

Primary Purulent Pericarditis due to *Staphylococcus Aureus* Methicillin-Resistant in HIV Infected Patient: A Case Report and Review of the Literature

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Abstract

Purulent pericarditis is rarely primitive. It is commonly the spread from an adjacent focus of infection of neighbourhood, pneumonia or empyema particularly. The hematogenous contamination arises most of the time on state of Immuno suppression which it would be necessary to look for if no risk factor is detectable initially. We report a case of purulent pericarditis due to Community Acquired - *Staphylococcus Aureus* Methicillin-Resistant (CA - MRSA) at a patient with type 2 diabetes and HIV infection.

Keywords: Purulent Pericarditis; *Staphylococcus Aureus*; Methicillin-Resistant; HIV infection

Introduction

The purulent pericarditis is a rare but severe and sometimes fatal pathology. The mortality linked at the risk of tamponnade, can reach 30% [1].

The purulent pericarditis is rarely primitive. It is most of the time the complication of the infection of neighborhood [2].

During *staphylococcal sepsis*, the secondary localizations are frequent. The cardiac attainment is noted in 8 to 25 % of the cases and it is essentially endocarditic [2].

We report a case of peculiar sepsis, individual by its localization to the pericardium, its hematogenous contamination, the nature of the responsible germ Community Acquired - *Staphylococcus Aureus* Methicillin-Resistant (CA - MRSA) and especially the later discovery of an infection HIV in a patient with type 2 diabetes.

Case report

A 66-year-old Woman, diabetic type 2 with preexisting infected cyst mammary and fistula to the skin, was received in the emergency care unit of the Central Hospital of the Army

Mohamed Seghir Nekkache of Algiers. She presented a clinical signs of tamponnade with altered general state. A cardiac ultrasound was realized and found a pericardial effusion of big abundance. A surgical drainage was practiced and a purulent liquid was obtained and sent to the microbiology laboratory. The microscopic examination after coloring in the methylene blue showed very numerous altered polymorphonuclear neutrophils and bacteria in the form of cocci isolated and grouped in heap. The culture was practiced on simple media and enriched in the blood.

After 18 hours of incubation, yellowish colonies had appeared. The identification was made by the classic bacteriological methods: microscopic examination after coloring of Gram, test in the catalase, research for the coagulase and test of agglutination (PASTOREX™ STAPH- PLUS for BIO-RAD), finding a strain of *Staphylococcus aureus*. The antibiotic susceptibility of the isolate was realized and interpreted according to the recommendations of the Clinical and Laboratory Standards Institute (CLSI) on 2011. We noted a resistance to cefoxitin implying a resistance of oxacillin and all the beta lactam agents. The MIC of the oxacillin was 4 µg/ml. This strain was also resistant to kanamycin, Erythromycin, Clindamycin and Fusidic acid. The MIC of vancomycin was 1.5 µg / ml and this isolate was considered sensitive according to the standards of the CLSI on 2011.

The molecular study by a technique of PCR confirmed the Methicillin-resistance. This strain harboured the gene *mec A*, the *agr* type III and produced the leucocidine of Penton - Valentine (PVL). The patient was treated with vancomycin by intravenous way in the dose of 3 gms in the daytime. A clinical improvement was quickly obtained.

After five days of hospitalization, the patient presented arthritis of the right elbow and the knee with a fever in 38°C. In the articular draining, a purulent liquid was obtained. The direct examination showed altered polymorphonuclear neutrophils

but the culture was negative. Ciprofloxacin was indicated, justified by the good osseous distribution of fluoroquinolones. Seen her favorable evolution, the patient had gone out in the 15th day with fixed immobilization of 02 joints and stoppage of the vancomycin. The treatment by the ciprofloxacin was maintained for 03 months.

Six months later, the patient was readmitted for respiratory distress syndrome with altered general state. The radiography of lungs showed images of atypical pneumonia evoking a pneumocystosis and motivating a request of serology HIV. Two tests HIV 1 and 2, realized by 02 different ELISA (Enzygnost VIH Integral II Siemens R-Genscreen HIV Ag-Ac BioradR) gave positive results (DO out of range). A Western blot (New lav blot 1 BioradR) HIV 1 was made on a second serum and confirmed the infection by HIV 1 (all the proteins HIV 1 were found).

Discussion

The initial diagnosis retained at our patient was a sepsis with pericardial and articular localization. The site of entry would be a staphylococcal cutaneous infection of mammary localization. Clinically, no risk factor has been found at this patient's except the diabetes. But it was not a being argument enough for explaining the pericardial localization. No biological exploration in search of an Immunosuppression (serology HIV or other one) was made before the episode of the lung infection.

