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Case Report

Black hole sign migration in short-term brain CT scans: A possible link with clot evolution and histology ☆

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ABSTRACT

The black hole sign (BHS) is a rare radiological sign seen in the hyperacute phase of bleeding. It manifests within a hemorrhage in early hours, with limited studies exploring clot formation and evolution over a short duration. Despite various hypothesized mechanisms, the precise lifetime and dynamics of black hole sign development remain unclear. We describe the rare finding of a black hole sign within a deep brain hemorrhage, initially observed in the lateral portion of the clot during the first CT scan. Remarkably, in a subsequent CT scan, just 1 hour later, the BHS migrated towards the inner edge. Notably, while the hemorrhage size remained largely unchanged within this short timeframe, hyperacute bleeding led to increased perihematomal edema and sulci flattening. Histopathological features of the "evolving clot" are initially characterized by heightened cellularity. This increased cell density renders the hematoma less resistant to compressive forces, such as heightened endocranial pressure, offering a plausible explanation for the crushing and displacement of the BHS. Our study sheds light on the unique radiological progression of BHS within a deep brain ICH, emphasizing its association with dynamic clot formation and the consequential impact on surrounding structures.

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Abbreviations: CT, computer tomography; BHS, black hole sign; SS, swirl sign; ICH, intracerebral hemorrhage; ICP, intracranial pressure; BG, basal ganglia; HU, Hounsfield Unit; IM, imaging markers; RBC, red blood cells or erythrocytes.

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Introduction

Deep intracerebral hemorrhage (ICH) accounts for more than 50% of spontaneous cerebral hemorrhage. In about 60% it is caused by bleeding of arterial origin following hypertensive spikes [1]. Acute hematoma expansion (within 24 hours) can occur in up to 20% of patients, increasing the risks of death and severe disability [2]. Surgical treatment plays a controversial role, especially in large and deep ICH involving basal ganglia (volume >60 mL). On considering the elevated morbidity rates, surgical interventions are typically confined to addressing the drainage of subsequent hydrocephalus [3]. Recently, diverse neuroradiological signs have been delineated through various imaging modalities, as outlined by Huang and colleagues [4]. When dealing with ICH, computer tomography (CT) scan is typically the first imaging modality performed. This allows for the identification of various indicators of hyperacute bleeding [5]. Among these signs is the "black hole sign," characterized as a rounded hypointense area within the hyperintense clot, exhibiting a density approximately 28 Hounsfield units (HU) lower than the surrounding hemorrhage [6]. Being a hyperacute radiological indicator, the "black hole sign" present a brief and variable duration, indicating different bleeding moments. This phenomenon may be intricately linked to the dynamic histopathology of the clot, encompassing variations in cell and protein composition, as well as its response to neighboring forces [7]. This paper presents a case where the Black Hole Sign (BHS) migrated within a sizable deep ICH in just a 1-hour brain CT scan. Our investigation delved into potential mechanisms influencing the resizing and displacement of the BHS in the early stages, drawing from a thorough exploration of the literature and considering the chronohistogenesis of the clot.

