Saprophyticus bacteria: Cause of fulminant and lethal sepsis?

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ABSTRACT

Introduction: Sepsis continues to be a clinical presentation with high morbidity and mortality. The severity depends on the causal agent and the population which is affected. Below, we describe a case of septic shock in an immunocompromised patient, which triggered a multiple organ failure in a few hours, and finally, it caused her death. Splenectomized patients are at higher infection risk for less common pathogens. Case Report: Our patient is a 47-year-old, splenectomized woman who developed a clinical presentation of septic shock with purpura fulminant and disseminated intravascular coagulation caused by Capnocytophaga Canimorsus, a Gram-negative bacteria that lives in the mouths of cats and dogs. The infection is associated with bites, scratches and contact with secretions. Conclusion: Splenectomized patients are at increased risk of infections from less common microorganisms. In this case, we are facing with a very serious pathology that can trigger multiple organ failure and the death of the patient in a few hours, we must suspect it in patients at risk. The best treatment is preventive therapy, which can improve the survival rate of these patients.

Keywords: Capnocytophaga canimorsus, Purpura fulminant, Splenectomized

INTRODUCTION

Sepsis is still a clinical presentation of high morbidity and mortality. The severity of the disease depends not only on the causative agent but also on the population in which the infection occurs. Every day it is more frequent to treat immunosuppressed patients in the Emergency Department, in these patients, saprophyticus bacteria, usually non-aggressive, can trigger a septic shock with high mortality of vulnerable patients.

Next, we present a case of sepsis that, although it has moderate transcendence in the population with intact immunity, shows a high mortality in splenectomized patients. The patient is a 47-year-old woman, splenectomized, who presents septic shock with fulminant purpura with disseminated intravascular coagulation, triggered by Capnocytophaga Canimorsus [1]. It is a gram-negative bacillus (Catalase and Oxidase positives), which can be found in the oral cavity of dogs and cats, although it has also been described as a possible vector Hylobius Abietis due to insect bites. Human infection is associated with biting, scratching or contact with secretions [2]. Risk factors include immunosuppression, splenectomy,
alcoholism, cirrhosis, chronic obstructive pulmonary disease and malignant haematological diseases.

In habitual clinical practice, when faced with a splenectomized patient who presents with septic shock, we must keep in mind less frequent microorganism as possible cause of the disease. Early care with adequate antibiotherapy allows to modify the course of the disease, improving the survival of these patients.

CASE REPORT

A 47-year-old woman with a history of HBV, HCV, congenital solitary kidney (left), splenectomized due to abdominal trauma ten years previously and meningococcal meningitis 22 years ago with good progress and no sequels. Taken to A&E with a clinical picture of 24 hours developing fever, chills, shivering and intense miregane. This was accompanied by hypogastric discomfort relating to menstruation. The analysis upon arrival showed hemogram, coagulation and biochemical anodones, with no heightened inflammatory parameters. The urine analysis is normal, the radiography of the thorax shows no pathological findings and the electrocardiogram shows no arrhythmia or other alterations.

Due to the prior case of meningitis, a CT is carried out, ruling out organic pathology and a lumbar puncture was also carried out without incident. A clear liquid comes out without pressure and the biochemical breakdown has red blood count of 7hem/µL, 58 mg/dL of glucose (Glycaemia 86 mg/dL) and 28 mg/dL of proteins. The patient is in a stable condition in the examination in A&E, for this reason, 10 hours after her arrival, it is decided that she should be discharged with symptomatic treatment.

When getting ready to get dressed she feels dizzy with vasovagal symptoms. She was monitored again and intensive fluid therapy was initiated with good response. The vital signs are checked, and a tachycardia of 130 bpm and AP 80/50 mmHg is identified, intensive fluid therapy is given and the symptoms improves. Blood glucose levels are checked with a value of 45 mg/dL. The vital signs are checked, and a tachycardia of 130 bpm and AP 80/50 mmHg is identified, intensive fluid therapy is given and the symptoms improves. Blood glucose levels are checked with a value of 45 mg/dL. After carrying out a new blood test, with the new results, empirical treatment with ceftriaxone and bolus of corticosteroids was started, and the patient is admitted to the ICU, requiring vasoactive drugs.