The primitive infection of the pericardium is rare and the purulent pericarditis is most often occurs as a direct extension of infection from an adjacent tissues (pneumonia, pleural, mediastinal or diaphragmatic) [1]. It can result from a penetrating trauma of the pericardium or complicate cardiac surgery and also be of hematogenous origin from a distant infectious focus (otorhinolaryngology especially at the child) [1,2].

Streptococcus pneumoniae, *Haemophilus influenzae* and *Neisseria meningitidis* are bacteria the most frequently incriminated [1].

The infection by the HIV increased the incidence of pericarditis [3]. In African series, it is estimated from 25 to 35 % [3]. For etiologies, the tuberculosis is the most frequent, but viruses (Cytomegalovirus CMV) or parasites (*Cryptococcus neoformans*) can be incriminated [3, 4].

Six cases of pericarditis due to *S.aureus* at patients affected by HIV were the object of publications. In every case the bacteriological proof is brought by the culture of the pericardial fluid. The site of entry was not identified in the majority of the cases, suggesting the responsibility of the nasal portage [4,5].

Some authors also evoke as risk factors a cutaneous infection or a use of intravenous catheter [5]. For our patient, the existence of a cutaneous infection was considered as a possible site of entry.

Furthermore, at our patient the diabetes and the hyperglycemia could be factors stressing the general and local Immunosuppression and favoring the microbial multitude. All the patients had presented pneumonia to *Pneumocystis carinii* or to CMV, sarcomas of Kaposi before or after the episode of the

pericarditis. For the evolution, 3 patients died from pneumonia, other one of a tamponnade. Our patient had also died further to a pneumonia.

It seems that in every case reported, the HIV infection was already known, contrary to our patient where the pericarditis arose while the HIV infection was not diagnosed yet.

For the strains of *S. aureus*, one was Methicillin-resistant, another Methicillin-sensible and without precision for the other cases.

Three other cases of pericarditis due to *S. aureus* not bound to an HIV infection were also published. The first was reported by Charles O. Onyeama and al, at a child in malnutrition [1], the author connects the pericarditis with a lung infection. The second case is reported by M. Schouten in the patient with a tubercular calcified pericarditis. He was working in a slaughterhouse [6]. The author supposes an iatrogenic origin by direct inoculation during a pericardiocentesis. The strain was a typical SARM t011 animal-derived, of which the patient was carrier [6]. The third case is described by L. Gillet and all at a child having presented simultaneously a pneumonia and empyema [7]. In this case, the authors suppose that it is the first strain (CA-MRSA) isolated from pericardium producing PVL [7]. Our strain would be the second one.

S. aureus is one of the main pathogenic agents for the man. It possesses an impressive arsenal of factors of virulence [8]. The most characteristic both genetic elements of the CA-MRSA are the presence of the genes coding the PVL, the nature of the chromosome cassette which carries the resistance in beta lactam agents of type IV more rarely V but never of type I, II or III privilege of the hospitable MRSA [8]. The CA-MRSA producing PVL is responsible for cutaneous infections in 90 to 95 % of the cases, sometimes of pyomyositis and osteomyelitis as well as for other invasive locations such as the necrotizing pneumonias and the severe sepsis [9]. In Algeria, the highest rate of infection by producing strains of PVL reached 67.2 % [10].

HIV infected patients are recognized as one of these higher risks groups due to increased rates of both MRSA colonization and infection [11]. The organism's interactions and disease manifestations with the immunocompromised host are expected to be complex and involve defects in innate immunity. Chemo taxis, neutrophil phagocytosis and intracellular killing are vital steps in host defense against *S. aureus* [11]. Some studies showed significant dysfunctions concerning these elements in persons with HIV [11].

The treatment of the purulent pericarditis has to associate prematurely an antibiotic treatment and a surgical drainage [3]. The pericardial constriction is a rare complication, it is unpredictable and of arisen insidious after variable deadline going of a few weeks in several years [3]. A late diagnosis and treatment are two main predictive factors of this complication. Some authors think that the risk is particularly higher if the responsible germ is *S. aureus* or *Haemophilus influenzae* and

they propose a pericardiectomy or the association of a local fibrinolysis [3]. To avoid this complication, regular ultrasound checks are indicated.

Conclusion

The CA- MRSA is more frequently incriminated in invasive infections committing seriously the prognosis for survival. Its isolation from a pericardial liquid testifies of its virulence and its important pathogenic power. During the *staphylococcal* infections, the risk of appearance of secondary infectious focus of more marked gravity, especially in the presence of factors of comorbidity (cancer, chronic pathology, state of Immunosuppression), requires a rigorous coverage of these infections. But, on the other hand, for an unusual localization of this germ, it would be imperative to look for a state of Immunosuppression, even latent, in particular an infection HIV.

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