An interesting case of SAH presenting as INITIAL manifestation of CVT
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Abstract: ABSTRACT Cerebral venous thrombosis is difficult to diagnose because it presents with a wide variety of clinical symptoms and signs. It is further more complicated to diagnose if it presents as subarachnoid hemorrhage initially. Here we report a case of Cerebral venous thrombosis which presented to us initially as subarachnoid hemorrhage. 

Keyword: cerebral venous thrombosis, subarachnoid hemorrhage.

CASE REPORT: A 45 year male patient was brought by his brother with complaints of seizures –generalised tonic clonic, 2 episodes since morning. Patient was conscious in between seizures. Following the second episode, patient was agitated and he was brought in agitated state to us. The seizure episode was associated with bladder incontinence. No history of fever, headache, vomiting, trauma. No history of previous similar episode. Patient was an alcoholic - last intake 3 days back.

On examination, patient was agitated, vitals were stable. No pallor, no icterus, no cyanosis or clubbing, no lymphadenopathy, no pedal edema. Cardiovascular, respiratory, abdomen system were normal. patient was treated with lorazepam - on re-examining the patient 6 hours later, patient was conscious, oriented and had weak right handgrip. Rest of the CNS examination was normal. Complete blood count, renal function test, liver function test, serum electrolytes were normal. CT brain showed hyperdense foci in left sulcal space of left parietal region with no midline shift-suggestive of Subarachnoid hemorrhage

Next day, patient had seizures of right upper limb and lower limb followed by inability to use them. Examination revealed right hemiplegia with right UMN facial nerve palsy. patient underwent MRI with MRA & MRV- it showed SUPERIOR SAGITTAL SINUS THROMBOSIS, bilateral frontal lobe HEMORRHAGE – left greater than right and SUBARACHNOID HEMORRHAGE. It didn’t showed any evidence of aneurysms.
MRI report
Patient was treated with heparin along with warfarin with target INR of 2-3. Patient's weakness improved and by 6 months he was able to walk well. Investigations done for hypercoagulable state-APLA, ACLA, LAC, ANA, protein C & S deficiency, anti-thrombin III deficiency, Prothrombin20210 mutation, factor v leiden mutation were negative.

Discussion:
Excluding trauma, SAH is caused by ruptured saccular aneurysm in majority of cases. Other causes include arteriovenous malformation, dural arterial-venous fistula and extension from primary intracerebral hemorrhage. CVT is not listed as a cause of SAH so far. But, SAH may be a manifestation of CVT, which should therefore be considered in the diagnostic workup of SAH, especially when the basal cisterns are not involved. In this case too, the SAH didn’t involve the basal cisterns. SAH can be attributed to CVT, provided screening for other causes of SAH (aneurysms, AV fistula, AV malformations) is done and they are ruled out. The exact mechanism of SAH in CVT is not known. The sinus thrombosis may lead to increased venous pressure, rupture and bleed into the subarachnoid space, producing SAH. Once CVT is diagnosed, regardless of the presence of hemorrhage, ANTICOAGULATION has to be given. Hence the management differs significantly once the diagnosis of CVT is established. The duration of anticoagulation depends on whether the patient has a transient factor which predisposed to thrombosis (pregnancy) or he has an inherited thrombophilia.

CONCLUSION:
CVT has to be considered in the diagnostic workup of SAH. Particularly when basal cisterns are not involved. Other causes of SAH, like aneurysms, should be ruled out, before considering anticoagulation in SAH with CVT.

REFERENCES:


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