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Title: IRON-DEFICIENCY ANEMIA AS A PRECURSOR TO CEREBRAL VENOUS SINUS THROMBOSIS: A CASE REPORT

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Background and aims: Cerebral venous sinus thrombosis (CVST) poses a rare yet severe neurological challenge, with multifactorial etiology encompassing prothrombotic conditions, infections, trauma, medical procedures, hormonal factors, inflammatory disorders, dehydration, malignancies, and rare inherited disorders. This case report explores the association between iron deficiency anemia and the development of CVST, shedding light on the non-specific symptoms.

Methods: A 41-year-old female presented with: headache, dizziness, nausea, and vomiting. Examination revealed impaired consciousness, left-sided hemiplegia, and brisk reflexes. Diagnosis of CVST was made with computed tomography (CT) and computed tomography angiography (CTA). A comprehensive assessment, including blood count and iron studies, was conducted to evaluate the patient's iron-deficiency anemia. The case was initially managed with anticoagulation therapy and two units of packed red blood cells.

Results: Imaging studies displayed bilateral subarachnoid hemorrhage, intracerebral hematomas, and a filling defect in the superior sagittal sinus. Laboratory results indicated iron-deficiency anemia and thrombocytosis. Subsequent CT scans revealed hyperdense hemorrhagic changes, persistent SAH, and intracerebral hematomas. Physiologically, iron deficiency induces a prothrombotic state through increased platelet aggregation, alterations in clotting factor function, and impaired fibrinolysis. Additional links include endothelial dysfunction, elevated homocysteine levels, and reduced nitric oxide bioavailability.

Conclusions: This case underscores the significance of recognizing iron deficiency as a significant contributor to a hypercoagulable state in the context of CVST. The interplay of iron in physiological processes, coupled with its deficiency, heightens the susceptibility to thrombotic events. Recognition and management of iron deficiency anemia are crucial in mitigating the risk of complications associated with CVST.



Figure 1 Bilateral cerebral oedema

On behalf of: