

DELAYED HEMATOMA EXPANSION IN HEMORRHAGIC BRAIN METASTASES

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INTRODUCTION

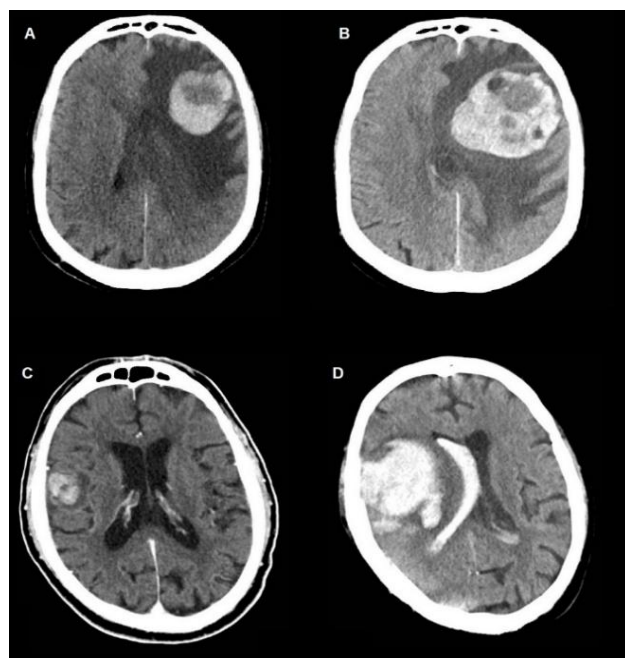
Hemorrhagic transformation represents a well-recognized complication in **brain metastases**, particularly in association with **highly vascular neoplasms** such as melanoma, renal cell carcinoma, and lung adenocarcinoma. Despite this, the clinical course and prognostic implications of hemorrhagic brain metastases remain not fully defined. We present two illustrative cases in which brain metastases with intralesional bleeding experienced significant **hematoma expansion (HE)** over the course of hospitalization.

MATERIALS AND METHODS

We conducted a **retrospective review** of two patients with **hemorrhagic brain metastases** admitted between 2024 and 2025. **Clinical data (demographics, oncologic history, comorbidities, treatments, neurological course)** were extracted from **electronic medical records**. **Brain MRI** was reviewed, and **hematoma volume** calculated with the **ABC/2 method**. **Hematoma expansion** was defined as an increase **>33%** and **>6 mL** compared to baseline **intracerebral hemorrhage (ICH)**.

RESULTS AND DISCUSSION

Variable	Case 1 (68/M) – Lung adenocarcinoma	Case 2 (73/M) – Cutaneous melanoma
Oncologic status	Stable; lung satellites + hilar LN	Stable; no extracerebral disease
Prior therapy	SRS (43 d before ICH), corticosteroids	Nivolumab (12 mo before ICH), none at onset
KPS at ICH	90	40
ICH site	Left frontal lobe	Right precentral gyrus
Baseline volume	19 mL	6 mL
HE	71 mL (day 13)	98 mL + IVH (day 3)
Comorbidities	PAD (stent 2023), ex-smoker	HTN, DM2, HCM, DVT/PE, ex-smoker
Antithrombotics	None	Apixaban for PE
Course / Outcome	Acute hemiparesis → sudden worsening; conservative care; death day 16	Acute hemiparesis → rapid decline; no reversal/ICU/surgery; death day 4



Images A and B refer to Case 1, showing baseline (A) and f-u CT (B)
Images C and D refer to Case 2, showing baseline (C) and f-u CT (D)

Our cases suggest that **hematoma expansion (HE)** — defined as hematoma growth occurring **>24h** after the index ICH— can complicate **tumor-related ICH**. Potential mechanisms include:

- **SRS-induced vascular damage** and perilesional edema (Case 1)
- **Intralesional necrosis** or **inflammatory vascular injury**,
- **Immune-mediated vascular toxicity** in patients with prior checkpoint inhibitors (Case 2),
- **Comorbidities and anticoagulation** (Case 2).

From a clinical standpoint, HE represents a **potentially preventable event**, and—unlike in primary ICH—may occur **with delay**, offering a **longer therapeutic window**. This underlines the importance of **individualized clinical and imaging surveillance** based on tumor type, prior therapies, and vascular risk factors.

CONCLUSIONS

Delayed hematoma expansion (HE) in hemorrhagic brain metastases is a severe and under-recognized complication that may occur even in otherwise stable oncologic disease. Unlike primary ICH, it can develop after a period of stability, offering a potential therapeutic window for timely detection. Vigilant monitoring is warranted in patients with recent SRS, prior immunotherapy, anticoagulation, or vascular comorbidities. Further studies are needed to define its prevalence, risk factors, and preventive strategies.