Korean J Neurotrauma. 2023 Sep;19(3):384-392
https://doi.org/10.13004/kjnt.2023.19.e40
pISSN 2234-8999·eISSN 2288-2243

KJNT

Case Report

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Spontaneous Acute Epidural Hematoma Associated With Metastatic Hepatocellular Carcinoma: A Case Report

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OPEN ACCESS

Received: May 31, 2023
Revised: Jul 31, 2023
Accepted: Aug 28, 2023
Published online: Sep 11, 2023

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Funding
This study was financially supported by Chonnam National University (grant number: BCRI23011).

ABSTRACT

Spontaneous acute epidural hematoma (AEDH) co-occurring with metastatic hepatocellular carcinoma (HCC) of the skull is rare, with only 7 documented cases in existing literature. This report describes the case of a 42-year-old man who presented with decreased consciousness following intermittent headaches following minor head trauma. Computed tomography imaging revealed an AEDH, prompting surgical intervention. Despite preliminary assumptions linking the causes of the trauma, surgical exploration revealed no evidence of traumatic injury. Instead, an infiltrative soft-tissue mass within the skull was identified. Histopathological examination confirmed that the mass was a metastatic HCC. Despite the successful hematoma evacuation, the patient's neurological status did not improve. This case underscores the importance of considering metastatic disease in the differential diagnosis of AEDH, particularly in patients with a history of malignant tumors, irrespective of prior indications of bone metastasis. Furthermore, it emphasizes the need to enhance diagnostic and therapeutic strategies for such complex cases.

Keywords: Head injuries; Cranial epidural hematoma; Hepatocellular cancer; Skull neoplasm

INTRODUCTION

Acute epidural hematoma (AEDH) typically develops between the skull and the dura mater due to a skull fracture, often instigating the rupture of the middle meningeal artery or venous system consequent to head trauma. Nontraumatic intracranial epidural hematoma (EDH), albeit rare, has been reported to originate from various etiologies such as infections,[1,7] coagulation abnormalities,[26,30] neoplasms,[2,12] and vascular malformations.[25] Hepatocellular carcinoma (HCC), a highly prevalent malignancy worldwide with higher incidence rates in Southeast Asia and sub-Saharan Africa primarily due to widespread hepatitis B and C,[9] predominantly metastasizes to the lungs, local lymph nodes, peritoneum, and adrenal glands. Bone metastases, including metastases to the vertebral body, sternum, ribs, and ilium, are observed in 2%–16% of the cases, while skull metastases present with a lesser frequency of 0.4%–1.6%.[13,15-17,28]

Conflict of Interest
The authors have no financial conflicts of interest.

Emerging literature suggests an escalating trend in extrahepatic metastasis, despite bone metastasis in HCC patients remaining a relatively uncommon occurrence, with reported incidences ranging from 2–25%. Further, the presence of bone metastasis has been associated with poor prognosis.[3,11]

Spontaneous AEDH concurrent with metastatic HCC of the skull is an rare phenomenon, with a mere 7 instances documented in existing literature.[15-17,28] This paper presents a case study of a patient diagnosed with spontaneous AEDH attributable to cranial metastasis of HCC, along with a discussion on its clinical features and pathogenesis through a literature review. This study was conducted in accordance with the tenets of the Declaration of Helsinki (Article 6 of the 2008 Amendment) and ensured complete anonymity of the subject.

CASE REPORT

A 42-year-old man with a known history of cirrhosis and HCC presented to our hospital's emergency department with complaints of diminishing consciousness. He reported a one-month history of recurrent headaches subsequent to a minor cranial trauma experienced a month prior. Despite the use of analgesics, the headache persisted without significant improvement. The patient's condition deteriorated from initial drowsiness to decreased consciousness, necessitating his transfer by a caregiver to a nearby local hospital. Upon admission, the patient's cognitive status further declined to a stuporous state. A computed tomography (CT) of the brain performed at the local hospital revealed a 134 mL EDH situated in the left parieto-occipital region, measured using the Kothari method, accompanied by a 13 mm midline shift.[18] The patient was subsequently referred to our hospital for surgical management.

