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Case Report

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Purulent Pericarditis and Pneumopericardium by Streptococcus Anginosus

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Abstract

We report a case of Purulent pericarditis and pneumopericardium by Streptococcus angiosus in 35-year-old man detained in prison, history of ischemic cerebrovascular event due to cocaine, with no recent surgical or traumatic history who had subacute clinical course of chest pain and signs of systemic inflammatory response. He was empirically treated with vancomycin and cefepime with subsequent pericardiotomy and drainage. The patient has a poor clinical evolution after the procedure, presents septic shock and dies. This is a rare case, a serious infection, the second reported in world, however in comparison to the first case the clinical course was different.

Keywords: Pericarditis, Streptococcus Anginosus, Infection

Case Report

A 35-year-old male, detained in a prison, with history of ischemic cerebrovascular event due cocaine use, with no recent surgical or traumatic history. He presented a 15-day course of diffuse chest pain with pleuritic features, occasional fever associated with orthopnea and functional class impairment due to dyspnea NYHA I to III. The physical examination documented poor oral hygiene, without pharyngeal abscesses, nor cervical lymphadenopathy, hypotension, tachycardia, fever (39.5°C), O2 saturation 81%, and pericardial rub without clinical signs of cardiac tamponade. Paraclinics were obtained in which were documented leukocytosis (16820 cls) neutrophilia (88.3%) and elevated C-reactive protein (218 mg/L). The electrocardiogram evidenced ST-segment elevation in the lower wall and ST depression in the anterior wall, the chest X-ray showed an increase in the cardiac silhouette as a relevant finding. We performed a complementary study with chest CT that revealed pneumopericardium (Figure 1) and an echocardiogram with a pericardial collection of 150 to 200 ml and segmental disorder of contractility of the left ventricle. Because of the above, purulent pericarditis was suspected, vancomycin and cefepime were empirically initiated and simultaneously was taken to pericardiotomy with extraction of 400 ml of purulent liquid sent to culture. Microbiological isolation corresponded to S. anginosus. During the hospitalization, in search of the entrance door, esophagogastroduodenoscopy was performed in search of gastrointestestinal fistulas or lesions, which evidenced an ulcer that compromised two-thirds of the esophageal circumference. Despite of surgical drainage and antibiotic redirection, the patient worsened his general condition, developing septic shock, multiple organ failure, disseminated intravascular coagulation with the appearance of multiple subsequent cerebral infarctions that eventually led to his death.

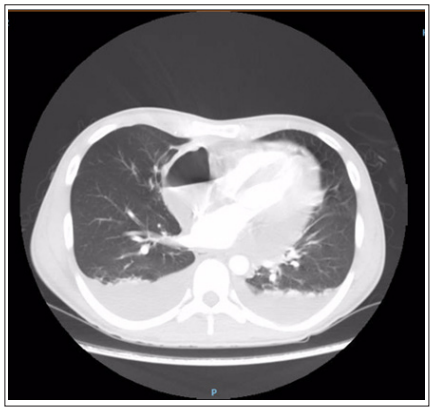

Figure 1

Discussion

Purulent pericarditis is a rare pathology in the modern antibiotic era, with an approximate incidence of 1 per 18,000 patients and can be caused by a wide variety of microorganisms. Its main routes of infection are hematogenous by bacteremia secondary to foci of infection at a distance, mucosal lesions, by contiguity from intrathoracic infections, or by direct inoculation through trauma or thoracic surgeries [1]. The Streptococcus anginosus group (SAG) is responsible for 6% of purulent pericarditis and this is the only group that has been related to the development of pneumopericardium, an extremely rare complication with isolated reports in the literature [2].

