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Case Report

Multiple Nodular Condensation from Peripheral Excavations Revealing Septic Thrombophlebitis of Jugular Vein: Think of Lemierre Syndrome

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ABSTRACT

Lemierre syndrome is a rare and unknown disease. It is defined as a septic thrombophlebitis of the internal jugular vein or one of its collaterals causing multiple septic pulmonary embolus.

It complicates an otorhinolaryngology infection of bacterial origin allowing the hematogenic invasion by anaerobic bacteria, in most cases Fusobacterium necrophorum.

The advent of antibiotics has drastically reduced the incidence and mortality of this disease.

The aim of our work was to recall the pathophysiology, clinical and treatment of this syndrome from a case treated in our department and a review of the literature.

Keywords: Lemierre syndrome; Necrophorum; Hematogenic invasion; Pathophysiology; Otorhinolaryngology infection

Introduction

Lemierre syndrome is defined as septic thrombophlebitis of the internal jugular vein (VJI) or one of its collateral causes septic embolus, following an otorhinolaryngological infection of bacterial or viral origin, allowing the invasion of blood by an anaerobic bacterium, in most cases Fusobacterium necrophorum[1]. The advent of antibiotics has drastically reduced the incidence and mortality of this disease. Currently, due to its rarity, it is a little-known pathology. The aim of our work was, from a case treated in our department and a review of the literature, to recall the pathophysiology, the clinic and the treatment of this syndrome.

Observation

Farid T, 30 years old, is hospitalized for infectious table with chest pain as part of a red angina treated by macrolides for 6 days. He has smoked 1 pack of cigarettes per day for 15 years and occasionally cannabis, has been intoxicated for 5 years and has a history of right pneumothorax in 2011 and a true allergy to pencilline the patient has clinical criteria for severity with systemic arterial hypotension (90/50mmHg) and respiratory rate at 27/min. Heart rate is 93/min, oxygen saturation at 92% in ambient air and temperature at 38.5 C.

The clinical examination finds chest pain and a productive cough. There is a discreet right jugular venous induration at cervical palpation. The oral cavity examination shows inflammation of the tonsillar region without phlegmon. Neurological examination is strictly normal **(Figures 1,2 and 3)**.

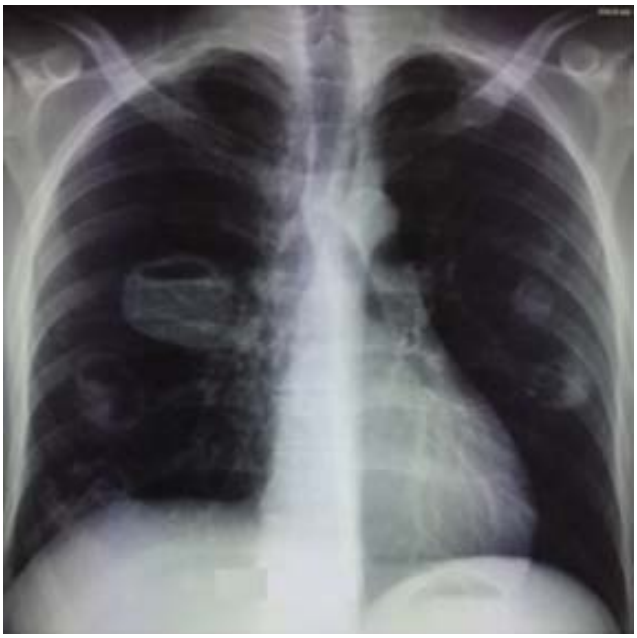

Figure 1: Chest radiograph at admission shows multiple bilateral opacities in the exclave.

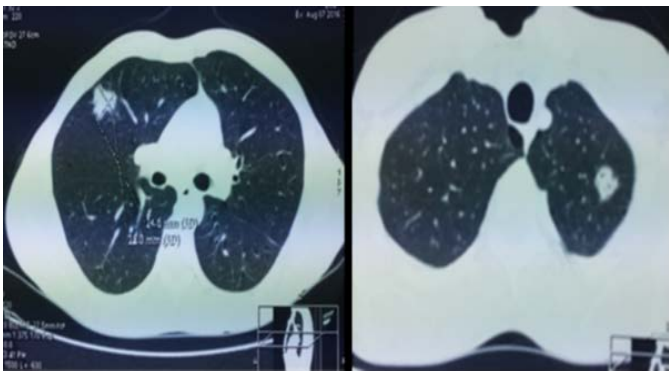

Figure 2: TDM thoracique: multiple bilateral pulmonary nodules ecaves, emphysemous lung.

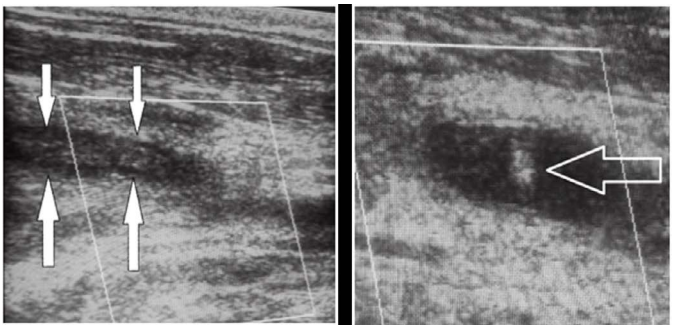

Figure 3: Echo-doppler cervical: Extensive thrombosis of the right jugular vein and multiple cervical adenopathies.

