

Lemierre-like Syndrome and Infective Endocarditis: A Triumph of Eponyms

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Commentary:

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The use of medical eponyms is widespread across hospital wards and medical school halls worldwide, and it clearly indicates that the history of medicine is much more than an arid catalogue of discoveries. It is estimated that more than 8,000 medical eponyms exist¹, and a certain degree of debate has been recently generated on eponyms being potentially archaic and scientifically misleading despite being of undoubted fascination as they embed different medical traditions and cultures². At the intersection of otolaryngology, infectious disease and vascular surgery, Lemierre Syndrome (LS) was first described by Andre Lemierre in 1936 as ‘anaerobic postanginal sepsis’, consisting of septic thrombophlebitis of the internal jugular vein secondary to uncontrolled oropharyngeal infection by *Fusobacterium* species³.

In this issue of the Journal, Morishige and colleagues⁴ reported the case of an uncommon variant of this rare syndromic disease, which has an annual incidence of 3.6 cases per million people in its classic presentation⁵. The Authors presented a case which stood out under multiple standpoints:

1. First, the pathophysiology was unique. They presented a case of infective endocarditis secondary to thrombophlebitis in the left vertebral vein which was secondary to pharyngitis caused by methicillin-sensitive *Staphylococcus Aureus*. They identified this condition as a rare LS variant. Interestingly, the primary focus of infection had spread to the left ventricular wall and created a pedunculated,

encapsulated, purulent abscess attached to the trabeculae carneae of the anterior ventricular wall. Additional eponymic signs of systemic septic embolization were appreciated, namely Osler nodes (painful, red, raised lesions on extremities) and Janeway lesions (non-tender, small haemorrhagic lesions on the palms and soles), but not Roth-Litten spots (small, white-centered retinal hemorrhages) – all of which may be typical in patients with infective endocarditis.

2. Secondly, the decision-making process for optimal treatment was multidisciplinary, as suggested by the guidelines on infective endocarditis⁶. The Authors had cardiologists, infectious diseases physicians and neurologists involved in rapidly assessing the patient pre-operatively and ruling out potential contraindications to early surgery such as embolic stroke⁷. Notably, the patient was neurologically compromised at presentation with acute cerebral and cerebellar dysfunction and a Glasgow Coma Scale of 7. However, after careful evaluation of the size (<10 mm) and locations (frontal cortex, caudate nucleus, and left cerebellar hemisphere) of the hyperdense lesions at brain magnetic resonance imaging, the neurologic status was mainly attributed to sepsis-associated encephalopathy rather than septic embolization, thus allowing for prompt surgical control of the infective source.
3. Finally, the outcome was excellent. The patient underwent early surgery (transseptal abscess excision) followed by long-term antimicrobial therapy to control the primary source of infection and anticoagulation therapy to prevent left vertebral vein thrombophlebitis from recurring, with complete normalization of neurologic status at discharge and no relapsing at latest follow-up.

In conclusion, Morishige and colleagues must be congratulated for their comprehensive, collaborative, multidisciplinary efforts which led to the successful treatment of a rare and highly comorbid syndromic condition.

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