The Streptococcus intermedius, constellatus and anginosus constitute the SAG, also called milleri and have been widely recognized for

their ability to produce intrathoracic infections. They are facultative, immobile anaerobes that express an inconsistent phenotype of hemolysis B, A or Y in blood agar, express antigens A, C, F or G and differentiate from the other streptococcus by the small size of their colonies [3]. The urogenital, oropharyngeal and digestive tract microbiota are the potential entry points to systemic infections of these microorganisms, as is the case of our patient, whose probable focus of entry to the pericardium was bacteriemia secondary to the esophageal lesion that presented

Although SAG microorganisms are highly virulent, they produce rapidly progressive infections and have the ability to cross tissue planes being highly prone to abscess formation, their pathogenic mechanisms are not yet well known due to both the difficulty of its identification as well as the nature commensal of it [4]. Through in vitro models, mixed anaerobic infections with SAGs have been shown to potentiate the growth of the latter. In addition, its great ability to express adhesins facilitates its adhesion and colonization of the different substrates of the host. Other factors of virulence are the ability to bind to fibronectin through a cell binding protein, its polysaccharide capsule which hinders phagocytosis, the presence of laminin binding proteins, the occasional production of intermedilysin (B-hemolytic protein) and the production of a wide variety of hydrolytic enzymes such as hyaluronidases, deoxyribonuclease and chondroitin sulfatase [3,5].

Regarding the formation of abscesses, an important relationship has been found between SAG and human polymorphonuclear leukocytes (PMN), which, although not adequately identified, has been associated with a decrease in chemotaxis and in capacity of the PMN to destroy the SAG. In addition, the production of hydrogen sulphide by the enzyme L-cysteine desulphurase leads to the production of sulfahemoglobin, methemoglobin, erythrocyte lysis and the increase in abscess formation [5].

In general, SAG infections have a high mortality despite being diagnosed early (15-30%) [3]. The involvement of these microorganisms must be suspected especially in male patients (83%) or with a history of periodontal disease, alcoholism, cancer, HIV, chronic obstructive pulmonary disease and cystic fibrosis [4]. In a study carried out at the Hospital Universitario La Samaritana, it was found that the mean age of presentation was 53 ± 14 years, with an incidence of 37% of polymicrobial infections and a mortality rate of up to 20% [6].

Almost all SAG strains are susceptible to penicillin G, with 80-90% of these strains having a minimum inhibitory concentration (MIC) of 0.125 mcg / ml, but some cases of penicillin G resistant strains have been described (CMI> 4 mcg / ml), which is reported in up to 9% of some series. All SAGs have innate resistance to bacitracin and nitrofurantoin. Mechanisms of resistance to beta-lactams are acquired through the horizontal transfer of genes between streptococci, which has also been described with erythromycin, clindamycin and lincomycin with an incidence of up to 12-14%. Sensitivity to cephalosporins is variable in the different series, having an adequate sensitivity to the first generation cephalosporins, variable with those of second generation and good with third generation parenterals, reaching a susceptibility up to 100% and being variable the activity of third generation oral cephalosporins. Fourth generation cephalosporins have a susceptibility profile of up to 98% [7].

It has been suggested to initiate treatment with beta-lactams such as penicillin G or cephalosporins, but in the face of increasing resistance to penicillin G, combinations of B-lactams and aminoglycosides type gentamicin have been suggested as initial therapy. In addition to antibiotic therapy, treatment should always be accompanied by drainage of abscesses [3].

In the world, only one case of pneumopericardium by S. milleri has been reported in an adult, without antecedent and in whom the clinical course was different to the present report, since in our case the development of the condition was subacute, did not present manifestations of cardiac tamponade and the fever was not constant [2]. There are other aspects that call attention to the diagnosis and the clinical outcome when compared with the case published in Spain; in the first place, the diagnosis was made by computerized axial tomography and the echocardiogram did not provide essential information regarding the presence of air in the pericardium. Second, the worsening of the clinical picture coincides a few days after pericardial drainage, in our case it was secondary to septic shock and in the other case due to constrictive pericarditis. Regarding this last aspect, in the article published by Ryan C. Maves in his review in 2016, it is shown how after the drainage of a purulent pericarditis by S. anginosus the patient immediately develops septic shock, which coincides with what happened to the patient in this case [8]. Additionally, although we do not know precisely the initial focus of infection in our patient, the presence of esophageal lesions has been related to the development of purulent pericarditis; however, what is even more striking is the high mortality when the possible focus is an esophageal lesion, of five cases of purulent pericarditis due to S. anginosus that had associated esophageal carcinoma, all five patients died [8].

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