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Letter to the Editor

Before attributing sick sinus syndrome to increased intracerebral pressure, all other causes must be carefully excluded

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We read with interest the article by Al-Attas et al. about a 47-year-old man with venous sinus thrombosis (VST) complicated by increased intracerebral pressure (ICP) and sick sinus syndrome (SSS) [1]. The patient made a full recovery with heparinization and implantation of a pacemaker [1]. The report is noteworthy, but several points should be discussed.

The first point is that causes of SSS other than increased ICP have not been adequately ruled out [1]. SSS can be primary (hereditary) or acquired. Primary SSS is due to mutations in SCN5A, MYH6, KCNG2, MECP2, LMNA or HCN4. Since no genetic testing was performed, primary SSS cannot be definitively ruled out. It was also not mentioned whether or not there was a consanguinity between the parents of the index patient. Secondary causes of SSS include advanced age, infections (e.g. Chagas, typhoid, SARS-CoV-2), inflammation, hypoxia, muscular dystrophy, infiltrative diseases, hyperthyroidism, trauma, previous cardiac surgery, refeeding syndrome or medication [2]. To rule out secondary SSS, it would have been useful to perform a magnetic resonance imaging (MRI) of the heart with contrast, especially to determine whether the patient had myocarditis. As the case occurred during the pandemic, it must be ruled out that the patient was infected with SARS-CoV-2, which can be complicated not only by myocarditis but also by VST [3, 4].

The second point is that the cause of VST has not been clarified [1]. VST can generally be caused by thrombophilia, dehydration, infection, intracranial hypotension, malignancy, inflammatory bowel disease, sickle cell anemia, blood disorders (e.g., thrombocytopenia), obesity, granulomatosis with polyangiitis, traumatic brain injury, heart disease, nephrotic syndrome, or medications [5].

The third point is that reciprocal causality has not been considered. Instead of the VST causing the SSS, the VST could also be due to the SSS. SSS can be complicated by heart failure and low blood pressure, which can be secondary to VST. Therefore, we should know the

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serum levels of creatine kinase-MB, troponin and pro-brain natriuretic peptide (proBNP) as well as D-dimer.

The fourth point is the discrepancy between the history, in which the index patient is described as confused, and the clinical examination in the emergency department, in which the patient was described as oriented. This discrepancy should be clarified.

The fifth point is that the sinus rhythm obviously returned spontaneously, as no pacemaker beats were recorded on the normo-frequency ECG after the pacemaker implantation. What cardiological measures or medication other than the pacemaker did the patient receive?

Finally, we should know whether the patient was tested for SARS-CoV-2, whether there was thrombocytopenia, deep vein thrombosis or pulmonary embolism. It should also be indicated whether the papilledema has regressed or persisted.

In summary, before attributing SSS to increased ICP following VST, all different causes of SSS must be thoroughly ruled out. Similarly, all causes of VST must be considered and the correct cause of VST in the index patient identified. Assuming that high ICP actually causes SSS, the prevalence of SSS in patients with elevated ICP, regardless of the cause, must be much higher than reported in the literature.

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