LETTER TO THE EDITOR

Hiccups before a Pulmonary Embolism Speak against This as a Cause

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We read with interest the article by Goyal et al. reporting on a 66-year-old male with acute, right posterior inferior cerebellar artery (PICA) stroke complicated by cerebellar edema with a space-occupying effect on hospital day 2 (hd2) requiring mechanical ventilation and immediate decompressive craniotomy with an external ventricular drain (EVD).[1] On hd7, he was put off the ventilator and on hd8 the EVD was removed.[1] On hd9, he developed hiccups, and on hd11 sudden onset tachycardia, tachypnea, arterial hypotension, and drowsiness, due to which he was re-intubated.[1] Pulmonary embolism was diagnosed and the patient was put back on mechanical ventilation.[1] Hiccups spontaneously resolved after altogether 7 days.[1] Pulmonary embolism was made responsible for hiccups.[1] The study is appealing but raises concerns that warrant further discussion.

We disagree with the notion that hiccups were due to pulmonary embolism.[1] A strong argument against pulmonary embolism as the cause of hiccups is that hiccups started on hd9 whereas pulmonary embolism most likely occurred on hd11 manifesting with sudden onset tachycardia, drowsiness, tachypnea, and arterial hypotension. Therefore, causes other than pulmonary embolism were more likely responsible for the hiccups. Because hiccups developed immediately after the removal of the EVD on hd8, it is conceivable that an increase in the intracerebral pressure was causative rather than pulmonary embolism. Because the patient had an acute ischemic stroke with secondary bleeding in the territory of the right PICA,[1] it is also conceivable that hiccups were due to the space-occupying stroke and causally unrelated to pulmonary embolism. Hiccups caused by a stroke in the PICA territory have been previously reported.[2] Because glucocorticoids can trigger prolonged hiccups.[3] It is also conceivable that the antiedema therapy was causative if it included steroids.

The patient was diagnosed with pulmonary embolism, but repeated electrocardiography (ECG) recordings and echocardiography findings were non-informative.[1] In the case of pulmonary embolism echocardiography usually shows acute right strain, right ventricular dysfunction, or elevated pulmonary artery pressure. In case of pulmonary embolism ECG usually shows right type ECG or right anterior hemiblock. How do the authors explain this discrepancy? Was the D-dimer elevated in the index patient?

A limitation of the study is that the cause of the stroke was not specified.[1] Because the patient had an uneventful previous history with a negative cardiovascular risk profile, we should know whether the patient had newly developed atrial fibrillation, dissection of the right vertebral artery, left ventricular dysfunction, or endocarditis.

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We should know whether transesophageal echocardiography had been carried out to rule out endocarditis and whether pro-brain natriuretic peptide (pro-BNP) was normal or elevated. To rule out dissection of the right vertebral artery we should know whether the carotid ultrasound showed a double lumen or if axial T1, T2, or diffusion-weighted imaging (DWI) showed the hyperintense crescent sign or a flap.[4]

Overall, the study carries obvious limitations that require re-evaluation and discussion. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Before attributing hiccups to pulmonary embolism, alternative etiologies should be considered and ruled out.

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