During his initial presentation to the local hospital, the patient was in a stuporous state with significant cognitive decline, as determined by an initial CT scan. Approximately 2 hours later, he was transferred to the emergency room (ER) of our hospital. Preliminary reports from the local hospital attributed the patient's altered consciousness to the progression of an EDH following the head injury. Upon arrival to our ER, the patient's cognitive status was determined as comatose, with absence of pupillary reflexes and fixed dilation of 7 mm in both pupils. The physical examination revealed the presence of scalp swelling and bruising over an area suspected of a skull defect.

A follow-up CT scan demonstrated a progressive increase in midline shift, now measuring 17 mm, compared to the previous imaging. Moreover, the EDH volume was quantified at 153 mL, indicating hematoma enlargement (**FIGURE 1B**). Concurrently, a focal skull defect adjacent to the left occipital hematoma was identified (**FIGURE 1A**).

The patient, diagnosed with liver cirrhosis and HCC 3 years prior to admission, had undergone multiple rounds of Lipiodol-transcatheter arterial chemoembolization and radiofrequency ablation. A Technetium-99m whole-body bone scan (WBBS) conducted 4 months before the EDH onset showed no evidence of skeletal metastasis from the HCC (**FIGURE 2**). Upon admission, the patient presented with normal liver function parameters. However, his platelet count was lower than the standard range (78 K/uL; normal range 130–450 K/uL) and his prothrombin time exhibited a slight prolongation (14 seconds; normal range 10–13.6 seconds).

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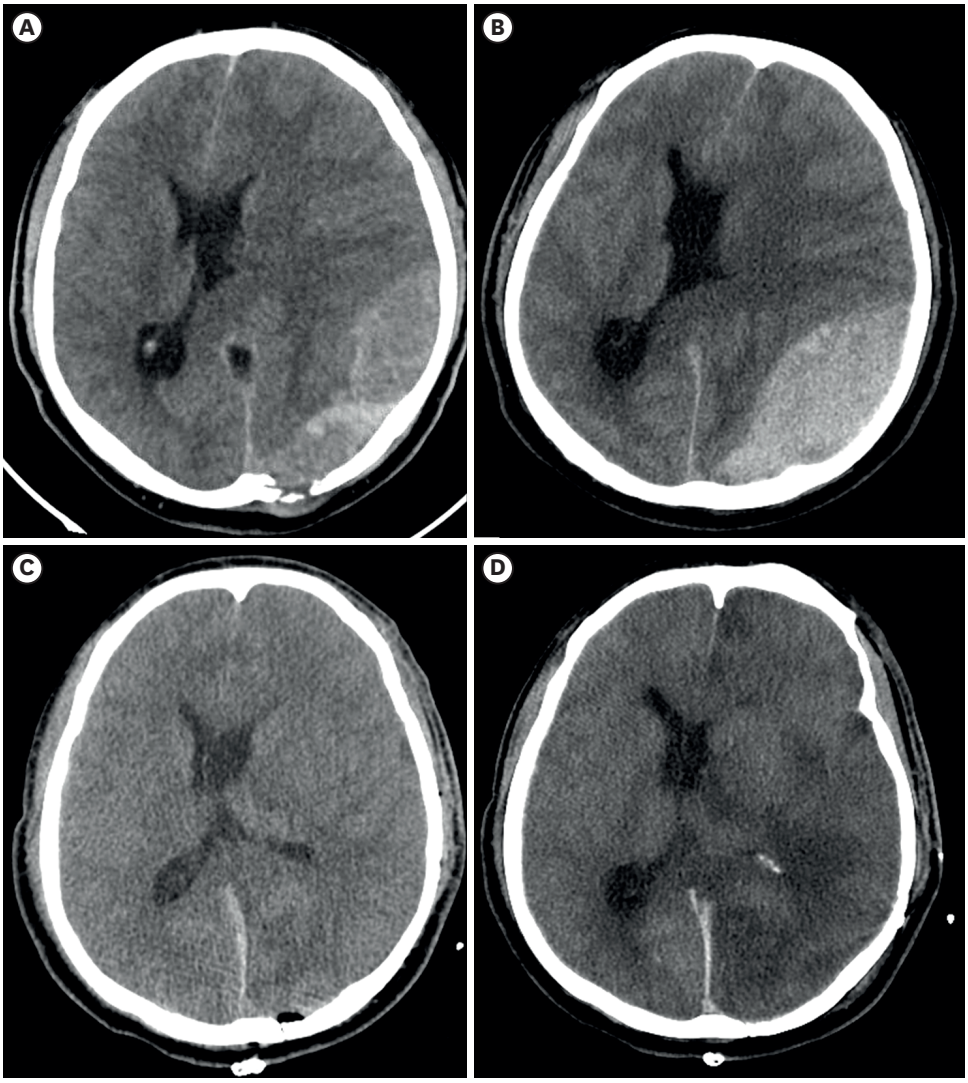

