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Case report

A case of Lemierre syndrome caused by *Staphylococcus aureus* and complicated by a superior vena cava thrombophlebitis

Arnaud Salami, Romaric Beheton*, Camille Assouan, Arsène Coulibaly

Maxillofacial Surgery Department, Teaching Hospital of Treichville, BPV3 Abidjan, Côte d'Ivoire

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ABSTRACT

Lemierre syndrome (LS) is a rare disease characterized by a septic thrombophlebitis of the internal jugular vein, generally due to *Fusobacterium necrophorum*. We report a rare case of LS due to methicillin-susceptible *Staphylococcus aureus* and complicated by a superior vena cava (SVC) thrombophlebitis in a 52 years old patient. The treatment associated antibiotics, anticoagulants and surgical debridement of the necrotic tissues. The outcome was favorable.

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1. Introduction

Lemierre syndrome (LS) is a rare disease, first time described in 1936 by André Lemierre. It is characterized by a septic thrombophlebitis of the internal jugular vein generally due to *Fusobacterium necrophorum* [1]. We report a rare case of LS due to methicillin-susceptible *Staphylococcus aureus* and complicated by a superior vena cava thrombophlebitis.

2. Case report

A previously healthy 52-year-old woman presented with a right cervical skin necrosis, extensive which had begun six days earlier. She reported an accidental swallowing of fish bone 12 days ago, causing odynophagia. She started a self-medication with diclofenac 50 mg capsules (one capsule three times per day) and amoxicillin 500 mg capsules (one capsule three times per day). Odynophagia worsening and a right cervical necrosis occurred. She had no cardiovascular disease before. At admission she was conscious. Oral cavity and oropharynx examination showed a deep and painful ulceration

of the right tonsillar lodge. The ulceration was clean with jagged edges and did not bleed on contact. The right palatine tonsil was not visible (Fig. 1). We noted a purulent 6 cm² right cervical skin necrosis (Fig. 2). The necrosis was linked to the oropharynx, realizing a pharyngostoma. Cardiopulmonary auscultation, electrocardiogram and chest X-ray were normal. Blood examinations showed leukocytosis (18,000 cells/mm³ with 80% of neutrophils) and a high protein C-reactive at 68 mg/L. The cervical and thoracic scanner showed thromboses of the superior vena cava (SVC) and the left internal jugular vein. Fishbone was not viewed (Fig. 3).

Bacteriological examination of the pus isolated methicillin-susceptible *Staphylococcus aureus*, also susceptible to Ceftriaxone and Aminoglycosides. Blood culture and anaerobic culture of the pus have not isolated germs. The treatment was medico-surgical, associating antibiotics (Ceftriaxone 2 g intravenous per day for 21 days, Gentamycin 80 mg intramuscular daily for 5 days). A surgical debridement of the cervical necrotic tissues and a drainage with corrugated drain were done. A Twice daily washing by Dakin solution was realized. A nasogastric tube feeding has been established for 47 days, until the complete healing of the pharyngostoma. 4000UI enoxaparin subcutaneously every 24 h was administered for 10 days, relayed by Acenocoumarol for three months. The dosage of Acenocoumarol was regularly adapted in order to obtain an INR rate between 2 and 3. The outcome was favorable after 21 days of antibiotic therapy with disappearance of infectious syndrome. Complete healing of the oropharyngeal ulceration was observed after three months (Fig. 4). The head and neck CT scan control after three

* AsianAOMS: Asian Association of Oral and Maxillofacial Surgeons; ASOMP: Asian Society of Oral and Maxillofacial Pathology; JSOP: Japanese Society of Oral Pathology; JSOMS: Japanese Society of Oral and Maxillofacial Surgeons; JSOM: Japanese Society of Oral Medicine; JAMI: Japanese Academy of Maxillofacial Implants.

* Corresponding author.

E-mail addresses: behetonromaric@gmail.com, behetonr05@yahoo.fr (R. Beheton).

