

Haemorrhagic intracranial metastasis: a diagnostic and therapeutic challenge in the Emergency Department

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Dear Editor,

Haemorrhagic brain metastases are a feared and relatively frequent complication of systemic malignancies. Brain metastases occur in up to 30% of adults with cancer, and intracranial haemorrhage (ICH) complicates approximately 20–50% of these lesions [1, 2]. The overall risk of haemorrhage in patients with intracranial neoplasms is estimated at 2.5%, with reported ranges from 1.4% to 10%, and the role of neurosurgical intervention in haemorrhagic metastases remains limited, with outcomes generally poor [3–6].

Patients frequently present to the Emergency Department (ED) because of the sudden and dramatic onset of symptoms. These lesions can mimic spontaneous ICH or an acute stroke-like syndrome. Typical presentations include seizures, focal neurological deficits, headache, vomiting, and features of raised intracranial pressure. Notably, in some cases, haemorrhagic brain metastasis may represent the first manifestation of an undiagnosed primary malignancy.

Several cancers have a strong association with haemorrhagic intracranial spread, including melanoma, renal cell carcinoma, choriocarcinoma, thyroid carcinoma, lung cancer, breast cancer, hepatocellular carcinoma, hepatoblastoma, and osteogenic sarcoma. Among these, lung cancer is the most common source, with up to half of patients developing brain metastases during the course of disease [7].

Initial assessment should follow standard resuscitation priorities (Airway, Breathing, Circulation, Disability, Exposure). A non-contrast computed

tomography scan of the brain remains the first-line investigation, allowing rapid confirmation of haemorrhage and guiding subsequent management.

Once ICH is confirmed, care should follow adapted principles from established haemorrhage protocols.

Neurological monitoring: Patients require regular neurological observations and should be positioned with the head of the bed elevated at 30°.

Blood pressure control: If systolic blood pressure exceeds 150 mmHg, reduction to a target of 120–140 mmHg within the first hour is recommended (particularly within six hours of onset). Intravenous labetalol is commonly used, with nicardipine or glyceryl trinitrate as second-line options. Monitoring should ensure heart rate remains > 50 bpm [8].

Reversal of anticoagulation: Prompt reversal of anticoagulation is critical to limit haemorrhagic expansion and neurological injury. Rapid correction within the first hour is essential, aiming for an INR < 1.3, which should be monitored every 3–6 hours. Management depends on the anticoagulant involved: vitamin K and four-factor prothrombin complex concentrate (PCC) for warfarin; idarucizumab for dabigatran, protamine for heparin and four-factor PCC or andexanet alfa for factor Xa inhibitors. Knowledge of the last dose and renal function is vital to estimate drug clearance. Importantly, treatment should not be delayed while awaiting laboratory results [9].

Coagulopathy assessment: Additional bleeding risks may arise from hepatic metastases, chemotherapy-induced marrow suppression, or thrombocytopenia and hypofibrinogenemia

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[10–12]. These should be promptly investigated and corrected when possible, with platelet transfusions, cryoprecipitate or plasma; current knowledge advises against empiric administration of platelet transfusions or PCC in patients with normal platelet counts [13].

In patients with haemorrhagic brain metastases, reversal strategies should be individualised, balancing haemostasis against thromboembolic risk, which is one of the few contraindications to anticoagulation reversal. Furthermore, frequent neurological assessment and repeat imaging, when indicated, are essential for guiding management.

Symptom control: Antiemetics (ondansetron or metoclopramide) and appropriate analgesia should be provided. Antiepileptic therapy, most often levetiracetam, is indicated for patients presenting with seizures.

Neuroimaging typically demonstrates multiple hyperdense lesions with surrounding vasogenic oedema. Contrast-enhanced imaging may reveal ring-enhancing lesions. In patients with significant neurological compromise, high-dose dexamethasone (up to 16 mg per day) is recommended to reduce mass effect and perilesional oedema [14].

Further management requires multidisciplinary input from emergency physicians, neurosurgeons, and oncologists. Decision-making becomes particularly complex when haemorrhagic metastasis represents the initial presentation of an undiagnosed primary malignancy. The overall prognosis is poor; ICH secondary to brain metastases carries a 30-day mortality rate of approximately 31% [15]. Early recognition, rapid stabilisation, and timely multidisciplinary involvement are therefore essential to optimise outcomes.

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