FIGURE 1. Serial brain CT scans.
(A) Initial brain CT reveals AEDH on the left parietal-occipital convex area with midline shifting and skull defect. (B) Increased AEDH upon arrival at our emergency department (C) Post-craniotomy brain CT follow-up shows successful excision of AEDH and midline shift improvement. (D) The third-day postoperative CT reveals worsened brain swelling, possible infarctions in the left hemisphere and right frontal lobe, transtentorial herniation, and a 15 mm rightward midline shift.
CT: computed tomography, AEDH: acute epidural hematoma.

Considering a recent history of trauma a month prior, an absence of skull metastasis in the WBBS performed 4 months earlier, coupled with the presence of a skull defect, scalp swelling, and adjacent bruising, the EDH was hypothesized to be trauma-induced with a high degree of certainty. Consequently, an emergency craniotomy was performed.

However, there were no indications of skull fracture or trauma during surgical exploration. A soft mass adhered to the dura and skull was discerned, demonstrating invasive features toward the skull which remained resistant to extensive saline irrigation (**FIGURE 3**). No other signs of arterial injury or sinus bleeding were observed, thus, the EDH was presumed to originate from this skull mass (**FIGURE 3**). Despite its proximity to the transverse sinus and superior sagittal sinus, no injuries were apparent in the sinus bleeding and diploic

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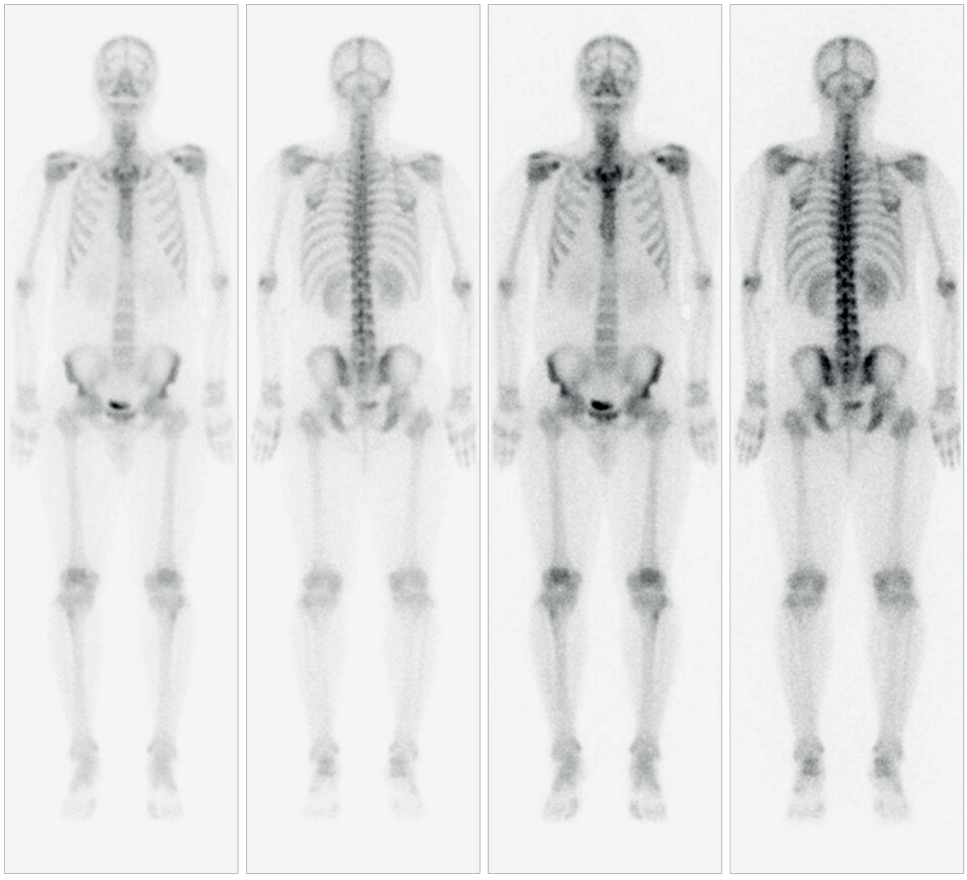

