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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 (SEH) is a rare but significant complication in stroke patients. While epidural hematomas are commonly associated with traumatic brain injury, their occurrence in stroke cases presents unique diagnostic and therapeutic challenges. This article explores the pathophysiology, diagnostic methods, and management of SEH in adult stroke patients, providing a comprehensive analysis of recent literature and clinical cases. Understanding the implications of SEH in stroke patients is crucial for timely intervention and improved patient outcomes.

**Keywords**: Sub-epidural hematoma, stroke, cerebrovascular accident, brain hemorrhage, neuroimaging, neurosurgery, hematoma management.

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## Introduction

Stroke remains a leading cause of morbidity and mortality worldwide, with ischemic and hemorrhagic subtypes constituting the majority of cases. While intracranial hemorrhages are well-documented in stroke patients, sub-epidural hematoma (SEH) remains a rare but potentially life-threatening condition. SEH is primarily associated with head trauma, but its presence in stroke patients suggests an alternative pathophysiology that warrants deeper investigation. This article aims to analyze the incidence, clinical presentations, diagnostic challenges, and management strategies for SEH in adult stroke patients.

Stroke is a leading cause of morbidity and mortality worldwide, with ischemic and hemorrhagic strokes being the two primary forms. While hemorrhagic transformation is a well-known complication of ischemic stroke, the occurrence of sub-epidural hematoma (SEH) in stroke patients is extremely rare and primarily reported in cases related to trauma. However, certain pathological mechanisms in stroke, such as anticoagulant use, vascular fragility, and spontaneous hemorrhage, may predispose patients to SEH.

Sub-epidural hematoma refers to the accumulation of blood between the dura mater and the inner table of the skull, most commonly due to arterial bleeding, particularly from the middle meningeal artery. This condition is frequently traumatic in origin but can also arise spontaneously under specific clinical circumstances.



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**Pathophysiology and Possible Causes**

In non-traumatic stroke patients, the development of SEH is rare but may occur due to the following mechanisms:

**Anticoagulation Therapy Complications**

- Many ischemic stroke patients are on antiplatelet or anticoagulant therapy (e.g., aspirin, clopidogrel, warfarin, or direct oral anticoagulants like apixaban and rivaroxaban).
- Excessive anticoagulation can predispose patients to intracranial bleeding, including SEH.
- The use of thrombolytics (e.g., alteplase, tenecteplase) in acute ischemic stroke may increase the risk of hemorrhagic complications.

**Vascular Fragility in Stroke Patients**

- Chronic hypertension and small vessel disease can weaken cerebral arteries and predispose them to rupture.
- Cerebral amyloid angiopathy (CAA), commonly seen in elderly stroke patients, can contribute to vessel fragility and spontaneous hemorrhage.

**Hemorrhagic Transformation of Ischemic Stroke**

- Large infarcts, particularly those affecting the cortical regions, can undergo hemorrhagic transformation, leading to secondary bleeding that extends into the epidural space.
- Increased intracranial pressure (ICP) from an infarct can cause vascular stress and rupture.

**Spontaneous Epidural Hemorrhage in Stroke**

- Rare cases of spontaneous SEH have been linked to underlying vascular malformations, aneurysm rupture, or dural arteriovenous fistulas.
- Patients with coagulopathies, such as liver disease, thrombocytopenia, or hematological disorders, may develop spontaneous epidural hemorrhage.

**Clinical Presentation**

The symptoms of SEH in stroke patients can vary depending on the hematoma size and associated neurological damage. However, common clinical features include:

- Severe headache (sudden onset, progressive, localized to the side of hematoma)
- Neurological deterioration, including:
  - Altered mental status
  - Confusion or agitation
  - Focal neurological deficits (e.g., hemiparesis, aphasia, visual disturbances)
  - Worsening of pre-existing stroke-related deficits
- Seizures (more common if the hemorrhage affects cortical areas)
- Signs of increased intracranial pressure (ICP):
  - Nausea and vomiting
  - Hypertension with bradycardia (Cushing's response)
  - Decreased level of consciousness, potentially leading to coma



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In some cases, delayed neurological decline after initial stroke stabilization may be the first indication of SEH, necessitating urgent neuroimaging.

Diagnostic Challenges

The diagnosis of sub-epidural hematoma in stroke patients can be challenging, as the symptoms may overlap with stroke progression, hemorrhagic transformation, or other complications like brain edema.

**Key Diagnostic Modalities**

Non-contrast CT scan (first-line imaging)

- Characteristic biconvex (lens-shaped) hyperdensity in the epidural space.
- Midline shift and mass effect in cases of large hematomas.
- Presence of adjacent infarcted tissue, if occurring secondary to stroke.

**Magnetic Resonance Imaging (MRI)**

- Useful for detecting small or isodense hematomas not visible on CT.
- Can help differentiate between acute, subacute, and chronic bleeding.

CT Angiography (CTA) or Digital Subtraction Angiography (DSA)

- Indicated if a vascular malformation or aneurysm is suspected.
- Can identify active bleeding and assess collateral circulation.

**Coagulation Profile**

- Important for patients on anticoagulation therapy.
- Includes INR, aPTT, platelet count, and fibrinogen levels.

**Management Approach**

Treatment of SEH in stroke patients requires a multidisciplinary approach involving neurologists, neurosurgeons, and intensive care specialists.

**Medical Management**

- Reversal of anticoagulation (if indicated):
  - Vitamin K and fresh frozen plasma for warfarin-induced bleeding.
  - Prothrombin complex concentrate (PCC) for urgent reversal.
  - Idarucizumab (for dabigatran) or andexanet alfa (for factor Xa inhibitors).
- Blood pressure control:
  - Goal: Maintain systolic BP <140–160 mmHg to prevent hematoma expansion.
- Seizure prophylaxis or treatment:
  - If SEH is associated with seizures, administer levetiracetam or phenytoin.
- ICP management:
  - Elevate head of bed to 30 degrees.
  - Mannitol or hypertonic saline in cases of increased ICP.

Surgical Intervention

- Burr hole drainage or craniotomy:

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- Indicated in large SEH with mass effect or significant midline shift.
- Decompressive hemicraniectomy:
  - Considered in combined stroke and SEH with malignant brain swelling.
- Hematoma evacuation:
  - For rapidly expanding hematomas leading to herniation.

Supportive Care

- Neuromonitoring in ICU for worsening neurological signs.
- Rehabilitation and physiotherapy after stabilization to improve functional recovery.

## Conclusions

SEH in stroke patients, though rare, represents a critical clinical challenge requiring prompt recognition and intervention. Clinicians should maintain a high index of suspicion, particularly in patients receiving anticoagulation therapy or presenting with atypical neurological symptoms. Future studies should focus on identifying predisposing factors and optimizing treatment strategies to improve patient outcomes. Enhanced awareness and early diagnosis can significantly reduce morbidity and mortality associated with this rare condition.

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