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## Thrombosis in a patient with thrombocytopenia

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Cerebral venous thrombosis (CVT) is an acute life-threatening condition that usually occurs due to an underlying hypercoagulable state. The infrequency with which it presents insidiously and with underlying thrombocytopenia makes it a formidable diagnostic challenge. A 43-year-old woman presented with 1-month history of intermittent throbbing bi-temporal headache mild to moderate in severity, associated with nausea, vomiting, and blurring of vision not responding to analgesics. Her past medical history includes diabetes, hypertension, dyslipidemia, obesity and a 3-year history of menorrhagia. Neurological examination revealed bilateral papilledema. No evidence of meningism or focal neurological deficit. General examination and that of other systems was normal. Investigations showed microcytic hypochromic anemia (Hb: 7.5 g/dl), low platelet count (63,000), normal coagulation profile. Screening for vasculitis came back normal. No source of active bleeding or any malignant disease was found on upper/lower GI endoscopies. MRI brain and MR venography revealed right transverse, sigmoid sinus and right internal jugular vein thrombosis. Lumbar puncture showed high opening pressure, with no cells, normal glucose and protein.

The patient was treated with low molecular weight heparin, later switched to warfarin tablets. Iron tablets were also started to correct the anemia. Daily hemogram showed rapid platelet correction within a week (200,000). The patient's symptoms improved gradually. Upon discharge, the patient's Hb improved slightly (8.2 g/dl) and the platelet count came up to 336,000. The cause of anemia was presumed to be due to the longstanding menorrhagia, after ruling out all other possible causes. One theory to explain the link between IDA and thrombosis is that the increased viscosity, oxidative stress and cell deformability found in IDA, may contribute directly to a hypercoagulable state within the vessels.

This case illustrates the potential for CVT to occur simultaneously with thrombocytopenia and anemia. The correction of iron deficiency rapidly restores the platelet count as well, which may contribute to the reversal of symptoms, in the presence of adequate anticoagulation. Whether the menorrhagia is an independent risk factor for a hypercoagulable state has yet to be studied. Recognition of this rare but possible scenario is critical to the timely initiation of appropriate therapy.

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