Remote Site Hemorrhage following Evacuation of Left Fronto-Temporo-Parietal Subdural Hematoma: A Rare Case Report and Comprehensive Literature Review

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Abstract

This report presents a compelling case of remote site hemorrhage (RSH), a rare but severe complication associated with neurosurgery. RSH involves cerebral bleeding away from the surgical site and was first documented in 1937 by Van Gehuchten. Despite its rarity, RSH remains challenging, affecting less than 1% of cases, with an unclear cause. The case involves a 67-year-old male who developed severe symptoms within 24 hours post-surgery. The initial computed tomography scan showed an acute subdural hematoma. Surgical evacuation was followed by rapid deterioration, leading to multiple RSH and brain stem infarctions. Unfortunately, the patient did not survive. RSH poses significant morbidity and mortality risks. Potential factors include volume loss, dural opening, and blood pressure fluctuations. Management ranges from conservative approaches to surgery, with poor prognosis post-RSH intervention. This case highlights the need for thorough preoperative assessment and careful intraoperative management. It emphasizes the complexities of neurosurgery and underscores the importance of ongoing research for managing rare complications like RSH, ultimately improving patient outcomes.

Keywords
- neurosurgery
- remote site hemorrhage
- RSH
- SDH

Dear Sir,

I am writing to report a compelling case of remote site hemorrhage (RSH), a rare but devastating complication associated with neurosurgical interventions. RSH is defined as a cerebral hemorrhage occurring at a location distant from the surgical intervention site. The first documented instance of RSH was reported by Van Gehuchten in 1937 when a patient experienced pontine hemorrhage following subtemporal decompression for a temporal lobe meningioma.[1]

In the field of neurosurgery, tackling RSH remains an enduring challenge, impacting 0.08 to 0.6% of cases, yet its underlying cause continues to evade a definitive explanation despite the existing body of literature.[2] Our report features a 67-year-old male who presented with severe headache, vomiting, and progressive right-sided weakness within 24 hours of surgery, without any history of trauma or pre-existing conditions like hypertension. Upon arrival at the emergency room, his Glasgow Coma Score (GCS) was 13 (E3V4M6).

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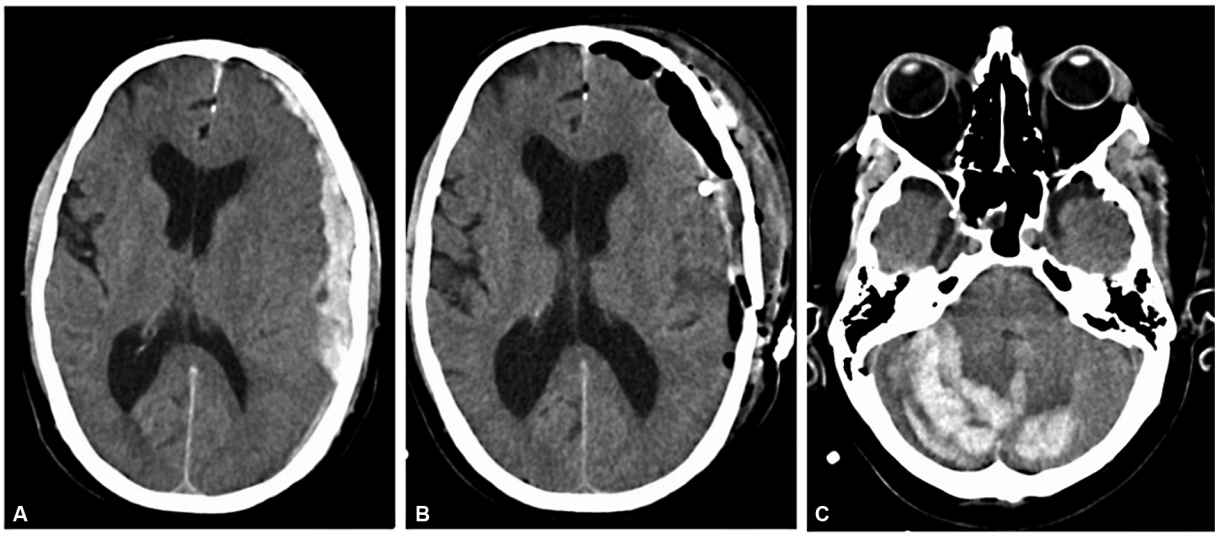

Fig. 1 (**A**) Axial computed tomography (CT) scan revealing an acute subdural hematoma on the left cerebral (fronto-temporo-parietal) convexity, and mild midline shift. Additionally, there is sulcal effacement in the left cerebral hemispheres and mild compression of the left lateral ventricle. (**B**) Postoperative CT scan demonstrating the successful evacuation of the acute subdural hematoma, with a drainage tube remaining in place within the left subdural collection. (**C**) Postoperative axial CT image, revealing acute hemorrhage in the cerebellum, predominantly on the right side suggestive of remote site hemorrhage. This hemorrhage is associated with effacement of basal cisterns, compression of the fourth ventricle, and obstructive hydrocephalus.

Comprehensive evaluations, including general, systemic, and laboratory investigations, revealed no abnormalities.

A plain computed tomography (CT) brain scan unveiled an acute left fronto-tempo-parietal subdural hematoma with mild midline shift (►**Fig. 1A**). Subsequently, the patient underwent a left-sided craniotomy for hematoma evacuation (►**Fig. 1B**). Intraoperatively, the brain appeared lax and shifted, with a gap between the brain and the dura, which was pulsating. The patient had high blood pressure before, during, and after surgery, necessitating labetalol infusion with frequent dose adjustments.