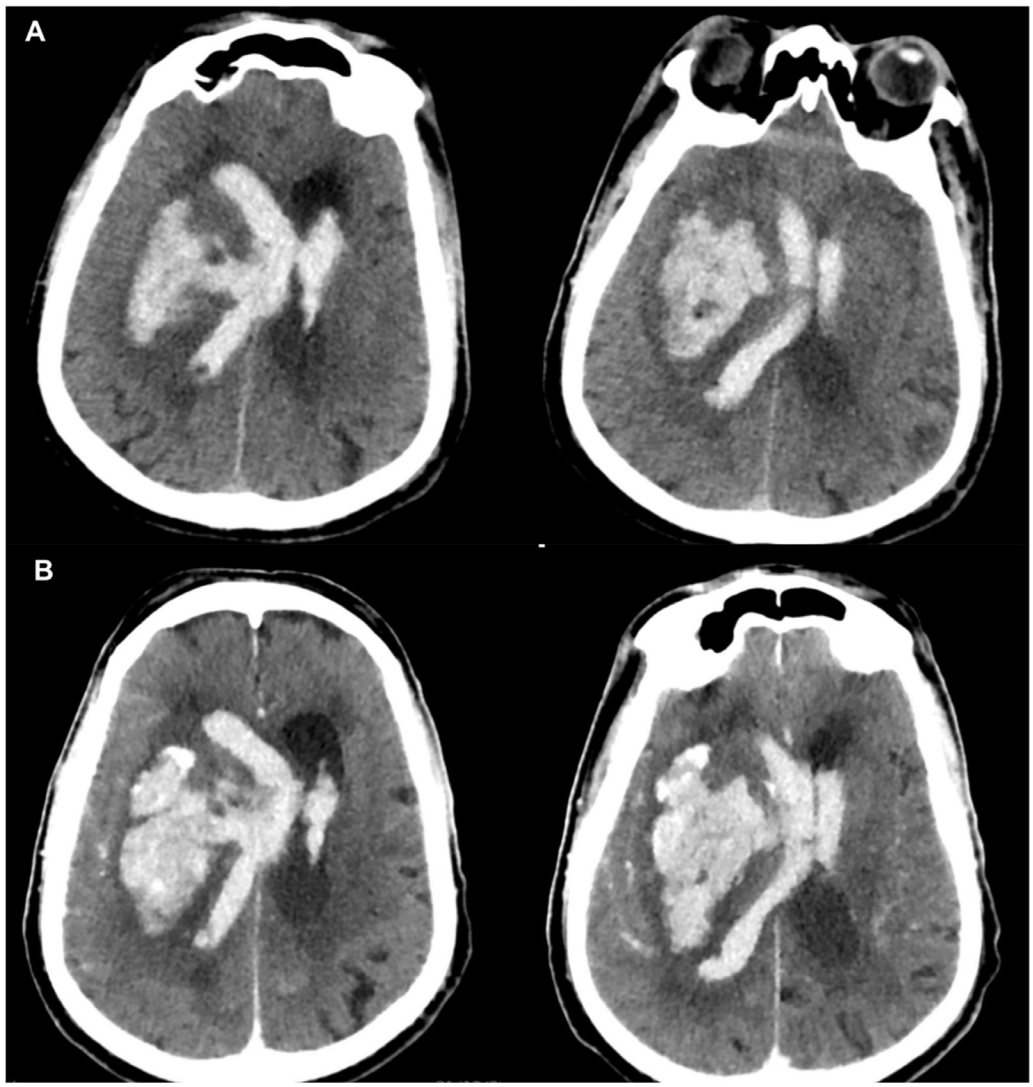

Fig. 1 – CT scans depict the motion of black hole signs, showcasing 2 distinct slices from the initial CT study (A) and 2 additional slices from the subsequent CT study (B), conducted 1 hour later. These images illustrate morphological alterations and the migration pattern of BHSs across various ICH sites.

Case presentation

A 74-years old male patient was admitted to our ER department due to the sudden onset of impaired consciousness state, left hemiparesis and slurred speech after a hypertensive spike. Notably, the patient had a history of hypertension but was not receiving pharmacological treatment. A brain CT with angiographic sequences done at the time of admission showed a massive right intra-parenchymal nucleocapsular hemorrhage (6.3 × 3.8 × 4.5 cm, 56 mL volume) with peri-lesional edema and 1.1 cm midline deviation. Angiosequences showed active bleeding with extra-vascular contrast blush into hemorrhagic volume. Due to the lesion location, the blood spreading into the semioval center and basal ganglia, it was not eligible for surgery. In the second CT re-evaluation, the hematoma measurements were documented at (6.6 × 3.9 × 4.5 cm), reflecting a volume of approximately 58 mL volume. Concurrently, there was a worsening of neurological symptoms with increased ventricular dilatation and midline shift. The BHS sign migrated to the medial compartment of the hematoma (Fig. 1).

Discussion

Spontaneous deep ICHs typically result from vascular lesions affecting the basal ganglia or capsular region, including ruptured Charcot-Bouchard microaneurysms (0.3-0.9 mm) or lenticulo-striate arteries dissection. These lesions often occur in anterior circulation terminal branches, particularly following hypertensive spikes [8]. The bleeding phases are categorized as hyperacute (<24 h), acute (24-72 h), subacute (72 h-13 days), and chronic (>14 days) [9]. Hematoma expansion risk peaks within the first 3 hours after neurological onset, observed in approximately 33% of cases [10]. During the acute phase, the clot appears hyperdense compared to normal brain tissue, following a hyperacute phase characterized by iso-hypodensity due to the challenge of ionizing radiations to capture blood turbulence [11]. CT scans, with their prompt availability and ability to display evolving lesion radiodensity patterns, serve as the primary diagnostic tool [5].

BHS presents as a hypointense area with well-defined margins within a hyperintense component, unrelated to the surrounding hematoma [12]. It is defined by a density 28 HU lower

