

Walking the tight rope of anticoagulation in the context of bleeding in cerebral venous thrombosis (CVT)

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CASE PRESENTATION:

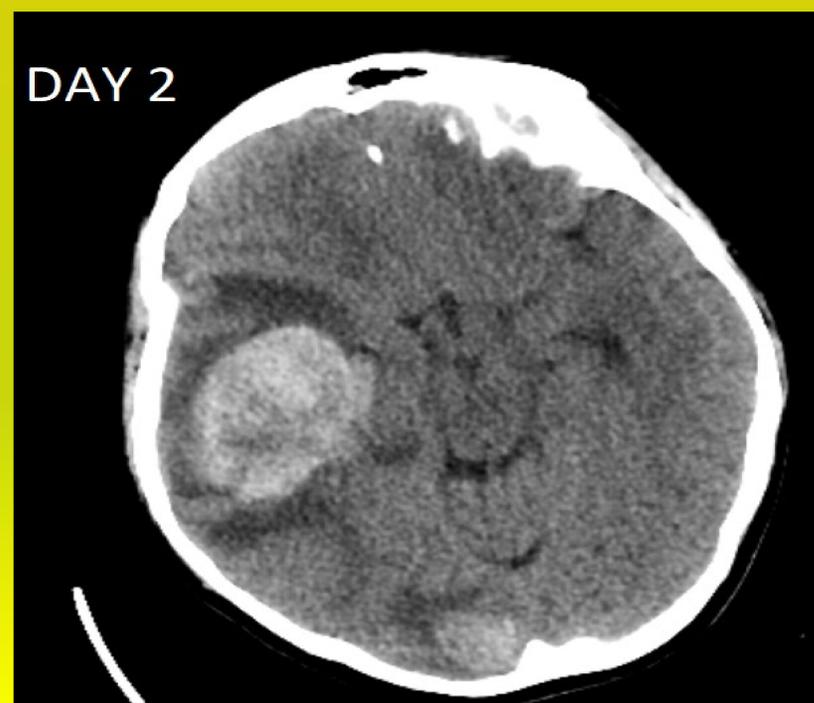
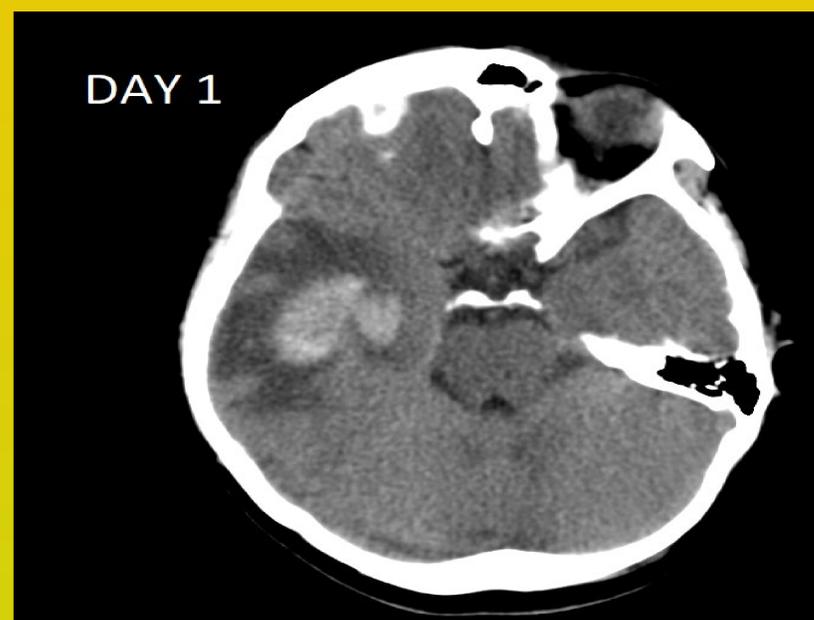
A 32-year-old female, who was a known case of systemic lupus erythematosus (SLE) presented to the emergency department with complaints of acute onset of headache associated with projectile vomiting. She was on mycophenolic acid, hydroxychloroquine and prednisolone and had received rituximab 4 months prior to this attendance. She was hemodynamically stable. Here initial blood results were nil acute (Hb 105g/L, WCC $7.4 \times 10^9/L$; platelets $233 \times 10^9/L$, CRP 19mg/L).

CT head was done since as a SLE patient she was predisposed to thromboembolic episodes.

CT showed extensive acute parenchymal haemorrhage with oedema within the right temporal and occipital lobes with non-occlusive thrombus in the right transverse sinus extending into the sigmoid sinus, with small linear filling defects in superior sagittal sinus.

After due consideration, since no other cause of haemorrhage was apparent except the non-occlusive thrombi, IV heparin was started. After 24 hours, patient had a drop in GCS. Repeat venogram showed an increase in the size and burden of the thrombi with multifocal haemorrhages. Heparin was stopped and patient was treated with cyclophosphamide and high dose steroids. Ischemic insult worsened in the follow up scans and patient was put on end-of-life pathway.

Images showing the progression of intracranial bleed



KEY LEARNING POINTS:

- The 2017 European Stroke Organization guidelines for the diagnosis and treatment of cerebral venous thrombosis, recommend heparin at therapeutic dosage to treat adult patients with acute CVT, including those with an intracerebral haemorrhage at baseline. **The presence of haemorrhagic venous infarction, intracerebral haemorrhage, or isolated subarachnoid haemorrhage are not contraindications for anticoagulant treatment in CVT** ⁽¹⁾.
- Autoimmune vascular injury in SLE may predispose to accelerated atherosclerosis from excessive oxidative stress and dysfunctional proinflammatory proteins. Glucocorticoids also promote hyperlipidaemia, diabetes, and obesity; all associated with worsening risk factors for thrombotic events ⁽²⁾.
- Thunderclap headache is often equated to subarachnoid haemorrhage (SAH), though there are a plethora of causes that need to be considered like reversible cerebral vasoconstriction, cerebral infection, cerebral venous thrombosis, dissection, pituitary apoplexy etc. This case emphasises the importance to consider these differentials apart from the usual suspect – SAH ⁽³⁾.

REFERENCES:

- (1) PMID 28833980
- (2) PMID 19454606
- (3) PMID 26252591

