Commentary

The deep cerebral venous system largely consists of two internal cerebral veins and the great cerebral vein, better known as vein of Galen [Figure 1]. Vein of Galen receives venous inflow from both basal ganglia, thalami, geniculate bodies and midbrain. It is said that one of the deceased patients of Quinke, the clinician credited for developing the procedure of lumbar puncture, had thrombosis of transverse sinuses and the vein of Galen.[1] In the past, deep cerebral venous thrombosis was thought to be exclusively limited to infants and young children and was almost uniformly fatal. However, the condition has become increasingly recognised in adults since the 1980s with the advent of modern neuroimaging and the experience of a favourable outcome in paediatric and adult cases.

Occlusion of deep cerebral venous system occurs less commonly than dural venous sinuses. Spontaneous thrombosis of vein of Galen has been frequently reported with aneurysmal malformation of the vein. Secondary causes of deep cerebral venous thrombosis are broadly similar to dural venous sinus thrombosis. Sickle cell anaemia, dehydration, autoimmune vasculopathies and hyper-coagulable states are commonly implicated, it may not always be possible to identify a cause in about a third of cases. Extension of venous thrombosis to cortical and deep veins is usually a late complication of progressive dural venous sinus thrombosis. Unlike the clinical signs and symptoms of dural-based venous sinus occlusion that are often diffuse and non-localising (cavernous sinus is a notable exception), patients developing thrombosis of vein of Galen and in ternal cerebral veins have a characteristic syndrome due to the anatomical localisation of lesions, often presenting with altered level of consciousness and arousal. Early symmetric, hyper-intense signal changes in bilateral thalamic and basal ganglia in T2-weighted magnetic resonance imaging (MRI) of brain reflect vasogenic odema. These signal changes are potentially fully reversible with the resolution of thrombus, established infarcts due to deep venous occlusion are best appreciated in diffusion-weighted MRI and could be unilateral or occasionally haemorrhagic.

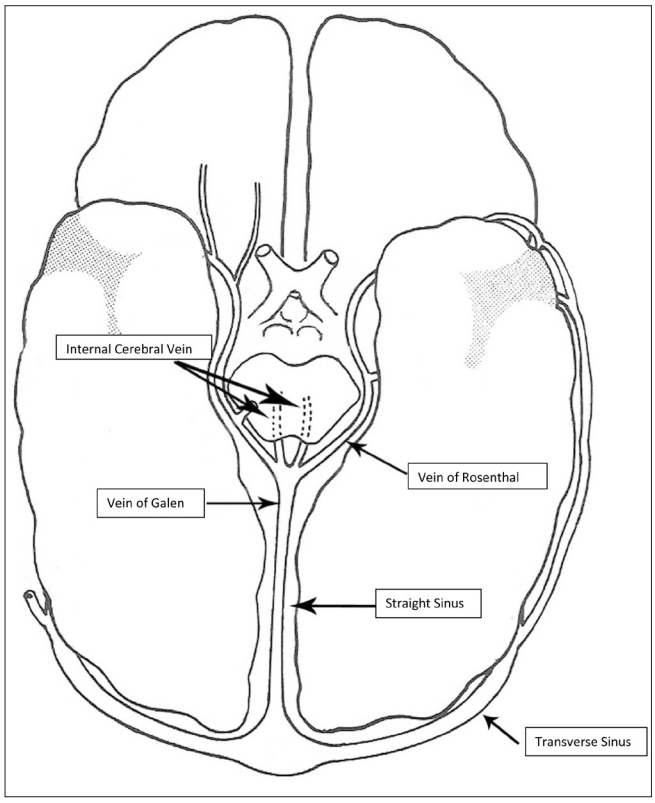

Figure 1: Line drawing of the base of the brain showing deep venous system and dural venous sinuses

In this issue of the JNRP, Hassan and Kumar report a case of reversible diencephalic dysfunction due to deep cerebral venous thrombosis from hyper-homocysteinemia and protein S deficiency.[2] The report merits attention because of the presented clinical syndrome and the meticulous investigations that were undertaken to identify the cause of deep cerebral venous system occlusion (hyper-homocysteinemia and protein S deficiency). In general, the correlation between protein C and S levels and the risk of venous thrombosis is not always as precise or predictable as in antithrombin III deficiency, a systemic co-factor or co-morbidity is often considered to play a per-missive role to trigger venous thrombosis in these patients. Hyper-homocysteinemia increases the risk of both arterial and venous strokes due to endothelial dysfunction and was identified as a co-factor for deep cerebral venous occlusion in this case.

Anti-coagulation remains the preferred treatment for cerebral venous thrombosis, and body-weight adjusted low molecular weight heparin or dose-adjusted unfractionated heparin followed by warfarin is usually deployed. Currently recommended duration of warfarin therapy in cerebral venous thrombosis is three months for transient and non-recurrent risks (for example, dehydration as in puerperal cerebral venous thrombosis), 6-12 months for those with indeterminate cause or mild thrombophilia and life-long in patients with recurrent venous thrombosis or severe hereditary thrombophilia.[3]

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