Following these findings, a new analysis is requested presenting 2.0x10⁹/L leucocytes, 1.4x10⁹/L absolute neutrophils, 9x10⁹/L platelets, Cr 1.99 mg/dL, increased clotting time, hypofibrinogenemia, lactic acid 16 mmol/L, pH 7.06 with pCO₂ 47 mmHg and HCO₃- 13 with EB -17.6.

In light of this situation, empirical antibiotic therapy is started with ceftriaxone and an injection of corticosteroids.

The patient continues to deteriorate in general; the skin turns mottled especially the torso, with distal hypoperfusion in the limbs. Due to the septic shock which arises, intensive care is contacted. Upon being admitted to ICU, the patient is agitated with a feeling of imminent death, tachypnoea, with progressive ecchymosis, developing purpura over the whole surface of the body. She has AP 75/35 mmHg, a heart rate of 120 bpm, with bad perfusion of noradrenaline at 0.7 mcg/kg/min. Due to severe desaturation, orotracheal intubation proceeds as well as connection to mechanical ventilation. Hemodynamic resuscitation maneuvers are started.

An echography of the abdomen is carried out and a lack of portal flow is observed, the liver has a normal aspect without other findings. Haemocultures are extracted, urine culture, antigen test for pneumococcus and legionella. Clindamycin is added to Ceftriaxone due to suspected encapsulated bacteria (meningococcus, haemophilus influenza, neisseria meningitides, pneumococcus) in the splenectomized patient, as well as the possibility of staphylococcal toxic shock.

Despite intensive fluid therapy, the patient requires an increase in dosage of noradrenaline to 2 mcg/kg/min to maintain MAP 65 mmHg. Anuria is found and severe coagulopathy is manifested, Cr 1.78 mg/dL Sodium 148 mEq/L, Potassium 2.72 mEq/L, lactic acid of 25 mmol/L. Considering this situation, it is decided that they should start continuous therapy of extra-renal depuration with a dose of 60 cc/kg.

Despite all these measures, the patient falls in refractory shock with disseminated intravascular coagulation (DIC), and finally the patient expired. The family is asked if an autopsy can be carried out, and this is agreed to 24 hours after death, Gram-negative bacilli begin to grow in haemocultures. After 72 hours, Capnocytophaga Canimorsus is identified. The findings are told to the family who stated that the patient lived with two dogs. The patient did not mention any possible bites, nor did she show any signs of bites.

DISCUSSION

We are looking at a case of septic shock with DIC, with an uncommon development, causing the death of an immunosuppressed, splenectomised patient, in under 6 hours after being admitted into ICU [3].

In splenicomized patients, there is a reduction in clearance of bacteria in the blood and a deficiency in the humoral immune system. The spleen is the most efficient organ for the clearance of IgG antibodies which are joined to coated bacteria and is fundamental in the clearance of encapsulated bacteria which are not opsonised by antibodies or the complement system [4]. In studies carried out on splenicomized animal models, it is necessary to increase the number of antibodies compared to bacteria and the concentration of anticapsular antibodies in serum so that normal hepatic bacterial clearance can occur.

Splenectomized patients demonstrate a dysfunction of the humoral immune system with a reduction of IgM antibodies in the plasma, which are fundamental
in the defense against polysaccharides in the bacterial membrane as well as a reduction in the number of memory B lymphocytes which produce antibodies [5].

A limited development of IgM antibodies faced with polysaccharides is accompanied by a delay and reduction in magnitude of the response to bacterial vaccines compared to the response in normal subjects. Purpura fulminant [6] is a thrombotic disorder which progresses rapidly and causes cutaneous haemorrhagic infarctions as well as DIC with high morbimortality.

Initially erythematous blemishes appear which quickly spread into central areas of irregular necrosis, surrounded by an erythematous border. The spreading is related to the occlusion of small vessels in the dermis due to microthrombosis. In more advanced stages, it produces irreversible endothelial ischaemia with red blood extravasation to the dermis and necrosis.

Among the possible causes of Purpura fulminant is infection, notably sepsis due to Neisseria Meningitidis (10–20% of cases) and Varicella, as well Streptococcus Pneumoniae, Streptococcus Group A and B, Haemophilus Influenzae, Staphylococcus Aureus, Capnocytophaga Canimorsus and Plasmodium Falciparum. Regarding the non-infectious causes, of particular note is a deficit of Proteins C and S either with autoimmune or congenital cause.