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Fig. 1. Oral view showing the ulceration of the right tonsillar lodge.



Fig. 2. Purulent right cervical skin necrosis.

months of anticoagulation therapy has not revealed thromboses (Fig. 5).

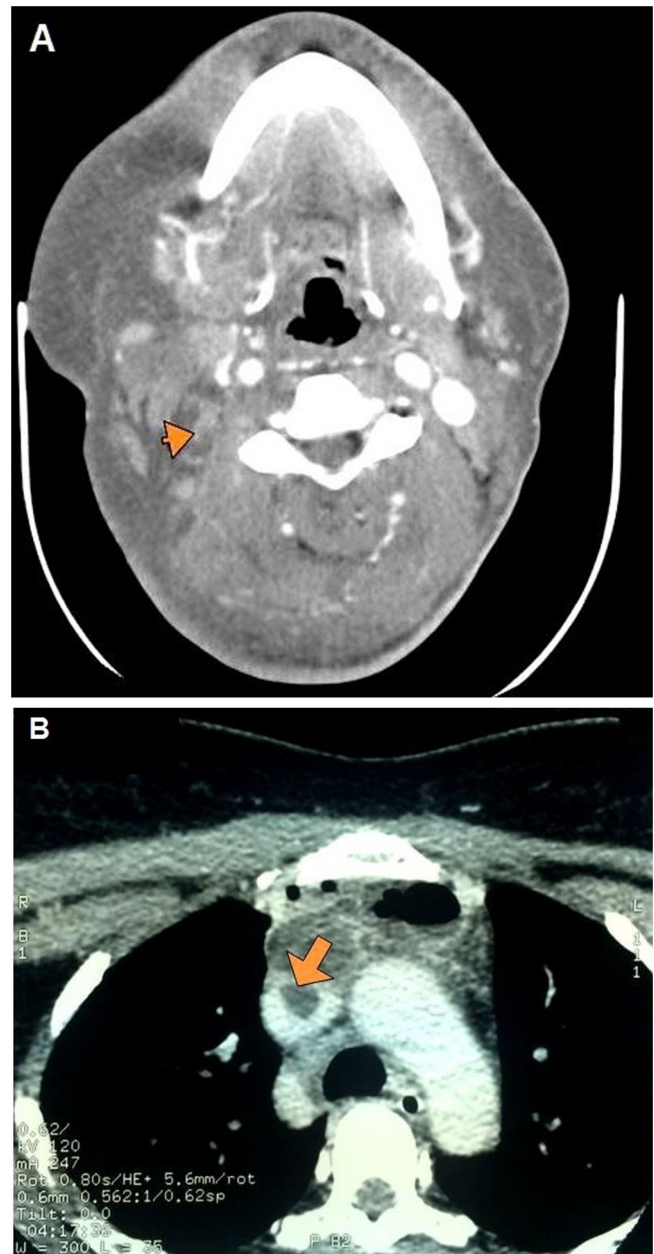


Fig. 3. (A) Cervical CT Scan (Axial section) after contrast injection: Right internal jugular vein thrombosis (Arrowhead); (B) Thoracic CT Scan: Superior vena cava thrombophlebitis (Arrow).

3. Discussion

This observation reports a case of LS with SVC thrombosis. The association of LS with SVC thrombophlebitis and *Staphylococcus aureus* has never been described in the literature. Massive inflammation, intrinsic thrombogenic and pro-inflammatory potential are features associated with this organism, responsible of altered coagulation [2–4].

S. aureus produces coagulase, which specifically interacts with fibrinogen and causes coagulation [5]. Thrombus presence in the SVC may result from the extension of the phlebitis in the SVC's wall or from the migration of septic emboli [6]. SVC syndrome was not observed in this case because of the partial obliteration of the SVC.

In this case, non-adapted self-medication and long consulting delay probably contributed to the thrombosis extension.

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