After the surgery, the patient's clinical condition deteriorated within 12 hours, characterized by irregular breathing and a GCS score of 4 (E1V1M2). Nevertheless, bilateral pupils remained equal (3mm) and reactive to light, while brain stem reflexes were still present (►**Fig. 1C**). His neuroclinical state further declined, with an absence of brain stem reflexes, and was continued on the best possible medical management. A subsequent CT scan performed after 20 hours revealed multiple RSH along with areas of hypodensities (indicating infarction) in the brain stem and cerebellum (►**Fig. 2A–D**). Regrettably, the patient succumbed to his condition within 24 hours following the initial surgery.

RSH is a rare but serious complication associated with substantial morbidity and mortality risks.[2,3] Existing literature suggests multiple potential contributing factors, such as abrupt volume loss, dural opening, intra- and

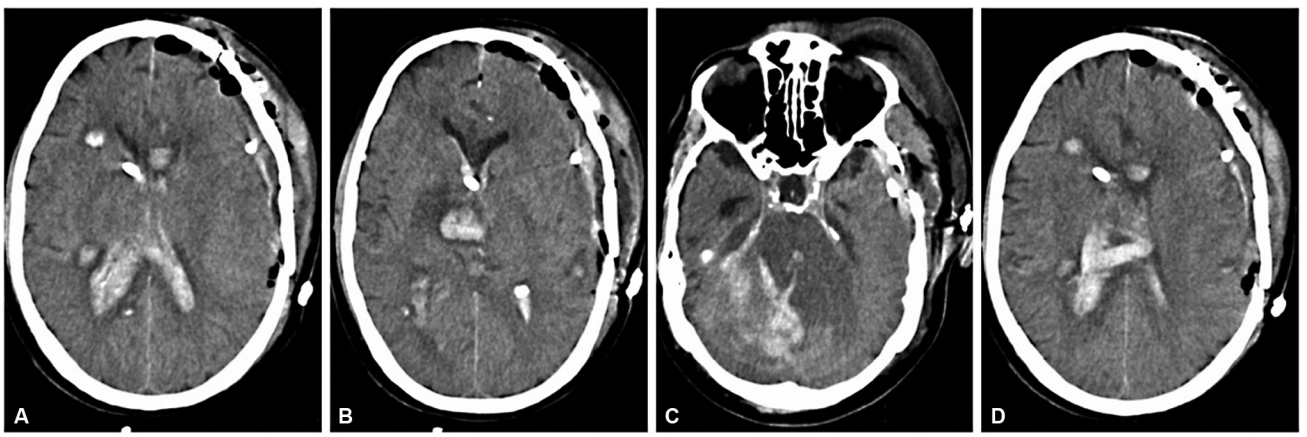

Fig. 2 (**A**) Axial computed tomography (CT) scan illustrating the presence of interventricular hemorrhage, alongside the presence of an external ventricular drain. (**B**) CT scan exhibiting the emergence of new intraparenchymal hemorrhage, observed in the right thalamic region and left parietal region, which is indicative of remote site hemorrhage (RSH). (**C**) CT scan revealing extensive hypodensities within the brain stem and cerebellum, suggestive of infarction, along with the presence of posterior fossa hemorrhage. (**D**) CT scan displaying an RSH in the corpus callosum and periventricular region, accompanied by surrounding edema.

postoperative cerebrospinal fluid (CSF) loss leading to cerebellar sag, variations in systolic blood pressure (SBP) or intracranial pressure (ICP) during surgery, patient positioning causing cerebral venous hypertension, preoperative aspirin use, coagulation abnormalities, intraoperative high blood pressure, and impaired venous drainage.[2–4] The management of RSH ranges from conservative treatments to surgery, contingent upon clinical conditions and radiological findings. Nevertheless, previous literature has demonstrated that post-RSH surgical intervention is associated with a poor prognosis.[2–4]

Our case had a normal coagulation profile and no history of antiplatelet or anticoagulant use before surgery. Blood pressure was effectively managed through the infusion of beta- and alpha-adrenergic receptor blockers. We hypothesize that the dural opening and sudden volume loss resulted in cerebral hypotension, leading to an elevation in venous or venular transluminal pressure and subsequent vascular tearing, contributing to the catastrophic event. Another potential factor could be intraoperative CSF loss resulting in cerebellar sag. Unfortunately, an magnetic resonance imaging could not be done on this critically ill patient to confirm the presence of multiple hemorrhagic venous infarcts. In this particular case, the patient's relatives were informed of the situation and, after a family discussion, opted not to pursue surgical intervention.

In conclusion, we recommend that patients undergoing neurosurgical procedures undergo a comprehensive preoperative laboratory assessment, including a coagulation profile, and take measures to minimize significant fluctuations in SBP and ICP, as well as prevent abrupt volume loss and excessive neck compression during preoperative positioning. This case serves as a reminder of the complexities inherent in neurosurgical practice and the need for ongoing research to better understand and manage rare complications such as RSH. We hope that by sharing this case, we can contribute to the collective knowledge of the medical community and ultimately improve patient outcomes in neurosurgical care.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient's relatives has given their consent for the patient's images and other clinical information to be reported in the journal. The patient's relatives understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Data Availability Statement

It can be made available as per the reader's request.

Conflict of Interest

None declared

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