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Case Report
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Pleuropulmonary Staphylococcal Disease in Young Immunocompetent Man: A Case Report

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Introduction

Staphylococcal pleuropulmonary disease (SPP) is an infectious pathology caused by *Staphylococcus aureus*, a germ responsible for a wide range of suppurative, toxigenic and multifocal infections (cutaneous, osteoarticular, pulmonary, etc.).

This entity is most often associated with staphylococcal acute community-acquired pneumonia, the prevalence of which remains low at 2-5% [1]. It mainly affects the elderly, people living in institutions or with co-morbidities. Recent studies have shown that the epidemiological profile of this infection is rejuvenated by the characteristics of certain strains of *Staphylococcus aureus*, notably those that secrete a synergohymenotropic toxin known as Panton-Valentine Leukocidin (PVL) [1].

We report a case of pleuropulmonary staphylococcal disease in a young immunocompetent subject with a cutaneous onset and a favourable course.

Case Presentation

The patient is a 30-year-old Moroccan farmer. He has been exposed to passive smoking and pesticides since the age of 18. He has a history of tuberculosis of the left cervical lymph nodes, treated at the age of 23. He has no other comorbidities and no toxic habits.

The patient presented with minimal dyspnoea, progressing over 20 days to dyspnoea at rest, a productive cough with copious purulent sputum, bilateral side-tipped chest pain, general ill-being and feverish sensations, for which he self-medicated with paracetamol before initially consulting the emergency department, where a chest X-ray and CT scan were performed. The initial chest X-ray showed a bilateral interstitial syndrome with multiple hydroaerosic images (Figure 1), and the injected chest CT revealed multiple bilateral excavated masses and nodules with minimal right pleural fluid effusion (Figure 2). A probabilistic antibiotic therapy was indicated (amoxicillin clavulanic acid 3g/d), but the clinical course was not favourable.

On somatic examination, the patient is eupneic, with room air saturation at 95%. He is tachycardic at 123 beats per minute. All other vital signs were normal. On respiratory examination, bilateral crackles are audible, predominating in the lower half of the right thorax, where there is a cavitary murmur. Skin examination reveals crusty, impétigineous lesions on the right labial commissure and upper lip (Figure 3). These skin lesions may be associated with manipulated herpes labialis. The rest of the clinical examination was normal. Chest X-rays taken three days apart showed an extension of the radiological lesions (Figure 4), and thoracic ultrasound revealed a heterogeneous

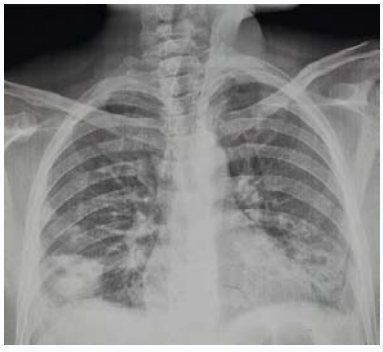

Figure 1: Initial chest x-ray.

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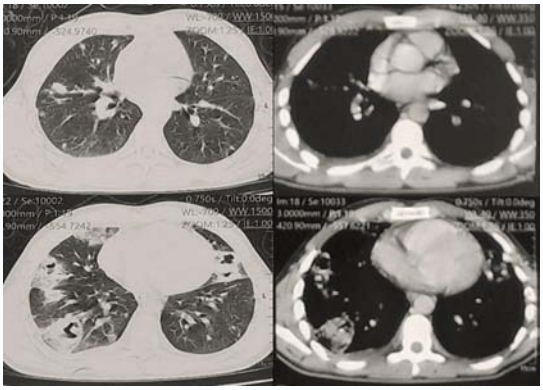

Figure 2: Initial thoracic CT scan.

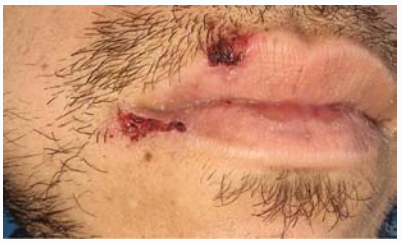

Figure 3: Manipulated facial herpes.

