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Intracerebral haemorrhage due to cerebral venous sinus thrombosis

Sir,

Cerebral venous sinus thrombosis (CVST) is a common cause of stroke in young in India especially during post-partum period.1 It has diverse clinical presentations including headache, seizures and focal neurological deficit in various combinations. In the causes of intracerebral haemorrhage (ICH), CVST is not mentioned in standard text. A Medline search using terms ICH and CVST revealed only four articles comprising of eight patients.2–5 In last 3 years we have managed 33 patients with CVST and three of them had ICH and were initially misdiagnosed as aneurysmal or arteriovenous venous malformation (AVM) bleed. We report these patients and review the contemporary literature.

Case no 1

A 65-year-old female presented with sudden onset of left sided headache for first time in life 2 weeks back associated with vomiting. For 2 days she was unable to recognize her relatives and gradually became comatose. Her blood pressure was 120/86 mm of Hg and Glasgow Coma Scale (GCS) score 7. She had bilateral papilloedema. There was no focal weakness. Tendon reflexes were brisk and plantar bilaterally extensor. Her systemic examination was normal. Haematology, serum chemistry and coagulation profile were normal. CT scan revealed left temporal and right occipital haematoma (Figure 1A). D Dimer was positive, protein C low (57%), protein S normal and MTHFR and Factor V Leiden mutations were absent. Arterial phase on 4-vessel digital subtraction angiogram (DSA) was normal. MR venography revealed superior sagittal venous sinus and straight sinus partial thrombosis (Figure 1B). Patient was treated with subcutaneous low molecular weight heparin (LMWH) and improved by second week. At 3 weeks she had left homonymous haemianopia.

Case no 2

A 22-year-old unmarried female had sudden onset of severe headache, projectile vomiting and became unconscious a few hours later. After 8–10 h, her consciousness improved and she was able to walk to the hospital. On examination she was conscious, BP 130/80 mmHg, pulse 102/minute and neck was stiff. She had bilateral papilloedema and right subhyaloid haemorrhage. Rest of the neurological examination was normal. She again became unconscious two days later (GCS score 9) and had mild haemiparesis. A day later her consciousness improved but was abulic. Her investigations of blood and urine were normal. CT scan revealed left frontal haematoma with intraventricular extension. DSA revealed left transverse and sigmoid sinus thrombosis (Figure 1C and D). She was treated with LMWH 2500IU twice daily. She improved in 2–3 days, her haemiplegia improved but abulia persisted at 1 month, which also improved at 3 months.

Case no 3

A 42-year-old female developed severe uterine bleeding after a gynaecological procedure and was prescribed oral estrogen preparations. She developed severe headache, vomiting, seizures and altered sensorium two days later. One day later she developed left partial seizures with secondary generalization lasting for 10–15 min. On examination she was drowsy (GCS score 11), had bilateral papilloedema and mild left sided haemiparesis with brisk tendon reflexes. Her CT scan revealed left parietal haematoma and MR venography revealed superior sagittal and straight sinus thrombosis (Figure 1E and F). The patient was treated with LMWH, phenytoin 300 mg and later acitrom 2 mg daily. She improved completely by day 10. Her abdominal ultrasound revealed hydatid cyst in the liver.

In our study 3 out of 33 CVST patients presented with medium size ICH. In the available literature this seems to be rare.2–5 The CT scan of the report of five cases by Singh and Chakra reveals haemorrhagic infarctions rather than primary ICH.2 In another report one of the patients had associated aneurysm
and CVST was diagnosed at autopsy only. This patient underwent stenting with high dose heparin therapy, which might have contributed to ICH.4 Lobar haemorrhage in young is often attributed to aneurysm or AVM. All our patients underwent emergency DSA for excluding these possibilities. Presence of bilateral haematoma in CT may be a clue for CVST. Haemorrhagic infarction is common in CVST and is reported in 35–50% of patients, which is attributed to blockade of venous sinuses. There is increase in venous and capillary pressure resulting in diapedesis of RBC and subsequent rupture of small vessels.5 Presence of ICH may be an extension and exaggeration of above-mentioned sequence. Rapidity of venous thrombosis and lack of fibrinolysis may contribute to occurrence of ICH. All our patients had acute CVST for <15 days. We could not find common underlying predisposing or precipitating factors in these patients although one patient each had low protein C and estrogen therapy. Haemorrhagic transformation in CVST is regarded a poor prognostic factor.7 One of the earlier reported patient died and one had poor outcome.2,4 All our patients however improved completely following anticoagulation, which reiterates the safety and efficacy of anticoagulation in CVST.

It can be concluded that ICH may be a rare presentation of CVST and this possibility should be considered in appropriate clinical setting.

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Transfer of patients with acute kidney injury to specialist renal services—physiological early-warning systems, applied prior to transfer from outside hospitals, can identify those at risk of deterioration*

Sir,
The UK National Service Framework for Renal Services—a template for both clinical practice and organization—acknowledges the variety of settings in which acute kidney injury (AKI) may be managed. However, specialist renal management may well be needed for those whose case complexity or severity requires transfer from non-specialist hospitals or for those receiving single-organ (renal) support on the ICU. Unfortunately, as demonstrated in this journal, significant delays in transfer do exist and may consume ICU resources and adversely affect patient outcomes. Although timely renal management is the core goal, the arrival of such patients on a specialist unit with unheralded critical illness is a potential disaster in terms of both immediate patient safety and the unexpected burden that this may place on local critical care services. Our challenge is how best to decide, prior to actual transfer, whether such patients should be managed solely by renal services or in conjunction with critical care.

With this in mind, we assessed the utility of a generic illness severity scoring tool as a predictor of later escalation of care in all off-site AKI patients already accepted for transfer to our regional renal unit. In the 1 year from 15th October 2005, a SOFA score, a composite assessment of organ dysfunction, was calculated for all such patients at the time of referral. The subsequent need for escalation to higher-level care was determined, retrospectively. AKI patients accepted onto our unit with an initial SOFA score ≥ 6 carry a higher risk of early escalation of care. These findings might well be regarded as intuitive but it is only recently that the UK National Institute of Health and Clinical Excellence has had to reinforce the importance of well-defined protocols for the early recognition and management of the acutely unwell in-patient. Although our tool cannot determine the most appropriate venue for transfer, it can augment subjective assessments of illness severity, trigger preemptive responses, such as early liaison with critical care and senior colleagues and warn of the

*The results presented in this paper have not been published previously in whole or part, except in abstract format (UK Renal Association, annual meeting, May 2007).