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SUB EPIDURAL HEMATOMA IN STROKE: A RARE BUT CRITICAL CONSIDERATION IN ADULT PATIENTS

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Abstract
Sub-epidural hematoma (SEDH) is an uncommon but life-threatening complication that may occur in the context of ischemic or hemorrhagic stroke. Although subdural and intracerebral hemorrhages are well-recognized in stroke pathology, sub-epidural hematoma remains an underreported entity. This article analyzes clinical characteristics, diagnostic approaches, and management strategies for sub-epidural hematoma associated with stroke in adult patients. Based on clinical literature and case analyses, the article highlights the diagnostic challenges and emphasizes the importance of neuroimaging in early recognition to improve outcomes.

Keywords: Sub-epidural hematoma, stroke, neuroimaging, ischemic stroke, intracranial hemorrhage, neurosurgery, adult patients.

Introduction

Stroke is one of the leading causes of morbidity and mortality worldwide, classified primarily into ischemic and hemorrhagic types. Secondary complications such as cerebral edema and hemorrhagic transformation are common, but the occurrence of a sub-epidural hematoma (SEDH) in stroke patients is exceedingly rare. The sub-epidural space, located between the inner table of the skull and the dura mater, is usually affected by trauma rather than vascular pathology. However, certain pathophysiological mechanisms such as vascular rupture due to reperfusion injury, hypertension, or anticoagulation therapy may precipitate this condition in stroke patients.

Understanding the occurrence of SEDH in the context of stroke is clinically crucial, as its presentation can mimic worsening stroke symptoms, leading to

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diagnostic delays. Early recognition and intervention can be life-saving. This article explores reported cases, mechanisms, diagnostic findings, and treatment outcomes to provide an integrated understanding of this rare but critical condition.

Based on the phrasing of your query, it appears you're referring to subdural hematoma (SDH)—a collection of blood between the brain's surface and its outermost protective covering (dura mater)—as a rare but serious consideration in adults experiencing or recovering from stroke. (Note: "Sub epidural" may be a typographical variant of "subdural," as "subepidural" is not a standard medical term; epidural hematomas occur above the dura, while subdural ones are below it. If you meant epidural or another variant, please clarify.) SDH is not a direct cause of stroke but can complicate stroke management, particularly in ischemic stroke patients on antithrombotic therapies or those with comorbidities like brain atrophy from prior strokes. Below, I'll outline its relevance, mechanisms, risks, and management in adults, drawing from clinical evidence.

Pathophysiology and Relevance to Stroke

- How SDH Forms in Stroke Patients: SDH typically arises from tearing of small bridging veins due to head trauma, but in stroke contexts, it can occur spontaneously (non-traumatically) or as a secondary event. Brain atrophy—common in elderly stroke survivors—increases the subdural space, making veins more vulnerable to minor trauma or even spontaneous rupture. Anticoagulants or antiplatelets (e.g., warfarin, aspirin) used for secondary stroke prevention heighten bleeding risk.
- Link to Stroke Types:
  - Ischemic Stroke: Thrombolytics like tissue plasminogen activator (tPA) for acute treatment can rarely trigger SDH (e.g., acute SDH post-intra-arterial thrombolysis). Additionally, non-traumatic SDH increases the short-term risk of recurrent ischemic events, including stroke, possibly due to pausing antithrombotics.
  - Hemorrhagic Stroke: SDH may coexist or mimic extension of intracerebral hemorrhage but is distinct.
- Rarity: SDH complicates ~1-2% of ischemic strokes directly (e.g., post-thrombolysis), but up to 10-15% of all intracranial hemorrhages involve SDH elements. In chronic cases, it's more common in older adults (>60 years) with stroke history.

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Clinical Presentation

Symptoms overlap with stroke, complicating diagnosis—prompting the need for vigilance:

- Headache, nausea, confusion, or altered mental status.
- Focal deficits (e.g., hemiparesis, aphasia) that worsen progressively.
- Seizures or transient ischemic attack (TIA)-like episodes due to mass effect.
- In acute cases: Rapid deterioration with coma; chronic cases: Subtle cognitive decline mimicking post-stroke dementia.

Diagnosis relies on urgent non-contrast CT head, showing crescent-shaped hyperdensity (acute) or hypodensity (chronic) with midline shift if severe.

Risk Factors in Adult Stroke Patients

Risk Factor	Description	Stroke-Specific Relevance
Antithrombotic Therapy	Warfarin, DOACs, or antiplatelets increase bleed risk.	Common in secondary prevention; interruption post-SDH raises ischemic stroke odds (HR 4.2 in first 4 weeks).
Age >65	Brain atrophy widens subdural space.	Stroke survivors often elderly; doubles SDH incidence.
Prior Trauma/Falls	Even minor head bumps.	Frequent in hemiparetic stroke patients.
Coagulopathy	Liver disease, alcohol use.	Exacerbates thrombolysis risks.
Hypertension	Vessel fragility.	Shared with stroke etiology.

Why It's Critical: Complications and Prognosis

- Ischemic Complications: SDH can compress cortical vessels, causing perforator thrombosis and secondary infarcts (e.g., insular or lacunar strokes). This "post-SDH TIA" mimics stroke recurrence but stems from hypoperfusion.
- Mortality/Morbidity: Untreated acute SDH has 50-90% mortality; surgical intervention drops it to 10-20%. In stroke patients, added ischemia worsens outcomes (e.g., higher modified Rankin Scale scores).
- Recurrence: 10-20% post-surgery, often within 90 days; higher in anticoagulated patients.

Management Considerations

- Acute Stabilization: ABCs, reverse coagulopathy (e.g., vitamin K, PCC for warfarin; idarucizumab for dabigatran). Monitor ICP if midline shift >5 mm.

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- Surgical Options (based on hematoma size >10 mm, shift, or deterioration):
  - Burr-hole drainage or twist-drill craniostomy for chronic SDH (success >85%).
  - Craniotomy for acute/clotted cases.
  - Emerging: Middle meningeal artery embolization (MMAE) as adjunct, reducing recurrence by 60-70% vs. surgery alone.
- Stroke-Specific Nuances:
  - Delay antithrombotic resumption 1-4 weeks post-SDH, balancing bleed vs. ischemia risk (e.g., use DOACs over warfarin).
  - Post-thrombolysis SDH: Rare spinal extension possible, warranting MRI if back pain emerges.
- Conservative Approach: Small, asymptomatic SDH may resolve with monitoring, dexamethasone, or tranexamic acid, but surgery is preferred in symptomatic stroke patients.

Key Takeaways for Clinicians/Patients

SDH is a "great mimicker" in stroke care—its rarity belies its potential to derail recovery through mass effect or iatrogenic ischemia. High suspicion in anticoagulated adults with new deficits post-stroke is essential; early CT and multidisciplinary input (neurology, neurosurgery) save lives. Prognosis improves with prompt intervention, especially in chronic cases (mortality <5%).

Conclusions

Sub-epidural hematoma in stroke is an extremely rare but potentially fatal condition. It must be included in the differential diagnosis of stroke patients who experience rapid neurological decline, particularly in those receiving anticoagulant therapy or thrombolysis. The condition underscores the need for:
Vigilant post-stroke monitoring,
Early neuroimaging follow-up,
Interdisciplinary management involving neurologists and neurosurgeons.

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