Sudden-Onset Headache And Seizures In A Young Man
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Citation

Abstract
We report a case of a 23 year-old Malay man who was admitted after a generalized tonic-clonic seizure. He had a second seizure in the emergency room. We discuss the diagnosis and treatment of the origin of the seizures.

CASE REPORT
A 23 year-old Malay man was admitted after a generalized tonic-clonic seizure. He had a second seizure in the emergency room. A known infrequent migraineur, he complained of severe dull occipital headaches of recent onset, different from his usual migraines. He denied head injury. Clinical examination was unremarkable. CT brain was significant for hemorrhage in the left frontal lobe (Figure 1A). In view of the absence of previous history of seizures, head trauma or cerebrovascular risks, as well as the presence of new-onset severe headaches, MRI brain was performed, showing infarcts in both frontal lobes, with hemorrhage in the left anterior frontal lobe (Figure 1B & C). Magnetic resonance venogram (MRV) showed filling defects in the superior sagittal (Figures 1E-F and Video) and left lateral venous sinuses (Figure 1D), consistent with the diagnosis of cerebrovenous thrombosis (CVT). He was given phenytoin, anticoagulated with intravenous heparin, and later discharged with oral warfarin for 6 months. He remained well. Procoagulant and autoimmune screens were negative, as was CT scan of the sinuses.

Figure 1
Figure 1: CT scan (1A) showing small left frontal hemorrhage (thick white arrow). Fluid attenuation inversion recovery (FLAIR) and diffusion weighted (DWI) sequences (1B and C) showing frontal infarcts (2 in left frontal, 1 right frontal, unfilled white arrows) bilaterally. Magnetic resonance venograms, MRV, showing left lateral sinus (1D, thin white arrow) and superior sagittal sinus (1E, dotted white arrows) filling defects, consistent with cerebral venous thrombosis. Figure 1E is a sagittal view of the venous phase of angiography showing superior sagittal sinus thrombosis (short black arrows).

Video: Video-angiogram showing filling defect in the superior sagittal sinus, consistent with thrombosis.

CVT, which accounts for less than 1% of all strokes [1], can affect all ages, from the neonate to the elderly. Its presentation and neuroimaging picture are diverse, and its potential for recovery is extraordinarily good if recognized early and definitive therapeutic measures instituted early [2]. Four main patterns of presentation emerge, the most common having focal neurologic deficits and seizures, with or without headaches. The second pattern is that of isolated
intracranial hypertension, the third that of a subacute encephalopathy and the last pattern is that of a cavernous sinus thrombosis [2]. Comatose patients with intracranial hemorrhage (ICH) on presentation have the worst prognosis [1]. First-line treatment for CVT is anticoagulation, with some requiring thrombolysis [1,2], even in the presence of ICH. Oral anticoagulation is continued for 3 months in uncomplicated CVT and up to 12 months in patients with acquired or inherited thrombophilia [1]. Recurrence is rare, reports ranging from 6 to 12% [1,3], with the first 12 months being the vulnerable period. Outcome after CVT is heterogeneous. The disease has been recognized for more than a century, and was almost invariably fatal. With the advent of anticoagulation, survival has improved dramatically, but outcomes still vary, ranging from comatose or hemiplegic patients to those who, like our patient, recover dramatically without sequelae [2].

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References
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