Teaching Neuro*Image*: MRI visualization of papilledema associated with cerebral sinovenous thrombosis in a child

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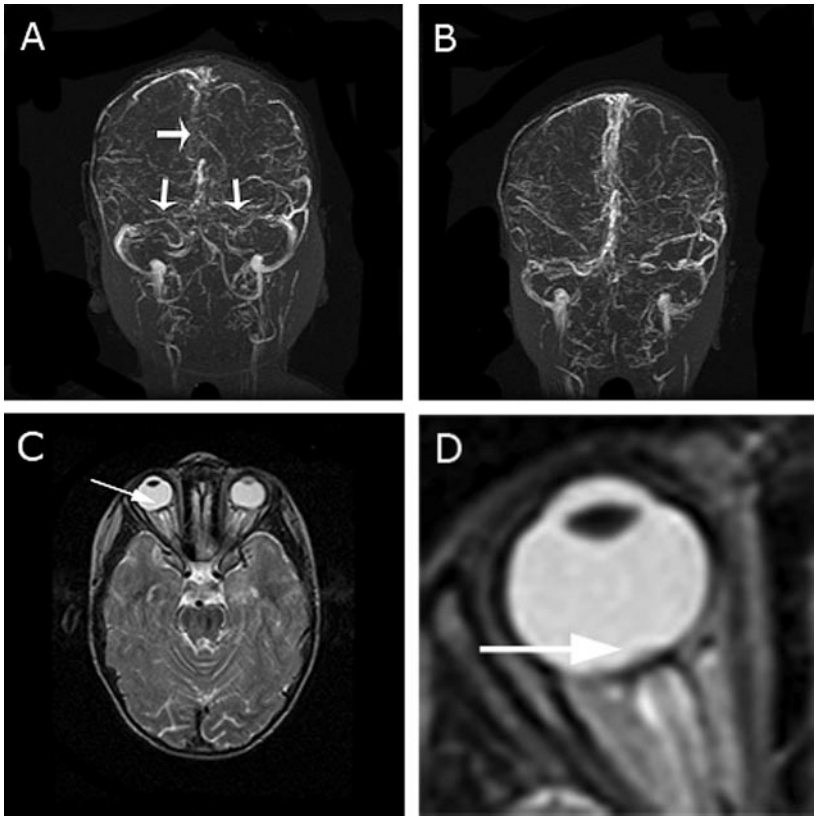
Figure MRI



A) Initial MR venography demonstrated partial thrombosis of the superior sagittal sinus, torcula, and proximal transverse sinuses (arrows). B) Follow-up MR venography at the time of visual worsening showed improvement in venous flow, with mild residual thrombosis. C) Fast-spin echo T2-weighted axial imaging demonstrated reversed optic nerve cupping in the right eye with posterior scleral flattening and protrusion of optic nerve papilla into the globe (arrow) (subsequent image on MRI demonstrated similar findings in the left eye). D) Close-up of right eye seen in C. MRI data: (A and B) repetition time (TR) 33.3 msec, echo time (TE) 6.9 msec, ST 1.5 mm; (C and D) TR 3,500 msec, TE 95 msec, ST 5.0 mm.

A 3-year-old boy with ulcerative colitis and developmental delay presented with a 3-week history of headaches, emesis, and dehydration. His neurologic examination was nonfocal, but MR venogram revealed cerebral sinovenous thrombosis (figure). He was rehydrated and discharged on enoxaparin. Three months later he starting having difficulty reaching for objects and was running into walls. Repeat MRI and MR venogram demonstrated residual thrombosis and interval development of papilledema (figure). Papilledema was confirmed on funduscopic examination. CSF opening pressure was 37 cm H_2O.

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When treatment with acetazolamide failed to control intracranial hypertension, he received a ventriculoperitoneal shunt.

Inflammatory bowel disease (IBD) is a risk factor for venous thromboembolism.[1] Attempts to identify a unifying genetic risk factor for thromboembolism in patients with IBD have yielded conflicting results. Endothelial damage from IBD, leading to platelet activation and release of inflammatory mediators by platelets, may play a role.[2] Our patient's prothrombotic workup demonstrated only heterozygosity for both the methylene tetrahydrofolate reductase (MTHFR) C677T and plasminogen activator inhibitor (PAI)-1 4G (also known as SERPINE 1) mutations.

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