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The Case of the Bright Splenium

Patricia Serrano*

Einstein Healthcare Network, 5501 Old York Rd., Philadelphia, PA 19141, Pennsylvania, USA

*Corresponding author: Patricia Serrano, Einstein Healthcare Network, 5501 Old York Rd., Philadelphia, PA 19141, Pennsylvania, USA, Tel: 2154981202; E-mail: serranop@einstein.edu

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Abstract

Altered mental status is a frequent reason for consult for neurology and psychiatry services. Multiple causes can be considered including: Wernicke's, hepatic encephalopathy, metronidazole-induced encephalopathy, and acute demyelination. We present the case of a 49-year-old Hispanic female with end stage liver disease admitted for altered mental status. Neurology and Psychiatry were consulted.

Keywords: Encephalopathy; Extrapontine myelinolysis; Wernicke's

Case Report

She had been at an outside hospital one week prior where she had been treated with metronidazole for *C. difficile*. Since discharge she had worsened with altered sleep-wake cycles and confusion. On the morning of her admission her family could not awaken her. On admission she would awaken to noxious stimuli, immediately falling back to sleep. She was hyperreflexic with bilateral ankle clonus and Babinski. Her neck extensors were stiff. No twitching or jerking was noticed. Her ammonia level was 36.5 $\mu\text{mol/L}$. EEGs showed generalized slowing. Head tomography did not show any acute findings [1]. Lumbar puncture was negative except for mildly elevated protein of 63 mg/dl. MRI showed T2 hyperintense signal associated with restricted diffusion of the splenium of corpus callosum [2]. Given that the patient was receiving metronidazole our initial diagnosis was metronidazole-induced encephalopathy. But after stopping it she did not improve. Most reported cases have excellent recovery. This prompted us to delve deeper. Her total dose of metronidazole was 15 gm over 10 days. In most reported cases the dose was actually

much higher and the duration much longer. We obtained reports from the prior hospitalization. The sodium upon discharge was 141 mg/dl. Her sodium on arrival was 156 mg/dl. Also, in spite of her family denying patient's alcohol use in the past 3 months, her ethanol levels at the outside hospital were 127 mg/dl. She had been treated at our institution upon admission for presumptive Wernicke's with a high dose of thiamine IV. Based on this new history and MRI our diagnosis was extrapontine myelinolysis (EPM). A follow-up MRI showed no improvement. We considered the damage permanent and when goals of treatment were discussed with the family she was discharged to hospice care.

Discussion

EPM, even though it is often a cause of rapid hyponatremia correction, may also present in patients with a history of chronic alcoholism. In this case, the condition is often unrelated to correction of sodium. EPM can present with rigidity of limbs, bradykinesia, tremors, and decreased blinking. Some of the behavioral manifestations are inappropriate affect, disinhibition, delirium, and in rare cases catatonia. Treatment is supportive, addressing underlying conditions. Complex cases such as this one requires an interdisciplinary approach with efficient communication among consultants, primary team, and caregivers; careful observation and documentation; a detailed history and review of prior records to provide appropriate care.

References

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