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Letter to the Editor

Sudden unexpected nocturnal death in Chiari type 1 malformation and potential role of opioid analgesics

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Sir,

Our recent article entitled "Sudden unexpected nocturnal death in Chiari type 1 malformation and potential role of opioid analgesics"[5] was criticized by Jonathan and Shlomi.[3] They challenged our opinion without any reference to authenticate their assertions.[3] In our original article, an axial T1-weighted MRI image of the posterior fossa [Figure 1] was replaced by a T2 fluid-attenuated inversion recovery image of a similar view. We do not believe that this oversight could have resulted in their error of mistaking a disproportionately large communicating fourth ventricle (DLCFV) for a "trapped" fourth ventricle.[3]

The term "trapped fourth ventricle" is used to describe a dilated fourth ventricle due to obstruction of both its inlet and outlet.[1,5] Shortly before his death, the patient had undergone brain imaging examinations at two different institutions. These studies as well as a postmortem examination excluded the possibility of a trapped fourth ventricle based on the apparent patency of the Sylvius aqueduct.[5] Cystic dilatation of the fourth ventricle is a well-known feature of DLCFV.[1,5] Different mechanisms have been postulated to explain the fourth ventricle dilation and distribution of periventricular edema observed in DLCFV.[1,5] Our experience and published literature indicate that the most likely etiology of DLCFV is increased fragility of the surrounding fourth ventricle tissues to elevated intraventricular pressure, or unequal distribution of intraventricular pressure in tension hydrocephalus such that the intraventricular hypertension primarily affects the ventricle closest to the point where the flow of the cerebrospinal fluid (CSF) is obstructed. In our patient there likely is fourth ventricle outlet obstruction associated with Chiari type 1 malformation (CM1).[1,5]

Sleep apnea is a frequent, and sometimes the sole, manifestation of CM1.[4,5] Hydrocephalus, chronic opioid use, and bolus dosing of opioid analgesics are also risk factors for disordered breathing during sleep.[2,4,5] Bolus opiate dosing and sleep apnea associated hypercapnia are known to contribute to intracranial hypertension.[2,5] Therefore in a patient with clinical evidence of high ICP and chronic opioid use, bolus administrations of opioid analgesics can further increase the ICP.[2,5] Increased ICP can give rise to further downward displacement of the brain into the spinal canal and additional compression of vital brain structures. These effects can cause more instability of the cardiopulmonary control centers in the brain stem, resulting in cardiorespiratory arrest with possible sudden "neurogenic" cardiorespiratory death.[5]

Therefore, although non–opiate related conditions may have played a role in our patient's clinical deterioration, the patient's death during sleep following administration of opioid analgesics strongly suggests a causal relationship.[5] We agree that some patients diagnosed with CM1 are asymptomatic, but our patient was not asymptomatic; he had a sub-acute clinical

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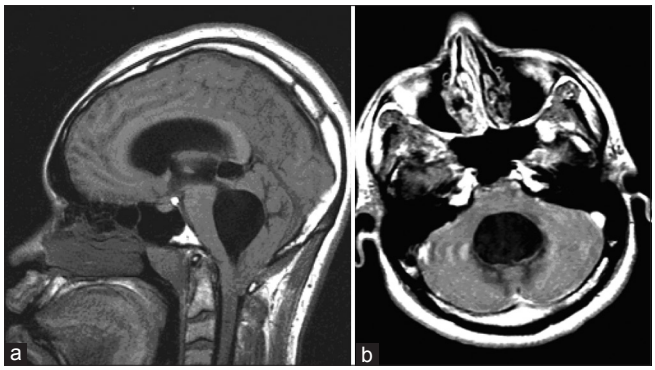

Figure 1: (a) T1-weighted sagittal MRI of the brain demonstrating inferior displacement of the cerebellar tonsils 18 mm below the level of C1 consistent with a Chiari I malformation. Note the relatively mild hydrocephalus and a pear-shaped disproportionately large communicating fourth ventricle. (b) Axial view, gadolinium-enhanced T1-weighted image of posterior fossa at the level of medulla and cerebellum. Note the enlarged fourth ventricle

course with a fatal outcome.[5] We think that the fatal outcome could have been prevented if his treating physicians were aware of the association between opioids and increased ICP and avoided opioids as much as possible, particularly bolus dosing of opioids before surgery. Therefore, we are ethically obliged to present this report and encourage our colleagues to consider opiates as a potential trigger for the further increase of intracranial hypertension in patients with reduced intracranial compliance.[1,2,5]

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