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Cerebrospinal Fluid (CSF) Lactic Acid Levels: a Rapid and Reliable Way To Differentiate Viral from Bacterial Meningitis or Concurrent Viral/Bacterial Meningitis

I read with interest the articles by Drs. Nolte and Basmaci, et al., regarding potential concurrent acute viral meningitis (AVM) and acute bacterial meningitis (ABM) (2, 10). I agree the probability of concurrent ABM/AVM is exceedingly rare, but their concerns merit a response.

The diagnostic difficulties mentioned could have been easily sorted out with cerebrospinal fluid (CSF) lactic acid levels. CSF lactic acid levels are a rapid, reliable, and inexpensive method for early differentiation of AVM from ABM levels. For over 30 years, Winthrop-University Hospital has used CSF lactic acids levels to rapidly differentiate AVM from ABM (1, 3, 4). Our CSF lactic acid level breakpoints are <3 mmol/liter for normal/AVM, 3 to 6 mmol/liter for partially treated bacterial meningitis (PTBM), and >6 mmol/liter for ABM. CSF lactic acid levels may be slightly increased with CSF red blood cells (RBCs). CSF lactic acid levels increase in proportion to the duration/number of RBCs in CSF (1, 3). When a patient presents with acute meningitis syndrome, a lumbar puncture is performed and CSF lactic acid levels are included as part of the CSF profile. CSF samples with normal lactic acid levels, indicative of VAM, have been so reliable that patients are not given empirical antibiotics while waiting for bacterial culture/PCR results. PCR results often take hours or days to be reported and are not helpful in therapeutic decision making. In over 30 years, we have never had a case of ABM without a highly elevated (>6 mmol/liter) CSF lactic acid level. With PTBM, as the authors point out, the Gram-stained CSF cultures may be negative. CSF lactic acid levels are also useful in the diagnosis of PTBM. In our view, in a patient who presents with a meningitis syndrome who has been recently treated with antibiotics, the best way to make a diagnosis of PTBM is by demonstrating CSF lactic acid levels of 3 to 6 mmol/liter. In such cases, we treat PTBM as ABM with a full 2 weeks of antimicrobial therapy.

It is not known how long PCR positivity for enterovirus/other viruses persists in the CSF after subclinical/clinical infection. It may be that some patients with so-called "coinfections" had antecedent enteroviral infections followed by subsequent bacterial infection, as occurs with community-acquired bacterial pneumonia following viral influenza (5–9, 11, 12).

I agree with the authors that clinical judgment is the ultimate arbiter of discordant, conflicting, or otherwise unexplained laboratory results (2, 10). In the cases cited by the authors, the differentiation of AVM from ABM could have been rapidly and easily made if CSF lactic acid levels were utilized (1, 3, 4). Aside from sorting out coinfections, the value of CSF lactic acid levels resides not only in their diagnostic accuracy but in the economic implications. In patients hospitalized with meningitis syndrome, CSF lactic acid levels of <3 units rapidly rule out PTBM/ABM. In our experience, if CSF lactic acid levels are unelevated in such patients, resources are conserved, hospital stays shortened, and antibiotic exposure diminished since no empirical antimicrobial therapy is necessary in such patients.

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