FIGURE 2. Technetium-99m WBBS.
WBBS performed for distant bone metastasis 4 months prior to the accident, showing no evidence of bone metastasis.
WBBS: whole-body bone scan.

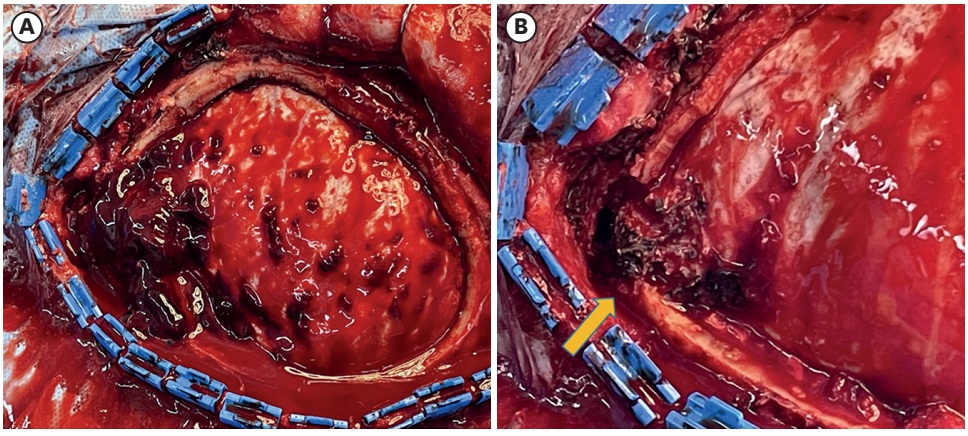

FIGURE 3. Intraoperative image.
(A) Epidural hematoma with no active bleeding focus (B) The osteolytic change of the skull and soft mass invading both the dura and the skull (indicated by the arrow).

vein. Due to the surgery being performed during the late-night hours, a frozen biopsy was unattainable; consequently, the soft mass and the osteolytic skull lesion were resected with a safety margin. Following evacuation of the EDH, a small incision was made in the dura mater to inspect for a subdural hematoma, which was not found, leading to the conclusion of

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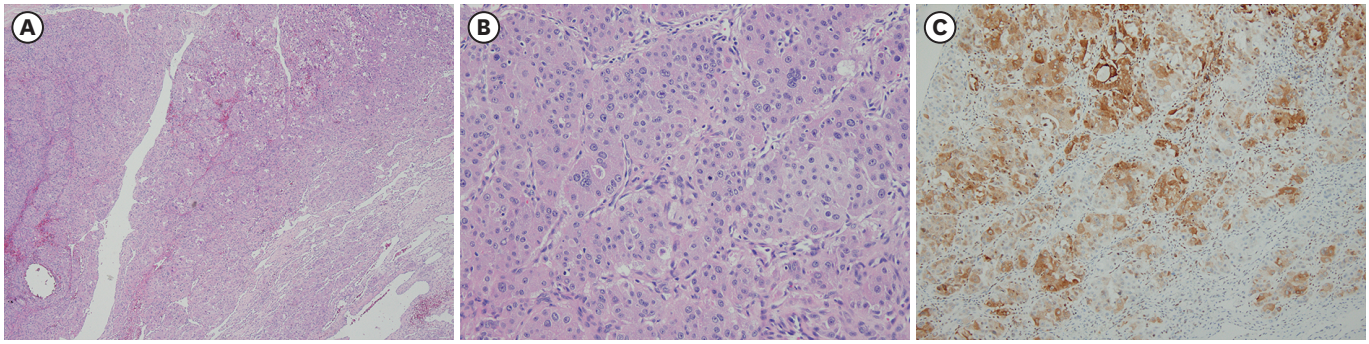