Biological balance: infectious and inflammatory syndrome.

Normal blood gas.

Normal clotting test.

HIV serology negative.

In contrast, an anaerobic GRAM-negative bacillus, identified as Fusobacterium necrophorum, grows in one in four blood cultures.

The patient received anticoagulant treatment and a switch to anitivitamine K and probabilistic intravenous bi-antibiotic therapy combining Cefotaxim and Gentamicine the first two days. Following the results of the additional examinations that point towards a diagnosis of Lemierre syndrome, this treatment is modified as follows: Ciprofloxacin in IVL + Flagyl. The patient's condition improves with apyrexy after day 5. A Doppler ultrasound of the neck vessels shows the persistence of extensive thrombosis of the right jugular vein. The inflammatory syndrome and patient's condition are significantly improved after 21 days of intravenous bi-antibiotic therapy.

The patient returned home after 21 days of hospitalization, with a per os relay by Ciprolon and anticoagulant treatment by vitamin K.

The radiological evolution was favorable **(Figure 4)** but a widespread thrombosis of the right jugular vein persisted.

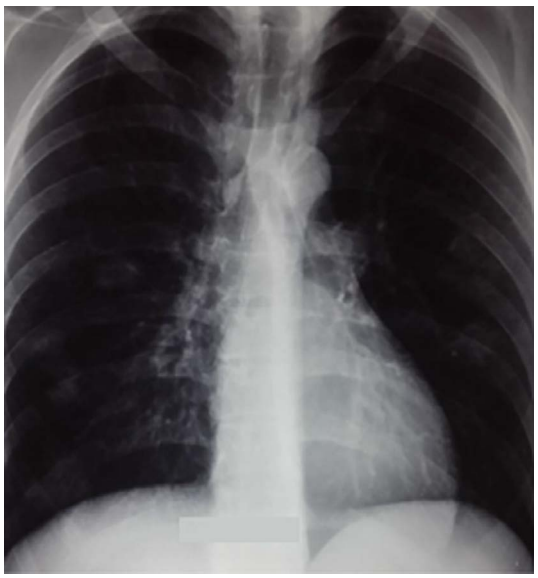

Figure 4: La radiographie thoracique à la sortie.

Discussion

Lemierre syndrome is a rare entity, described since 1936 and affecting mainly young healthy adults[2]. Its low incidence and the radical change in its prognosis since the advent of antibiotics have made it a lesser-known pathology. Nevertheless, its acute character and the risk of serious septic complications invite to make the diagnosis quickly[3].

The clinical picture includes an oropharyngeal, dental, otologic, mastoid or laterocervical infectious starting point. The association with pleuropulmonary manifestations and anaerobic germ sepsis should alert the practitioner. Cervical, ultrasound or CT imaging shows the thrombosis of the internal jugular vein, a key element of the syndrome. Our observation finds a splenomegaly that we can link to this syndrome.

Metastatic infectious sites have also been reported, with a lower frequency, at the cerebromeninged, hepatic, osteo-articular, renal or cardiac level.

On the pulmonary plane, computed tomography finds peripheral nodular condensations of variable size, sometimes excavated, corresponding to disseminated septic embolus. Pleural effusions or pneumothorax are common. The effect on respiratory function may be minimal as in our observation or become much more severe with acute respiratory distress syndrome leading to assisted ventilation.

The detection of the responsible germ can take several days or even weeks due to a 4-day incubation and a difficult culture:

Fusobacterium necrophorum, commensal Gramnegative bacillus from the pharynx, is isolated in hemocultures in about 70% of cases. Other anaerobic germs, fusobacteria or others, are more rarely involved. In any case, antibiotic treatment should be early, active on anaerobic germs and preferably use Metronidazole or Clindamycin given possible resistance to Penicillin.

In our case, even if the antibiogram did not reveal any resistance, it is probably the notion of allergy to penicillin that allowed the installation of such a table. Thus, the early and systematic treatment of any oropharyngeal infectious outbreak by Penicillin +/- b lactamase inhibitor explains the epidemiological changes observed.

In addition to antibiotic treatment that will continue for 3-6 weeks, surgical drainage or ligation of the internal jugular vein is rarely required and only affects advanced sepsis. Anticoagulant treatment has not been proven effective, but is usually initiated for a duration of 3 months by many authors. The peripheral nature of the lesions explains the painful component of pulmonary symptoms. An adapted analgesic therapy and respiratory physiotherapy to combat pleural sequelae are part of a comprehensive management of the disease.

Fatal 9 times out of 10 before the age of antibiotics, Lemierre syndrome is now a much better prognosis if treatment is early. The hope of complete recovery is therefore very real and confirms the interest of a rapid diagnosis based on the association of oropharyngeal infectious signs with pulmonary symptoms[4].

Conclusion

Currently, Lemierre syndrome is a rare and unknown pathology. This is a serious disease that develops in two phases: a pharyngeal infection followed by a cervical infection causing septic thrombosis of VJI or one of its branches, which itself causes remote septic embolus, usually pulmonary. Any delay in diagnosis and treatment leads to increased morbidity and mortality. The most common germ is Fusobacterium necrophorum.

The essential paraclinical examinations to be carried out are blood cultures and the cervico-thoracique scan injected. Treatment is primarily medical and based on extended broad-spectrum antibiotic therapy. The role of anticoagulant is not well defined. Surgery is indicated for pharyngeal, cervical or mediastinal abscesses. JIV ligature is currently exceptional[5].

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