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Case Report

Simultaneous Cerebral and Cerebellar Venous Thrombosis: Case Report

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ABSTRACT

Introduction: Cerebral and cerebellar venous thrombosis with subarachnoid hemorrhage is rarely seen in one patient, in the routine neurosurgical practice.
Case report: A 29 years old non hypertensive male presented with severe generalized headache and vomiting since 8 days and episode of generalized tonic and clonic seizures followed by left sided weakness.
Conclusion: Cerebral venous thrombosis must be considered in the differential diagnosis of patients presenting with a broad range of neurological presentations especially in the presence of new onset of seizures. Computed tomography offers many clues to the diagnosis of CVT when concomitant SAH is present. These include the presence of SAH at cerebral convexities with associated basal cisterns and skull base sparing. Recognition of these subtleties will allow prompt and appropriate management and, when in doubt, encourage further investigations.
The aim of presenting this case is, one rarely finds cerebral and cerebellar cortical venous thrombosis in one case.

Key words: Cerebellum**,** Posterior fossa**,** Cerebral venous thrombosis, Cerebellar venous thrombosis, Sub-arachnoid haemorrhage.

INTRODUCTION

Cerebral venous thrombosis (CVT) is the occlusion/thrombosis of the veins and/or venous sinuses in the brain. CVT is an uncommon cause of stroke. CVT was first described by Dr. Ribes, French physician in 1825, [1] who reported a 45-year-old man presented with severe headaches, epilepsy and delirium. The incidence of CVT accounts for 3–5 cases per 1million population or 0.5 % of all strokes. [2,3] Among patients with CVT, the incidence of cerebral hemorrhage is about 35 % to 39 %. [4,5] The advent of novel investigations including CT and MRI brain facilitate the clinical diagnosis of CVT. Cerebral venous sinus thrombosis most commonly affects young adults, predominantly in woman [4-7] with various clinical presentations, most commonly persistent headaches accompanied by focal neurological deficit or seizures. However, the presentation with cerebral hemorrhage may be challenging and a common pitfall in diagnosing cerebral venous sinus thrombosis. In addition, cerebral hemorrhage in CVT is prognostic factor, usually associated with poorer outcomes. [5,7,8]

PRESENTATION OF CASE:

A 29 years old non hypertensive male presented with severe generalized headache and vomiting since 8 days and episode of generalized tonic clonic seizures followed by left sided weakness. On examination patient was drowsy, arousable, responding to simple verbal commands. His pupils were normal size and reacting to the light. He was having left sided upper motor neuron facial paresis with left hemiparesis. There was no other cranial nerve involvement noted. His follow up imaging was suggestive of increase in the size of haemorrhagic component with significant midline shift. Lesion in the cerebellar region remains relatively unchanged. Patient was undergone emergency craniotomy with evacuation of the venous infarct on right side.

