# 1

From bleed outside of brain,
Crescent-shaped, from tear in vein,
Can cause shift, and cross bone lines,
And on CT, see bright it shines.

***Hint #1***

The answer is in your potential space.

***Hint #2***

Do not falsely localize the wrong answer.



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# Subdural Hematoma

Characterized by blood in the potential space between the arachnoid membrane and the dura mater, subdural hematomas are most commonly caused by the tearing of bridging veins as a result of brain movement within the skull. These veins drain from the brain surface into the dural venous sinuses. The elderly and individuals with alcoholism are most prone to developing subdural hematoma.

Subdural hematomas can occur both acutely and chronically, each with significantly different clinical presentations. Acute subdural hematoma often occurs after acute closed-head trauma and by definition becomes symptomatic within 72 hours. These patients often present with severe headache and acute alterations in consciousness. Depending upon the size and location of the subdural hematoma, patients could exhibit a variety of presentations on neurological examination. For example, a patient with a large subdural hematoma over the lateral convexity of the dominant hemisphere would most likely have contralateral hemiparesis, aphasia, and ipsilateral gaze deviation.

These signs are a result of underlying cerebral edema, caused by the subdural hematoma. Clinically, this presentation can be easily confused with an acute dominant hemispheric infarction in the middle cerebral artery territory. A carefully documented history of preceding head trauma, in addition to a CT scan, and severe headache can often differentiate between the two.

In severe cases, where the edema is severe enough to compress the ipsilateral oculomotor nerve exiting the midbrain, patient will have ipsilateral pupillary dilation. However, do not be fooled as these patients can also develop uncal herniation, producing compression of the contralateral oculomotor nerve and cerebral peduncle against the tentorium. Clinically, this phenomenon, known as *Kernohan's notch phenomenon*, appears as contralateral pupillary dilation and the "falsely" localizing ipsilateral hemiparesis.



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Chronic subdural hematomas more often occur in the elderly, alcoholic, and epileptic patients who are more susceptible to such injuries because of poor balance and gait issues. The relative brain atrophy of such patients puts them at a higher risk of developing subdural hematomas, even with trivial head trauma, because of the "stretching" of the bridging veins to account for the increased distance between the atrophied brain and the dura. As opposed to the predominantly focal symptomatology of acute subdural hematomas, the patients with chronic hematomas have vague and nonspecific symptoms like mild headaches, increased forgetfulness, gait ataxia, and so on. Both acute and chronic subdural hematomas can present with seizures and status epilepticus as well.

On non-contrasted CT, subdural hematomas typically appear as a crescent-shaped hyperdensity (acute) or isodensity-hypodensity (chronic) that commonly crosses suture lines. Chronic subdural hematomas are often isodense to cerebrospinal fluid (CSF) and hypodense to adjacent cortex, and hence they can be difficult to identify on a CT scan.

The treatment of acute subdural hematoma depends upon its clinical presentation and size. If the patient is on anticoagulation, reversal of the specific anticoagulant is recommended. For patients with rapidly declining consciousness, large hematoma ($>$10 mm), and midline shift ($>$5 mm), emergent neurosurgical consultation is warranted. Such patients often require emergent craniotomy with evacuation of the hematoma. The management of chronic subdural hematoma is not straightforward. Craniotomy with evacuation may be recommended for larger hematomas. Middle meningeal artery embolization is a new noninvasive treatment option available for such patients, which may decrease the likelihood of re-accumulation of the hematoma.



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