

Neurological picture. Horner syndrome due to jugular vein thrombosis (Lemierre syndrome)

Habek, Mario; Petravić, Damir; Ozretić, David; Brinar, Vesna V.

Source / Izvornik: **Journal of Neurology, Neurosurgery, and Psychiatry, 2008, 79, 293 - 293**

Journal article, Accepted version

Rad u časopisu, Završna verzija rukopisa prihvaćena za objavljivanje (postprint)

<https://doi.org/10.1136/jnnp.2007.124479>

Permanent link / Trajna poveznica: <https://um.nsk.hr/um:nbn:hr:105:867989>

Rights / Prava: [In copyright](#) / [Zaštićeno autorskim pravom.](#)

Download date / Datum preuzimanja: **2024-09-28**



Repository / Repozitorij:

[Dr Med - University of Zagreb School of Medicine
Digital Repository](#)





Središnja medicinska knjižnica

Habek, M., Petravić, D., Ozretić, D., Brinar, V. V. (2008) *Neurological picture. Horner syndrome due to jugular vein thrombosis (Lemierre syndrome)*. Journal of Neurology, Neurosurgery, and Psychiatry, 79 (3). pp. 293.

This article has been accepted for publication in JNNP following peer review. The definitive copyedited, typeset version is available online at :

<http://jppn.bmjournals.com/>

<http://medlib.mef.hr/421>

University of Zagreb Medical School Repository

<http://medlib.mef.hr/>

Horner syndrome due to jugular vein thrombosis (Lemierre syndrome)

Mario Habek, MD¹, Damir Petravić, MD¹, PhD, David Ozretić, MD², and Vesna V. Brinar MD, PhD¹

From the:

¹Referral Center for Demyelinating diseases of the Central Nervous System, University Department of Neurology, Zagreb School of Medicine and University Hospital Center, Zagreb, Croatia

²Department of Radiology, Zagreb School of Medicine and University Hospital Center, Zagreb, Croatia

Corresponding author:

Mario Habek, MD
University Department of Neurology
Zagreb School of Medicine and University Hospital Center
Kišpatićeva 12
HR-10000 Zagreb
Croatia
Phone: +38598883323; Fax: +38512388045; e-mail: mhabek@mef.hr

Word count: 357

Key words: Horner syndrome, Jugular vein thrombosis, Lemierre syndrome

Authors' contributions

Study concept and design: Habek and Petravić. Acquisition of data: Habek, Petravić, Ozretić, Brinar. Analysis and interpretation of data: Habek, Ozretić. Drafting of the manuscript: Habek. Critical revision of the manuscript for important intellectual content: Habek, Petravić, Ozretić, Brinar. Administrative, technical, and material support: Habek, Ozretić.

Conflict of interest statement: There is no conflict of interest.

The Corresponding Author has the right to grant on behalf of all authors and does grant on behalf of all authors, an exclusive licence (or non exclusive for government employees) on a worldwide basis to the BMJ Publishing Group Ltd and its Licensees to permit this article (if accepted) to be published in the Journal of Neurology, Neurosurgery & Psychiatry editions and any other BMJPGJL products to exploit all subsidiary rights, as set out in our licence (<http://jnnp.bmjournals.com/ifora/licence.pdf>).

A 60-year-old patient was referred to our Department for evaluation of anisocoria. His neurological examination revealed Horner syndrome on the right side. The rest of the examination was normal. Six months before, he had acute tonsillitis, throat cultures revealed a polymicrobial flora consisting of both aerobic and anaerobic bacteria, which was treated with 1600000 international units of bipenicillin intramuscularly for 10 days. On admission he had normal complete blood count, C-reactive protein, and chest x-ray. Throat and blood cultures were normal. Brain multi sliced computed tomography was normal. Color Doppler of carotid arteries revealed normal flow through both internal carotid arteries; however, the flow through the right jugular vein could not be recorded. Brain MRI and MRI venography records are shown in Figure 1. Neck ultrasound revealed a hypoechogenic nodule in the right submandibular gland (1.8x0.4x1.9 cm), whereas cytological analysis showed numerous neutrophils and cell debris which was consistent with pus. Numerous enlarged neck lymph nodes were present bilaterally. Treatment with metronidazole and cefuroxime axetil was initiated, with good recovery. Despite therapy, Horner syndrome persisted.

The classic presentation of Lemierre syndrome includes primary oropharyngeal, tonsillar or peritonsillar inflammation with later development of sepsis, internal jugular vein thrombosis, and septic emboli, and it is caused by *Fusobacterium necrophorum*. As a consequence of widespread antibiotic use for pharyngeal infections, the typical course of the disease has changed, so a high grade of clinical suspicion is needed to establish the accurate diagnosis [1]. The patient showed no signs of infection; his only clinical presentation was right-sided Horner syndrome. As the most feared cause of postganglionic Horner syndrome is carotid artery dissection, color Doppler of the carotid arteries was performed to reveal that there was no flow through the right jugular vein. Subsequent investigations led us to the correct diagnosis of jugular vein thrombosis due to Lemierre syndrome. As the patient had already received antibiotic therapy, the clinical picture of Lemierre syndrome was not fully developed, and

blood and throat cultures did not reveal *Fusobacterium necrophorum*. While postganglionic Horner syndrome is a well-recognized sign of a carotid dissection, it may also be an important sign of the adjacent vena cava pathology.

Reference:

1. Chirinos JA, Lichtstein DM, Garcia J, Tamariz LJ. The evolution of Lemierre syndrome: report of 2 cases and review of the literature. *Medicine (Baltimore)* 2002;81:458-65.

Figures

Figure 1. (A) Neck MRI, T1 weighted sequences showing hyperintensity with mild distention of the right jugular vein (small white arrow) compared with the lack of signal in the left jugular vein representing normal flow (big white arrow); (B) MRI venography showing absence of signal in the right jugular vein (small white arrow) with normal signal in the left jugular vein (big white arrow).

