors for infection by this microorganism are immunodeficiency or chronic diseases such as diabetes, chronic renal failure, falciform anaemia, alcoholism and the use of immunosuppressive drugs.⁶ These factors predispose for a primary bacteriemia²⁻⁵ with subsequent dissemination of the microorganism.

A 41-year-old man came to the Emergency Department for abdominal pain, nausea and bilious vomiting of 24 h duration after and excess of alcohol intake.

He had a prior history of dyslipidemia with elevated triglyceride counts, smoking, and chronic alcohol abuse. Three years prior he had been hospitalised for acute severe pancreatitis with an intraabdominal abscess and a second episode of hospitalization a year and a half later in the intensive care unit for severe acute pancreatitis of the neck and tail. He was diagnosed with a pancreatic pseudocyst and hepatic steatosis in a control CT.

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