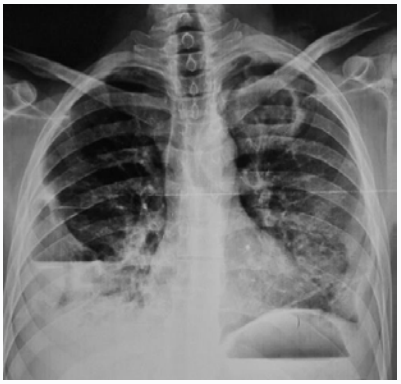

Figure 4: Control chest x-ray.

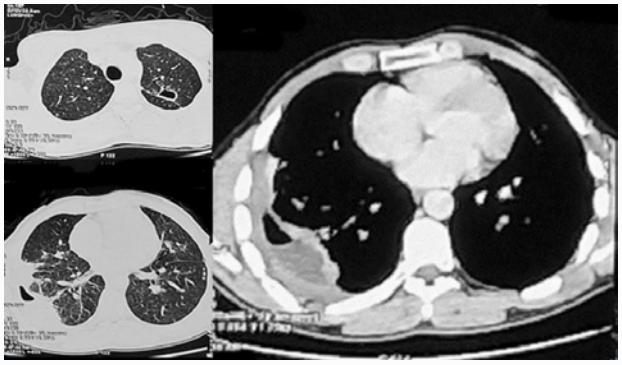

Figure 5: Control thoracic CT scan.

Antibiogramme des cocci gram positives	*Staphylococcus aureus*
Cefoxitine	Sensible
Gentamicine	Sensible
Tobramycine	Sensible
Kanamycine	Sensible
Erythromycine	Sensible
Clindamycine	Sensible
Triméthoprime – sulfaméthoxazole	Sensible
Ciprofloxacine	**Sensible Forte Posologie**
Lévofloxacine	**Sensible Forte Posologie**
Tétracycline	Sensible
Acide fusidique	Sensible
Tigécycline	Sensible
Chloramphénicol	Sensible
Rifampicine	Sensible
Marqueurs épidémiologiques	SARM négative

Figure 6: Bacteriological study of bronchial aspirates.

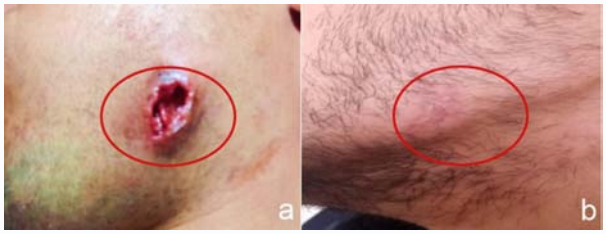

Figure 7: Manipulated furuncle of the face.
a) Before hospitalization. b) During hospitalization

right pleural fluid effusion of low abundance and multiclonality, with no discernible aeriform component.

At this stage, the infectious origin takes precedence over the neoplastic or inflammatory origin, which were possible but less probable.

The initial blood count showed a major hyperleukocytosis of 35,420/mm^3, mainly neutrophils at 31,063/mm^3. Other blood lines were normal. Initial CRP was elevated to 211 mg/l. Bacteriological study of sputum did not detect any common or specific germs. Liver function tests showed hepatic cytolysis syndrome (ALT: 169 ui/L - AST: 87 ui/L). Viral hepatitis serologies and abdominal ultrasound were normal.

In view of the hepatic cytolysis, the initial antibiotic therapy was discontinued and replaced by Levofloxacin (500 mg/d) for 21 days, with close monitoring of hepatic cytolysis. The patient was also put on chest physiotherapy for bronchial drainage and control of pachypleuritis, along with hygienic and dietary measures. Sputum curves and body temperature were also monitored. Eight days after treatment, chest CT showed partial radiological clearance, with the appearance of a left upper lobar cavitary lesion and persistent pleural effusion on the right (Figure 5).

Flexible bronchoscopy showed diffuse bronchial inflammation with abundant purulent secretions from the right bronchi, and bacteriological study of bronchial aspirates revealed a methicosensitive *Staphylococcus aureus* infection (Figure 6). To reinforce antibiotic therapy and with a normal renal balance, levofloxacin was combined with gentamycin 6 mg/kg/dr for 5 days.