FIGURE 4. Histopathological examination of skull metastatic tumor.
(A) The lesion appears highly cellular and is composed of epithelial cells (H&E stain; original magnification, ×100). (B) The tumor cells exhibit abundant eosinophilic cytoplasm and a trabecular growth pattern consistent with metastatic hepatocellular carcinoma (H&E stain; original magnification, ×200). (C) The tumor cells are markedly positive for Hep Par 1 (immunohistochemistry; original magnification, ×400).
H&E: hematoxylin and eosin.

the surgical procedure. Subsequent to mass removal, the extracted specimen was dispatched for histopathological analysis. A postoperative CT scan was performed, confirming the successful elimination of the hematoma and a reduction in midline shift to 9 mm (**FIGURE 1C**). Nevertheless, the patient remained in a comatose state post-surgery, with both pupils dilated to 7 mm, exhibiting no light reflex. Clinical indications of cerebral dysfunction, specifically elevated serum osmolarity and sodium levels suggestive of an electrolyte imbalance, were observed persisting for 4 days postoperatively. Moreover, subsequent CT scans showed reduced shadows, indicative of an infarction (**FIGURE 1D**). On the fourth postoperative day, the patient's guardians chose to discontinue life-sustaining treatment, which subsequently led to the patient's demise.

Histopathological examination of the lesion demonstrated a hypercellular neoplasm. The neoplastic cells, characterized by copious eosinophilic cytoplasm, exhibited a tightly packed trabecular arrangement (**FIGURE 4A & B**). The application of immunohistochemical techniques, specifically utilizing monoclonal mouse anti-human Hep Par 1 antibody (clone OCH1E5, Cat. No. M7158, dilution 1:100; Dako, Glostrup, Denmark), elicited pronounced cytoplasmic expression of Hep Par 1 (**FIGURE 4C**). Consequently, the histopathological determination confirmed the lesion as metastatic HCC.

DISCUSSION

HCC is recognized for its propensity to metastasize to local lymph nodes and lungs and less frequently to the skeletal system. Given the dismal survival rate associated with HCC, the incidence of extrahepatic metastases has been estimated at approximately 15%–17%.[27] Skeletal metastases tend to occur in a hierarchical order involving the spine, pelvis, ribs, and skull.[24] A survey of existing studies reveals a survival duration of less than 6 months in untreated HCC patients.[4] Nevertheless, the mean survival time following HCC skull metastasis in treated patients extends to 8.9 months.[14] Clearly, the prognosis for patients with HCC skull metastasis remains grim, though early diagnosis and interventions such as local resection surgery, radiation therapy, and chemotherapy may potentially extend survival.[20]

Our review, inclusive of our case, of published cases of AEDH attributed to HCC metastasis reveals that 6 out of 8 individuals (75%) exhibited a postoperative modified Rankin Scale