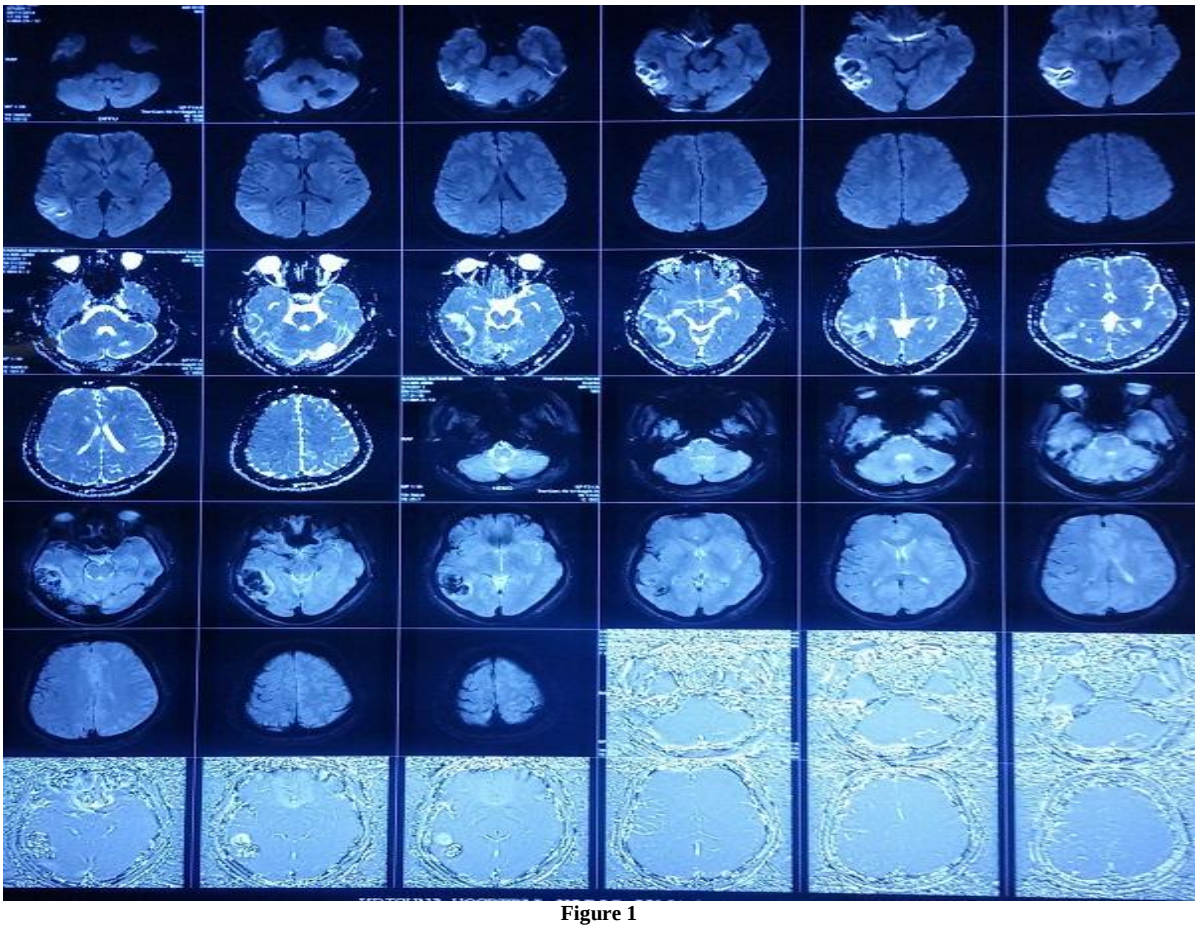

Figure 1

Investigations: Has normal hematological and biochemical parameters, with normal LFT.

Evaluation of procoagulant state showed PT 15 sec (control 16 sec) ,

Serum Homocystiene -31.05 (3.7-13.9), Protein C -92 (67-195), Protein S -30 (77-143), Antithrombin 3 -88 (70-122).

PTT Patient -64.2 (30-43) control 37.7 (30-43) Antiphospholipid antibodies IgG -1.7 and IgM -1.6 lupus anticoagulant –present.

Non contrast CT and MRI – Revealed right temporopareital hemorrhagic venous infarct with SAH with midline shift. There was similar lesion in left cerebellar region with perifocal oedema. No e/o hydrocephalus.

There was no visualization of Dural sinuses on MR Venogram.

Management:

Patient undergone right fronto-temporo -parietal craniotomy with evacuation of the venous infarct, lax duroplasty with repositioning of bone flap in the anterior abdominal wall.