Field work-up including fasting blood glucose, HbA1c, retroviral serologies, plasma protein electrophoresis, NAA and locoregional work-up (flexible bronchoscopy, abdominal ultrasound and cardiac ultrasound) was normal.

Although manipulated herpes labialis appears to be the most likely portal of entry for *Staphylococcus aureus* in the patient, it

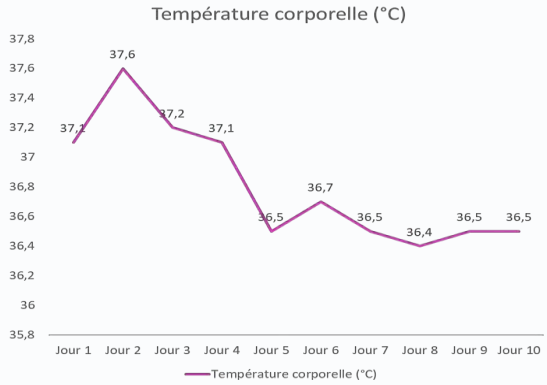

Figure 8: Body temperature curve.

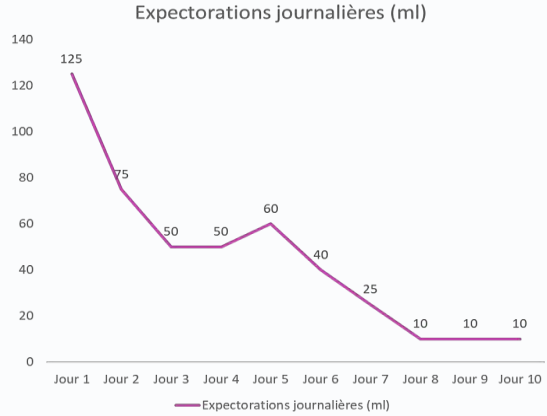

Figure 9: Sputum curve.

appeared one week after the onset of respiratory symptomatology, so its incrimination is unlikely. After a complete shave of the face, skin examination showed a discrete scar from a manipulated furuncle of the face dating back to a few days before the onset of symptoms (Figure 7). In the patient's case, this was the initial portal of entry for *Staphylococcus aureus*. The patient was educated about the risks of self-medication and the importance of respecting all skin lesions.

The clinical course was favorable. The patient had no febrile spikes during hospitalization (Figure 8), and sputum had dried up (Figure 9). Biological evolution was marked by progressive improvement of the inflammatory syndrome (Figure 10) and of the hepatic cytolysis syndrome (Figure 11). Radiological evolution showed progressive radiological clearance, with complete radiological clearance at 1 year (Figure 12).

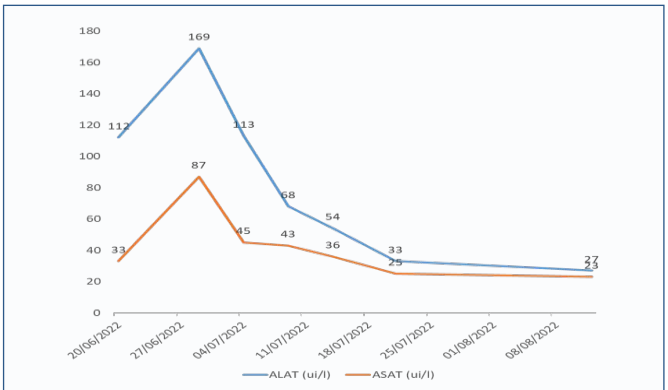

Figure 11: Progression of the hepatic cytolysis syndrome.

Discussion

Staphylococcus aureus is a pathogen responsible for a wide range of life-threatening osteoarticular, cutaneous, cardiac and pulmonary infections. It is also a commensal germ found throughout the world, colonizing mainly the skin and nasal cavities. It is estimated that only 20% of the human population are never carriers [2].

The widespread use of antibiotics is responsible for the emergence of resistant strains, altering the epidemiological spectrum of staphylococcus, which since the 1990s has been responsible for epidemics of mainly skin or healthcare-associated infections, largely due to methicillin-resistant strains (MRSA) [3].