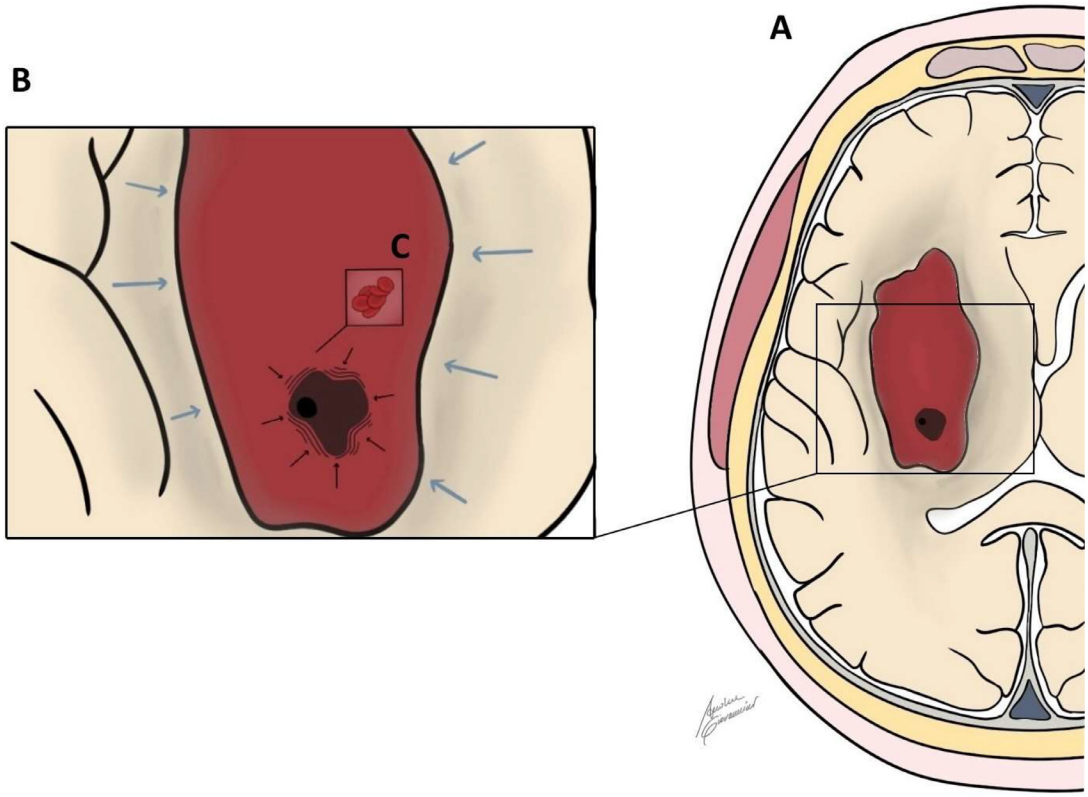

Fig. 2 – The illustration reveals pronounced peri-hematoma edema (A), exerting pressure on the hematoma walls and subsequently compressing the BHS (B). Notably, the hypercellularity of the clot during its initial formation (as indicated in the small box, C) enhances its compliance to ab-extrinsic stimuli. This composition facilitates the intralesional displacement of the hyperacute portion.

than the surrounding hemorrhage [6]. Although similar signs like the Swirl Sign (SS) is seen in rapidly expanding ICH, it is more typical of acute extradural hematomas, serving as a negative prognostic factor associated with an increased risk of death [13].

Xiong and colleagues [14] suggested that BHS is less sensitive but more specific in predicting cerebral hematoma expansion compared to SS itself (33.8% and 95.3% vs. 46, 71% and 71.3%). ICH outcomes are influenced by factors such as lesion volume, site, age, Glasgow Coma Scale (GCS), intraventricular clot and extension of perilesional edema [15,16]. In our report, while the size of ICH remained grossly unchanged in both controls, increased perilesional edema in the second CT scan should be considered. Indeed, a study by Appelboom and colleagues [17] found that peri-hematoma edema volume is a negative prognostic factor, even in patients with medium-sized hemorrhage.

Analyzing the histological composition of the hematoma might offer a plausible explanation for this radiological sign. During the initial stages of hematoma organization, deformation, edema, and necrosis of surrounding tissues occur [18]. Hence, it's conceivable that elevated intracranial pressure (ICP) caused flattening of sulci in the ipsilateral hemisphere, along with compression of the hematoma walls. Consequently, this alteration in clot plasticity may have led to changes in both the size and location of the BHS. Indeed, the early high radiodensity is associated with increased cellular density, including a higher concentration of red blood cells (RBCs) and martial stock within erythrocytes [19–21]. Furthermore, the clot observed in the early phases exhibits hyperdensity due to its composition featuring lower fibrin and higher erythrocyte content. This results in a reduced friction coefficient, making it more susceptible to compression by extrinsic forces [22] (Fig. 2).

Literature lacks consensus on the exact lifespan of BHS, with some evidence suggesting its persistence in 6-hour controls but not throughout the entire hyperacute period. This might be attributed to the absence of short-term distance CT scans (about 1 or 2 hours).

Changes in hematoma components and the migration of hyperacute signs, such as BHS or SS, can impact the radioheterogeneity of blood collection. Studies by Takeda [23] and Zhang [24] demonstrated that hematoma heterogeneity is linked to higher risk of rapid ICH expansion and, consequently, a worse outcome. However, interpreting this parameter can be challenging, making hyperacute neuroimaging markers crucial for diagnosis and prognosis.

Conclusion

BHS stands out as a valuable imaging marker in the assessment of ICH and its potential for rapid expansion. Since it is not always possible comparing 2 CT studies at close range, the precise timing and morphological modifications of this sign remain uncertain. However, it is established that the presence of this sign may not extend throughout the entire hyperacute phase. While the precise cause for its transformation within 1 hour is not fully elucidated, considering the histopathological modifiers of the clot in its early stages suggests a potential link to hematoma plasticity [25]. Indeed, reacting to external stimuli, such as heightened edema, a comprehensive centripetal force presses onto the clot walls, resulting in the crushing of BHS and influencing its evolution within the hematoma.

Patient consent

Written informed consent was obtained from the patient for use in this case report. No identifiable protected health information was utilized.

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