Isolated Cortical Vein Thrombus Presenting with Subdural Hematoma

Sir,

Cerebral vein and dural sinus thrombosis (CVST) is a treatable, grossly underdiagnosed and potentially life-threatening condition with death rates ranging from 5% to 30% in different study series.[1] Its variability in clinical and radiological presentation make it a diagnostic and therapeutic challenge. Clinically a patient with CVST can present with symptoms and signs which are acute, or subacute, and may be secondary to increased intracranial hypertension due impaired venous drainage or related to focal brain injury from venous ischaemia or haemorrhage. Isolated cortical vein thrombosis without sinus involvement is uncommon. Subdural Hematoma (SDH) in cortical venous thrombosis (CVT) is uncommon as well with less than 15 cases reported. We report a case, where a previously healthy 54-year male was admitted with left-sided headache in the preceding week with intermittent episodes of irrelevant talk. On examination he was found to have naming difficulties, visual and colour agnosia, right homonymous hemianopia, with normal spinocerebellar, sensory, autonomic and other cranial nerve examination. He had been evaluated prior to admission with MR imaging which had picked up an occipital infarct with SDH in the left occipital region. On the day of admission, the patient developed a generalised tonic clonic seizure. MRI brain with MR venogram was repeated, which were found to be showing the same left occipital infarct with SDH with the major venous sinuses being normal [Figure 1a-c]. Backed by a strong clinical suspicion and the imaging characteristics of the infarct, the patient underwent CT angiogram and venogram which showed a linear hyperdense vessel like structure within the infarct with a few filling defects noted within, probably a CVT [Figure 1d]. The patient was initiated on oral anticoagulation bridged by enoxaparin, and discharged with his INR in therapeutic levels. At discharge the patient's anomia, agnosia and visual defects had resolved.

Isolated CVT (ICVT) without dural sinus involvement is rare.[2] The clinical presentation of ICVT overlaps with that of CVST, without the presence of signs of increased ICT such as headache and papilledema, presumably because the venous outflow through the large sinuses are not affected.

Association of CVST with subarachnoid haemorrhage had been well established. Oda *et al*. found 3% of SAH was caused by CVT.[3] The postulated mechanisms are as follows: (a) the rupture of venous parenchymal haemorrhagic infarcts into the subarachnoid space, (b) venous hypertension and subsequent rupture of dilated, valve less, thin-walled, bridging subarachnoid cortical veins devoid of smooth muscle fibres, and (c) a local inflammatory response caused by CVT, which would increase the vascular permeability allowing for extravasation of blood into the subarachnoid space. ICVT associated with subarachnoid haemorrhage has also been established.[4]

However, the association between CVT and SDH is controversial: whether the SDH is a coincidental finding, or secondary to the CVT, or iatrogenic, secondary to treatment with anticoagulation is yet to be established. Bansal *et al*. reviewed literature with analysis of the then available nine case reports of SDH in association with CVT, and discussed cases which were either concomitantly present with SDH or SDH which developed following treatment. The probable mechanism proposed in his own case was possibly due to rupture of bridging veins resulted from high backpressure by the obstructed thrombosed vein.[5] In case reports of Zupan *et al*.[6] and Fabricias *et al*.[7], it is suggested that the presence of SDH in CVT was coincidental secondary to an underlying

cause. Akins *et al*., in his literature review, after manometric analysis presents that the hemodynamic consequences of CVST found in one of their patients included flow reversal in the transverse sinus, venous stasis, delayed drainage of the vein of Labbe, and elevated venous pressures. While the precise source of subdural bleeding had not been identified, they attributed the SDH venous bleeding to have been caused by cortical venous hypertension and associated venous engorgement.[8] We reviewed the above quoted case reports and analysed the side of SDH and the side of the CVT and found that in all the cases the side of the CVT corresponded with the side of the SDH, or with the definite presence of central sagittal thrombus. This would suggest a probable causal relationship between CVT and SDH though the mechanism seems uncertain at present.

We here, after review of the available literature, present a very rare of ICVT presenting with SDH, who subsequently recovered with anticoagulation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

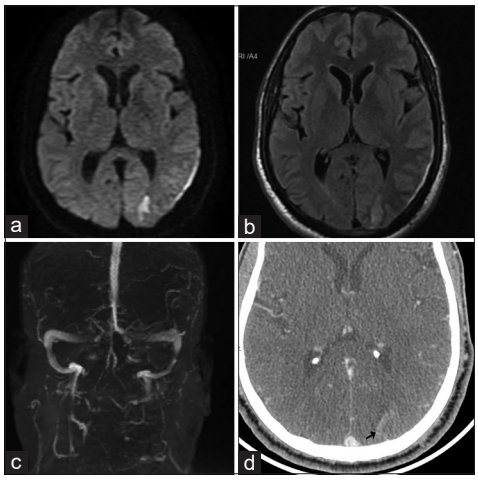

Figure 1: (a) Diffusion-weighted image of the patient showing left occipital infarct; (b) T2-weighted FLAIR image showing the thin rim of SDH in the left cerebral convexity with the occipital infarct; (c) the dural venous sinuses showing normal flow; (d) CT angiogram delayed phase showing a linear hyperdense vessel-like structure within the infarct with a few filling defects noted within (black arrow)

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Conflicts of interest

There are no conflicts of interest.

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