The cases of purpura fulminant found in some forms of severe sepsis is due to a severe inflammatory reaction, with endothelial dysfunction, with activation of coagulation and the paths of the complement system trigger DIC. There is a heightened consumption of factors of coagulation and platelet circulation. The loss of proteins C and S lead to the formation of thrombus, inhibition of fibrinolysis and increase in the inflammatory reaction.

The purpura initially starts in distal regions of the hands and feet, later spreading across the whole organism. Sometimes a general rash appears and spreads to affect the whole surface of the body, this is accompanied by microvascular thrombosis which causes progressive multi-organ failure with heightened mortality.

Capnocytophaga Canimorsus is a Gram-negative bacilli (Catalase and Oxidase positive) which forms part of the oral flora of cats and dogs. Another potential vector is Hylobius abietis, from bites and stings from this insect. Human infection is associated with bites, scratches or contact with secretions. The main risk factors are: immunosuppression, splenectomy, alcoholism, cirrhosis, COPD and malign haematological illnesses, although >40% of patients show no signs of risk [7].

The most common symptoms associated with these bacteria are: sepsis (30%), meningitis (with or without purpura) and others, such as endocarditis, pancolitis and osteomielitis [8].

Symptoms appear from within 24 hours to 8 days in people of all ages. They are most commonly found in men 3:1. The clinic varies depending on the degree of severity of the infection, ranging from light to fulminant.

Initially, unspecific symptoms are manifested, such as fever, myalgia, abdominal pain, dysnea, confusion and migraines. It can cause a severe inflammatory reaction, causing microvascular injury of the endothelium which causes DIC, gangrene and multiorgan failure [9].

The etiological diagnostics are carried out on a blood specimen. It entails a slow-growing bacteria which grows between 35 -37 °C, in an anaerobic environment, we can also identify it by mass spectometry (MALDI-TOF) or by PCR of the genetic sequence. Capnocytophaga Canimorsus is usually resistant to aztreonam, trimetoprim, aminoglycosides, first-generation cephalosporins and colistin. Mortality due to sepsis caused by these bacteria oscillates between 25-60%, with high mortality in patients with septic shock [10].

Treatment is based on haemodynamic Support and early antibiotic treatment targeting the etiological causal agent as early as possible. In splenectomized patients, we must think about encapsulated bacteria. An early diagnostic and prompt start to effective treatment can reduce morbimortality.

After 48 hours of the clinical presentation, the autopsy is carried out in which pulmonary and hepatic alterations associated with septic shock are identified. There are sharp pulmonary alterations which can constitute the initial stages of diffuse alveolar damage, with extensive vascular congestion. Regarding the hepatic alterations, there is passive centrilobular congestion with neutrophilic, parenchymatous microabscesses (Figure 1) and a 1.3 cm subhepatic accessory spleen. Regarding the sigmoid colon, there is an acute diffuse subserosal hemorrhage with no alterations in intestinal mucosa (Figures 2). On the skin there is vascular microthrombosis in superficial vessels and diffuse vascular congestion, without hemorrhage (Figure 3). The bacteria were only isolated in the haemocultures extracted upon admission, they were not isolated in the specimens taken from the autopsy.

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Figure 1: Histological image of hepatic parenchyma, in the center. The liver microabscess formed by polymorphonuclear cells and cellular detritus was observed.
CONCLUSION

When faced with a splenectomized patient admitted with septic shock, an early intervention is key to improve the prognosis of these patients. A good anamnesis is essential that allows us to guide the possible etiology of the disease, taking into account the possibility of less frequent microorganisms as possible responsible. Early care, with an adequate therapy of broad-spectrum antibiotics, can modify the course of the disease, improving the prognosis and survival of these patients.

List of Abbreviations

HBV: Hepatitis B virus
HCV: Hepatitis C virus
A&E: Emergency Area
CT: Tomography Computed
ICU: Intensive Care Unit
MAP: Mean Arterial Pressure
AP: Arterial Pressure
Cr: Creatinine
DIC: disseminated intravascular coagulation
COPD: Chronic Obstructive Pulmonary Disease

REFERENCES


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Author Contributions
Juan Francisco Martínez Carmona – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published
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Conflict of Interest
Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.

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