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Miller Fisher Syndrome and Cavernous Sinus Thrombosis Caused by Sinusitis

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Dear Editor,

We read with interest the case by Ramasamy et al. and explore the clinical entities of Miller Fisher syndrome (MFS) and cavernous sinus thrombosis (CST).[1–3] Additionally, we discuss the concepts of causation and verifying the diagnosis, with a focus on the adequacy and coherence methods in medical decision making.

Charles Miller Fisher (1913–2012) described a variant of acute idiopathic polyneuritis (Guillain Barré syndrome) with:

> [t]otal external ophthalmoplegia, severe ataxia, and loss of the tendon reflexes. The clinical picture in all three cases was so similar as to constitute an easily recognizable syndrome. (…) [t]he diagnosis is hardly to be suspected at the onset unless one realizes that such an atypical form exists (p. 57).[3]

Other symptoms and physical findings found included ptosis, paresthesias, headache, sensory deficits, and diplopia.[3] MFS neither "mimics" nor "masquerades" as acute sinusitis.[4] It is a rare but distinct clinical entity distinguished by an observed, recurrent, recognizable pattern, not all of which may be present or required for diagnosis.

The patient in this case had a thrombus involving the cavernous sinus precipitated by acute sinusitis. As stated by Eagleton:

> For early diagnosis it must be appreciated that the classical symptoms of exophthalmos, edema of the lids, and chemosis may or may not be present, depending on whether the sinus is suddenly and completely obstructed by an acute septic process, or gradually obliterated by a compensatory thrombus (p. xx-xi).[2]

In both CST and MFS, an infectious process, including sinusitis, frequently acts as a proximate cause leading to complex alterations in the immune response and disease expression. The computed tomography (CT) scan revealed bilateral CST, but it did not explain all the physical findings nor account for their persistence despite the spontaneous resolution of the thrombus. The presence of anti-GQ1b antibody in some MFS patients provides additional confirmation for the diagnosis of MFS. As explained by the authors, the absence of anti-GQ1b antibodies does not rule out the diagnosis, as they may not be present in all patients and are not required for diagnosis.[1] Although the absence of the antibody slightly reduces the probability of the disease, the pretest probability is already high in patients suspected of having MFS.

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This case serves as a reminder that in clinical decision-making, it is crucial to take a "diagnostic pause," consciously reflecting and considering the probability of the disease and the test result, in order to avoid making the fewest testing errors. Physicians must avoid the common pitfall of relying solely on a test(s) for diagnosis and develop a framework for assessing the significance of a findings. Verifying the diagnosis involves the process of adequacy and coherence.[5] Adequacy refers to whether a diagnostic hypothesis or test explains the symptoms and physical findings, while coherence is used to determine whether a diagnostic hypothesis is pathophysiologically sound.[5] By maintaining an open mind and applying the concepts of adequacy and coherence in the diagnostic process, the authors avoided the medical error of premature closure or accepting a diagnosis as final without considering other diagnostic possibilities.

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