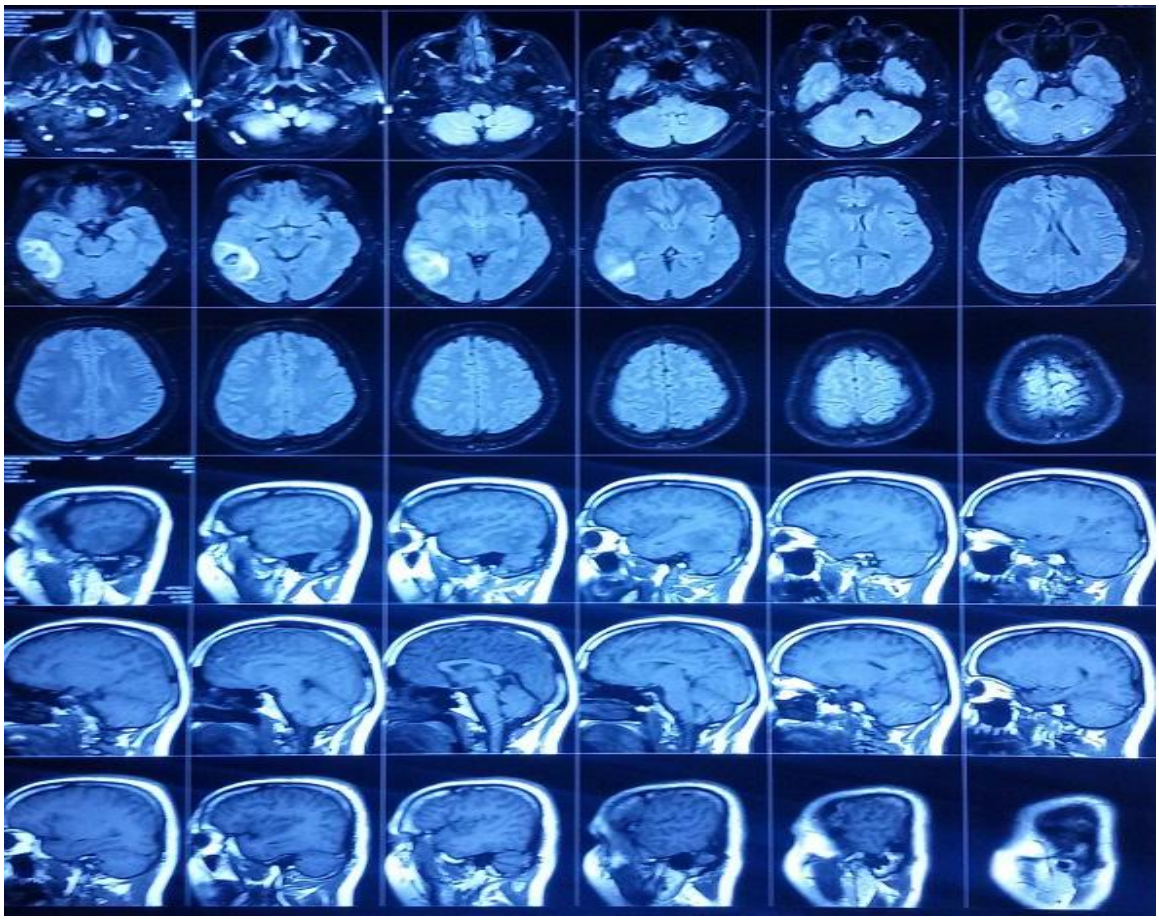

Figure 2

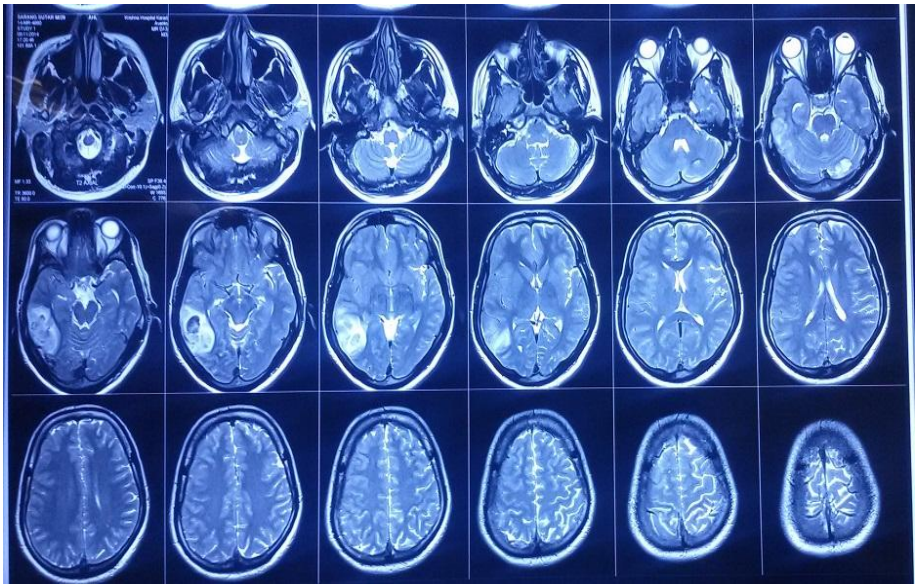

Figure 3

DISCUSSION

Thrombosis of the cerebral veins is a relatively uncommon but potentially life-threatening condition, accounting for 1–2% of strokes in young adults. Reported death rates range between 5% and 30%. [8-10] Thus, early diagnosis and treatment is crucial.

