Subacute subdural hematoma

- Effectiveness of subdural evacuating port system (SEPS) and middle meningeal artery embolization (MMAE) for chronic subdural hematomas - a multicenter experience
- Effectiveness of subdural evacuating port system (SEPS) and middle meningeal artery embolization (MMAE) for chronic subdural hematomas
- Endoscopic subdural membranectomy for multi-septated chronic subdural hematoma: Finding a safe solution when middle meningeal artery embolization is not feasible
- The safety and feasibility of anticoagulant resumption following middle meningeal artery embolization in patients with subacute subdural hematomas
- Adjunctive middle meningeal artery embolization for non-acute subdural hematoma: A GRADE-assessed meta-analysis and trial sequential analysis on randomized trials
- Subacute Subdural Hematoma Following Ventriculoperitoneal Shunt Procedure: A Case Report
- Brain sagging syndrome: Occult cerebrospinal fluid leakage as a cause of failed brain expansion after removal of bilateral chronic subdural hematomas
- Middle meningeal artery embolization for treatment of bilateral mixed-attenuation subdural hematomas in an infant: illustrative case

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Subacute subdural hematoma (SASDH) is known as the gradual pooling of blood in the subdural space that occurs in the period of 4-21 days from the head injury. Usually, it is caused by trauma. This collection causes compression on the brain which leads to the production of localized neurological manifestations, increased intracranial pressure, or altered level of consciousness.

Subacute subdural hematomas are a poorly individualized nosological entity, often equated clinically to chronic subdural hematomas. Yet, their neurological deterioration which is usually rapid seems to distinguish them from chronic subdural hematomas.

see Subacute traumatic subdural hematoma.

Retrospective case series

A **retrospective case series** involving **five patients** with subacute subdural hematoma (sASDH), who were managed conservatively using atorvastatin and low-dose dexamethasone without surgical intervention. It also includes a **non-systematic narrative review** of existing literature, lacking formal meta-analytic methodology [1)].

Critical Appraisal

Strengths

- The article raises an important question: can we optimize conservative treatment for sASDH in inoperable patients?

- A novel hypothesis is proposed, leveraging two commonly available pharmacologic agents.

Fatal Flaws

1. Sample Size and Selection Bias

The study is limited to **five hand-picked cases**, all of whom refused surgery. There is **no control group**, **no randomization**, and **no standardization** in patient selection. This introduces massive **selection bias** and **confounding**, rendering the findings anecdotal at best.

2. Lack of Statistical Power

With only five patients, the study is **grossly underpowered** to draw any conclusions on safety or efficacy. Even if all patients improved, the **positive predictive value is negligible**.

3. Absence of Mechanistic Evidence

The article alludes vaguely to the “possible mechanisms” of action of atorvastatin and dexamethasone but fails to elaborate with any **molecular, imaging, or biomarker-based support**. The hypothesized synergy is speculative and **not experimentally validated**.

4. Cherry-Picking Literature

The review portion pulls from only **six studies** without PRISMA methodology, inclusion/exclusion criteria, or risk-of-bias assessments. This is **not a systematic review** but rather a collection of cherry-picked studies to support a preconceived narrative.

5. Logical Fallacy: Post Hoc Ergo Propter Hoc

The authors infer that improvement after administration of atorvastatin and dexamethasone implies causality. This is a classic **post hoc fallacy**. No causation can be inferred from such a weak observational structure.

6. Ethical and Practical Concerns

Presenting this treatment strategy without rigorous evidence could **mislead clinicians**, **delay necessary surgery**, or foster **false confidence** in a pharmacological approach for a condition where deterioration can be catastrophic.

Bottom Line

The article is a speculative and weakly documented case series attempting to repurpose two drugs in the treatment of sASDH. While the intention is noble, the scientific execution is fundamentally flawed. **No clinical decisions should be influenced by this paper.** What is needed is a properly designed **randomized controlled trial**, not a narrative built on five anecdotal successes.

Case report from the HGUA

Q11755

81-year-old male brought in by his daughter due to a fall at home two days ago. He reports feeling disoriented since then, experiencing gait instability, and changes in behavior.

Left frontoparietal subdural fluid collection, isodense, with hyperdense areas related to acute/subacute subdural hematoma. It has a maximum thickness of 27 mm, causing a mass effect on the brain parenchyma and the ipsilateral ventricular system, which is compressed. It is associated with subfalcine herniation, with a displacement of the midline to the right of approximately 9 mm.

Postoperative CT Scan

1)
Liu T, Wu C, Jiang W, Liu M, Sha Z, Jiang R. Exploring conservative avenues in subacute subdural hematoma: the potential role of atorvastatin and dexamethasone as lifesaving allies. Chin Neurosurg J. 2025 Apr 2;11(1):7. doi: 10.1186/s41016-025-00393-8. PMID: 40176171.