A Blindingly Curious Case of Sepsis and Encephalopathy in a Healthy Adolescent

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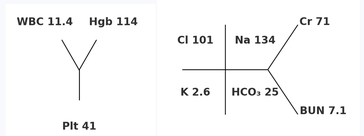
Case Presentation

- Previously healthy adolescent with 5-day history of sore throat, dry cough, diarrhea, progressive drowsiness, ataxia, urinary retention and generalized weakness.
- T 39.5 °C, BP 88/60mmHg, HR 109 with runs of SVT
- Found to be encephalopathic with cortical blindness; enlarged erythematous tonsils, flushed cheeks, crackles over left lower lobe on auscultation; right foot tenderness and swelling with petechiae over ankle; weakness on dorsiflexion and plantar flexion with hyperreflexia.

Labs

- Glucose 9.2
- Lactate 5.1
- INR 1.4, PTT 30
- Ca 1.03, Mg 0.78
- Bili 14.6, GGT 56, AST 40, ALT 20, ALP 130
- CRP 284.6, Albumin 18, Procalcitonin >100



Evidence of Conflict of Financial Interest	
Co Author	**Conflict Disclosures**
1. Mary Wood	*No conflicts to disclose*
2. Meghan Ho	*No conflicts to disclose*

Imaging

- CXR: Patchy left lower lobe airspace changes
- CT Head: Multiple mass-like regions of low attenuation predominantly within the subcortical and deep white matter throughout the cerebrum
- MRI Head: Numerous bilateral asymmetric small to moderate T2/FLAIR lesions predominantly involving the juxtacortical and cortical regions of the supratentorial brain and cerebellum associated with diffusion restriction but without significant mass effect.

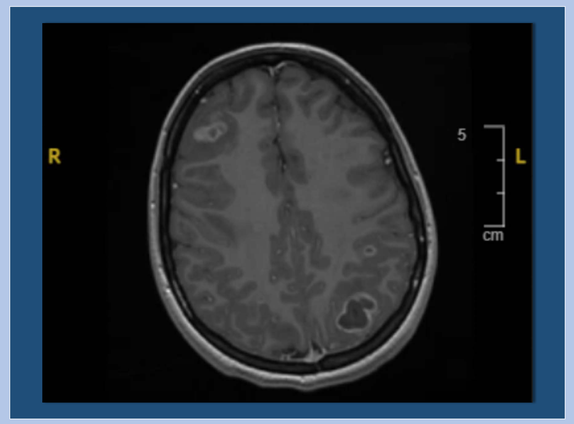


Fig 1. MRI After Initiation of Steroids and IVIG
Multiple peripherally enhancing lesions with reduced diffusion in the supratentorial and infratentorial brain compatible with abscesses

Clinical Course

- On admission the patient was initiated on broad-spectrum antibiotics for possible meningoencephalitis.
- CT head revealed possible septic abscesses vs. emboli, and initial MRI head was read as highly concerning for an acute demyelinating condition. They were treated with 5 days of methylprednisolone and IVIG for suspected ADEM given COVID-19 positive PCR.
- Neurology, Infectious Disease, Ophthalmology and Rheumatology were consulted, and MIS-C and acute COVID hyper-inflammatory state were also considered as potential etiologies, but ultimately these diagnoses were thought to be less likely given the acute onset and resolution of symptoms. A lumbar puncture was not completed given that the patient was severely thrombocytopenic.
- On day four of admission, initial blood cultures returned positive for Fusobacterium necrophorum and repeat MRI confirmed multiple brain abscesses with rim-enhancing lesions suggestive of hematogenous spread. Steroids were promptly discontinued and the patient was transitioned to metronidazole and ceftriaxone. Neurosurgical intervention was not indicated as the lesions were too small for drainage.
- Clinical re-examination revealed right submandibular swelling and pain, and a CT angiography was done which showed a complex cystic lesion in the right submandibular gland but no evidence of thrombosis. FNA was pursued with negative cultures.
- The patient's right ankle was also considered as a potential source of bacteremia or emboli and CT identified probable cellulitis but no collection large enough for FNA.
- Echocardiogram did not reveal any signs of endocarditis or vegetation. Stool cultures and autoimmune workup were also negative.
- The patient's vision, mobility, and strength gradually improved on antibiotics, but they had persistent proprioception deficits with some ataxia. They were discharged home with a total 6 week course of IV antibiotics with interval MRI surveillance and rehabilitation supports.

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Differential Diagnosis

	Key Clinical Features	Treatment
LS (Lemierre's Syndrome)	Presentation days to weeks post-oropharyngeal infection with IJ vein thrombosis; metastatic septic emboli; anaerobic gram-negative bacteremia [1]	Prolonged IV antibiotics ± drainage and anticoagulation
MIS-C (Multisystem inflammatory syndrome in children)	Presentation 2–6 weeks post-COVID infection or exposure, with unremitting fever; conjunctivitis; GI and mucocutaneous symptoms; shock; myocarditis; elevated CRP, D Dimer, ferritin, lymphopenia[2]	High-dose corticosteroids ± IVIG
ADEM (Acute disseminated encephalomyelitis)	Presentation days to weeks post infection with encephalopathy; multifocal neuro deficits; seizures; fever; headache; bilateral T2/FLAIR white matter lesions on MRI head[3]	High-dose corticosteroids ± IVIG PLEX if refractory to steroids

Learning Objectives

- Recognize the importance of keeping a broad and evolving differential when managing critically ill patients with diagnostic uncertainty.
- Utilize a multidisciplinary approach in the evaluation and treatment of Lemierre's Syndrome with atypical clinical features.

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Take Away Points

- Lemierre's Syndrome (LS), sometimes referred to as "*the forgotten disease*" is a rare diagnosis in the post-antibiotic era affecting approximately 1/million people yearly[4,5].
- Incidence is increasing, however, possibly related to changing antibiotic use, fewer tonsillectomies, and improved diagnostic techniques[6].
- Diagnosis is based on the classic triad of internal jugular vein thrombosis, oropharyngeal infection, with metastatic septic embolization to joints, soft tissues, kidneys, spleen, liver, or CNS and anaerobic gram-negative bacteremia[6,7].
- Appropriate management involves broad spectrum antibiotics (anaerobic coverage with meropenem or ceftriaxone + clindamycin or metronidazole), surgical drainage, and in some cases anticoagulation (if there is concern for thrombophilia, no improvement with antibiotics, or large bilateral thrombi)[5].
- There is a growing consensus that IJ vein thrombosis is *not* required for diagnosis, as clots may be transient or missed[1].
- Cases of *Atypical Lemierre's* have been reported with thrombi in the facial, iliac, and external jugular veins[7]
- While *F. necrophorum* is the most common pathogen, other organisms such as *F. nucleatum*, *Staphylococcus*, *Streptococcus*, and *Bacteroides* species have also been documented in atypical cases[1,7].
- Complications of LS include endocarditis, pericarditis, mycotic carotid pseudoaneurysm, meningitis, subdural/epidural empyema, cerebral venous thrombosis and stroke, and patients can develop persistent neurological sequelae, including cranial nerve palsies, blindness, hemiparesis, ataxia, and seizures[6,7,8].
- Early diagnosis and treatment are crucial to avoiding serious complications of LS, and a high index of suspicion should be maintained in patients presenting with septicemia and septic emboli.