The terminology of cerebral venous thrombosis is often applied liberally and is usually used to refer to thrombosis of one of the major dural sinuses. Cortical vein thrombosis is usually secondary to dural sinus thrombosis, with thrombus propagating in a retrograde fashion from the occluded sinus. [11]

There are myriad predisposing factors for cerebral venous thrombotic disease. These include pregnancy, dehydration, medications, hereditary coagulopathies, systemic disease, trauma, infection, and idiopathic causes. [8,12] In our case, the patient had hypercoagulopathy. The prospective clinical diagnosis of cerebral venous thrombosis is difficult because of a wide spectrum of clinical manifestations; the diagnosis is typically made on the basis of imaging studies. The most commonly described symptoms are headache, seizures, mental obtundation, and focal motor or sensory deficits. [13] The clinical manifestations of cortical vein thrombosis with cerebellar infarct are less well known, but review of the literature suggests that there is similarity with dural sinus thrombosis. [11,12] In our case, the patient presented with headache, vomiting and developed seizures, left sided weakness and paresthesia. These manifestations are likely related to the irritative effects of SAH around the central sulcus and localized venous hypertension that is radiographically unapparent. The varied clinical presentations may be explained by greater variability in size and location of the cerebral veins in comparison to the cerebral arteries. [14]

In the past, conventional angiography was regarded as the standard diagnostic examination for cerebral venous thrombosis. With the advent of MR imaging, however, conventional angiography is now rarely required for diagnosis. Because the accuracy of angiography depends on the lack of opacification of the cerebral veins, there are known limitations. The diagnosis of isolated CVT is particularly difficult on the basis of angiographic findings, because the pattern of cortical venous drainage is variable and often asymmetric. The MR appearance of a thrombosed cerebral vein is dependent on the age of the clot. Initially, when blood products are in the deoxyhemoglobin state, thrombus is isointense relative to parenchyma on T1-weighted images and hypointense on T2-weighted images. As the blood products in the thrombus evolve to the state of methemoglobin, the clot appears hyperintense on T1- and T2-weighted sequences. [15] A similar time course of signal intensity changes has been described in isolated CVT. [11,12]

In addition to avoiding intravenous contrast material administration and ionizing radiation, MR imaging is far more sensitive than CT or conventional angiography in detection of the parenchymal changes associated with venous thrombosis. Cerebral venous thrombosis often results in localized cerebral edema or infarction that does not conform to an arterial vascular territory. Hemorrhagic venous infarction is common. In contradistinction to arterial infarction, these parenchymal changes are usually subcortical. [15]

This patient showed SAH in bilateral temporo parietal lobes with right temporal and left cerebellar haemorrhagic infarct. SAH has rarely been reported in association with cerebral venous thrombosis. Sztajzel et al [16] described one patient who presented with right cerebellar SAH associated with thrombosis of the right transverse or sigmoid sinus. This patient subsequently developed hemorrhagic infarction of the cerebellum.

In cases of suspected CVT, spin-echo T1-weighted sagittal, double-echo T2-weighted axial, and axial FLAIR images are often adequate for diagnosis. In addition, spin-echo T1-weighted axial or coronal images can be useful in following the course of the thrombosed cortical vein, as well as for excluding entry and exit phenomena as the cause for luminal hyperintensity. Diffusion-weighted images can verify that the sulcal hyperintensity on FLAIR images is not related to an acute cortical infarction. Finally, 2D time-of-flight MR venography (acquired in the coronal plane) can establish the presence or absence of flow signal intensity in a cortical vein that is visualized on the anatomic images.

Conflict of interest: None
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Ethical approval: Written consent was obtained from the patient's relatives for the publication of this case report and accompanying images.

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