Staphylococcus aureus derives its pathogenicity from its membrane and intracellular components. The membrane microcapsule enables it to escape phagocytosis, while surface proteins known as adhesins enable it to attach to cell membranes and the extracellular matrix (MSCRAMM). Certain elements, such as toxins and coagulase, are responsible for the distant dissemination of septic foci. These are responsible for the formation of a staphylo-thrombinic complex which, under the action of staphylokinase, enables the staphylococcus to spread at a distance. Panton-Valentine Leucocidin (PVL) is unique

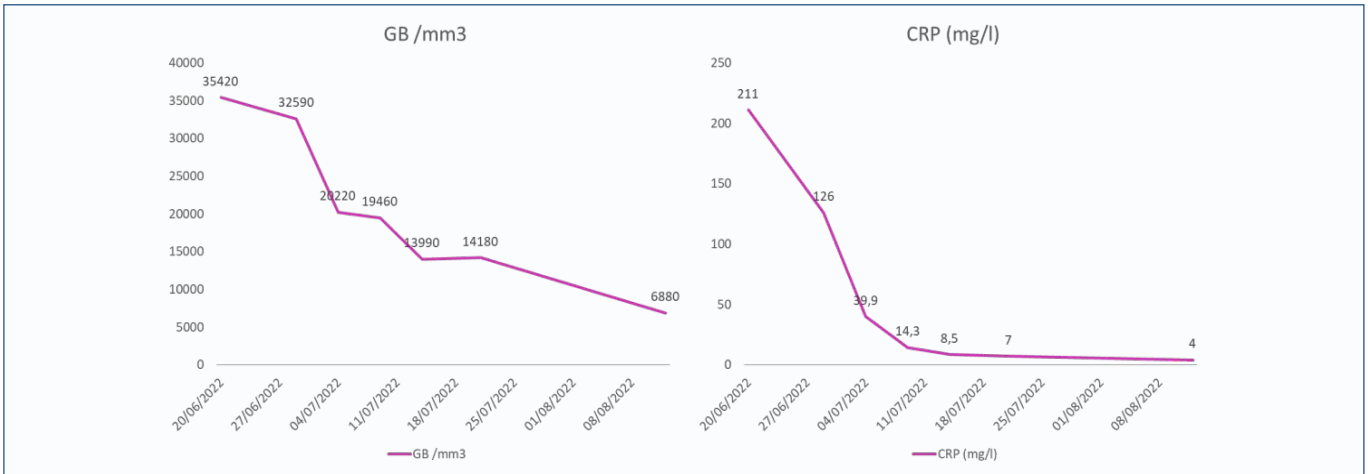

Figure 10: Inflammatory syndrome evolution.

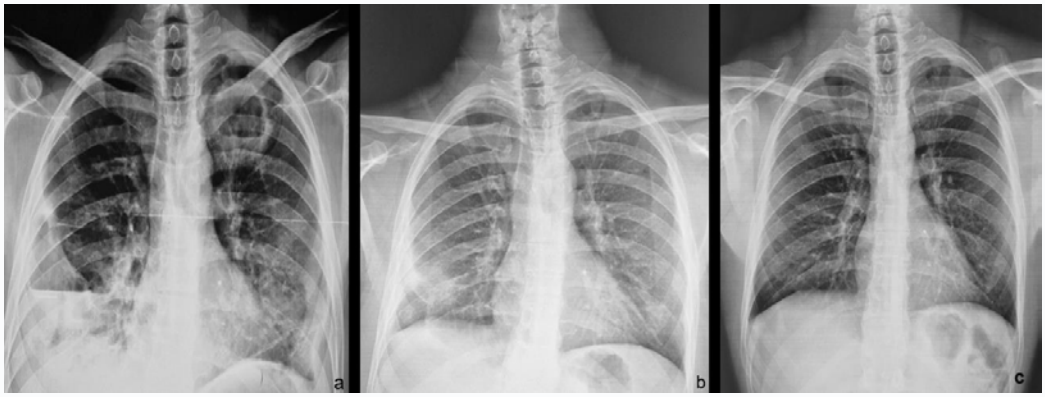

Figure 12: Radiological evolution: a) on admission - b) at 1 month - c) at 1 year.

in that it is secreted by only 5% of *Staphylococcus aureus* strains, most of which are sensitive to methicillin. It targets immune cells and is responsible for a rapidly progressive and fatal toxic-infectious picture in patients with no risk factors [4].

Staphylococcal community-acquired pneumonia mainly affects the elderly, people with co-morbidities or those living in institutions, sometimes with a recent respiratory viral episode. The clinical picture is characterized by a purulent bronchial syndrome, variable temperature and side-tipped chest pain. Involvement is often unilobar, but may be diffuse in cases of hematogenous dissemination, whether or not associated with endocarditis. Complications are mainly local (empyema, pulmonary abscess, etc.) and the mortality rate is low. Treatment is not codified, but is largely based on Penicillin M for methicillin-sensitive strains (MSSA). The duration of antibiotic therapy depends on the form (simple or complicated) and varies from 14 days to 6 weeks. Combination with clindamycin (1800mg to 2400 mg/day) is indicated from the outset in complicated forms, notably purulent pleurisy. In acute community-acquired MRSA pneumonia, treatment is based on a range of antibiotics as monotherapy in uncomplicated forms (vancomycin, teicoplanin, linezolid, etc.). In complicated forms due to MRSA strains, dual antibiotic therapy is indicated, with clindamycin at the same dose combined with vancomycin, teicoplanin or ceftaroline [5].

Panton-Valentine Leucocidin (PVL)-secreting strains are responsible for noisier staphylococcal community-acquired pneumonia, and can affect young subjects without comorbidities, with a recent respiratory viral episode found in 75% of cases. The clinical picture is characterized by fever to 39°C, tachycardia > 140 beats/min, arterial hypotension, hemoptysis (sometimes major), and a skin rash indicative of toxin shock. Involvement is often multilobar, frequently associated with pleural effusion. The course is often unfavorable, with rapid deterioration progressing to septic shock and acute respiratory distress, with a high mortality rate > 60%. The therapeutic arsenal is available at various levels, notably selenium for its antioxidant role in septic shock, and low-pressure alveolar recruitment, without increasing lung damage, essentially by means of ventral decubitus, whereas the use of extracorporeal oxygenation remains anecdotal to date, with only moderate efficacy. The use of oseltamivir remains debatable, given the absence of a specific epidemiological profile of subjects at risk. Antibiotic therapy does not rely exclusively on penicillin M, as beta-lactam antibiotics are less effective in the stationary growth phase of staphylococci, when LPV is secreted. A combination of penicillin M and clindamycin is indicated for methicillin-sensitive strains. For MRSA strains secreting LPV, antibiotic therapy is based on a combination of clindamycin and vancomycin or ceftaroline. The duration of treatment has not been codified, but a minimum of 14 days is recommended [5].

The patient is immunocompetent and has no risk factors for developing pleuropulmonary staphylococcal disease, but the favourable evolution under antibiotic therapy makes the association with LPV secretion highly unlikely, although the diagnosis of Panton-Valentine Leucocidin-secreting strains relies on PCR. This medical observation of pleuropulmonary staphylococcosis in a young, immunocompetent subject proves that this pathology can also affect subjects with no comorbidities and no risk, and that it can be responsible for necrotizing and extensive lesions, with a mortality rate that can be avoided by prompt and appropriate management. *Staphylococcus aureus* often enters the skin via a lesion that has often been handled and superinfected, which is why it is so important to educate patients and the general public about the septic risks of handling any skin lesion.

Conclusion

Acute community-acquired pneumonia caused by *Staphylococcus aureus* is rare. Although the pathophysiology of staphylococcal infection is better understood thanks to recent scientific advances that have improved its therapeutic management, certain difficulties remain. These difficulties are linked to the emergence of resistant strains and Panton-Valentine Leucocidin-secreting strains, which are correlated with a particular epidemiological, clinical and biological presentation, and a poor prognosis. Acute staphylococcal pneumopathy concerns a spectrum of at-risk subjects, and pleuropulmonary staphylococcosis mainly affects infants. However, staphylococcal pneumonia can also occur in young, immunocompetent subjects with no risk factors.

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