https://doi.org/10.13004/kjnt.2023.19.e40

TABLE 1. Published cases of acute EDH due to skull metastasis of HCC

Author, year	Age (years)	Gender	Clinical manifestations	Coagulopathy	Osteolytic change	Adjacent venous sinuses	Postoperative outcomes	Postoperative mRS score
Nakagawa et al., 1992[22]	52	M	Headache, mental deepening	Yes	No	SSS	Death d/t liver failure	6
Hayashi et al., 2000[13]	70	M	Headache, palpable mass, left hemiparesis	Yes	No	SSS	Death d/t pneumonia	6
McIver et al., 2001[20]	50	M	Palpable mass, slurred speech, right hemiparesis	Unknown	Yes	SSS	Completely recovered	1
Kanai et al., 2008[15]	56	M	Headache, palpable mass, mental deepening	No	Yes	Torcular herophili	Death d/t liver failure	6
Kim et al., 2010[16]	53	M	Mental deepening	Yes	Yes	Cavernous	Death d/t multi-organ failure	6
Woo et al., 2010[28]	46	M	Headache, mental deepening	Yes	No	-	Vegetative state	5
Kim et al., 2016[17]	41	M	Headache, vomiting, mental deepening	No	Yes	Torcular herophili	Death d/t liver failure	6
This case	42	M	Headache, palpable mass, mental deepening	Yes	Yes	Torcular herophili	Death d/t cerebral dysfunction	6

EDH: epidural hematoma, HCC: hepatocellular carcinoma, SSS: superior sagittal sinus, d/t: due to, mRS: modified Rankin Scale.

score of 6, while one individual (12.5%) scored 5, and another one (12.5%) scored 1 (**TABLE 1**). Given that AEDH typically results from trauma, the emergence of a spontaneous epidural hematoma is an exceptional occurrence. AEDH triggered by metastatic tumors in the skull has been documented in patients diagnosed with malignancies including lung, ovarian, esophageal cancers, Ewing's sarcoma, and Langerhans cell histiocytosis.[5,8,19,29] Despite the frequent metastasis of malignant tumors to the skull in patients with lung, breast, and prostate cancer, skull metastasis in HCC patients is relatively infrequent.[6] Thus far, 7 instances of AEDH induced by HCC with skull metastases have been reported,[13,15-17,20,22,28] with our case marking the eighth. Six out of these 8 patients died following craniotomy, with mortality attributable to the primary disease. This underscores the necessity for preventive measures prior to an incident occurrence. The importance of early diagnosis of skull metastases is reiterated here, and timely treatment may prevent sudden mortality even in HCC patients with a poor prognosis.

In the context of HCC, the investigation for extrahepatic metastasis is typically initiated upon the observation of local recurrence following primary treatment. The most frequent sites of extrahepatic metastasis, in descending order, include the lungs, lymph nodes, bones, adrenal glands, and peritoneum. Routine diagnostic methods involve CT or magnetic resonance imaging of the chest, abdomen, and pelvis using contrast medium to detect extrahepatic metastasis, with the addition of bone scans in the presence of skeletal symptoms like bone pain. The most common clinical manifestations of HCC skull metastasis include headache, painful subcutaneous edema, neurological deficits, and seizures.[21] Upon reviewing 8 cases, inclusive of our patient, 6 reported headaches, and 4 presented with a palpable scalp mass (**TABLE 1**). Nevertheless, it is important to note that persistent headaches, unless accompanied by neurological symptoms such as hemiplegia, are often not recognized by patients or physicians as indicative of metastatic tumors. Typically, a patient with HCC reporting a headache might receive pain medication without further diagnostic evaluation. In the presented case, the patient had undergone a WBBS 4 months before the onset of AEDH, which revealed no bone metastatic lesions in the skull or elsewhere (**FIGURE 3**). The patient had experienced minor head trauma, colliding with a bed frame a month prior to the hospital visit, after which he reported localized scalp edema and a headache at the injury site. These symptoms were attributed to the head trauma by the patient and his caregiver, and no further imaging tests were pursued; the patient merely took medication

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for the headache. Recognizing a persistent headache as a potential symptom of HCC skull metastases could prompt additional diagnostic procedures, facilitating early detection and treatment, thereby potentially extending patient survival time. Although HCC skull metastases and the consequent spontaneous AEDH are rare, it is worth noting that WBBS is infrequently employed in clinical practice due to its high radiation exposure and likelihood of false-positive results, thus limiting its regular usage.[23] The most common radiological findings during skull metastasis of HCC are destructive or osteolytic lesions,[21] enabling differentiation from other types via a simple skull X-ray as opposed to more expensive tests such as bone scans.

Given that HCC is characterized by the presence of numerous sinusoidal blood vessels, coupled with their inherent fragility and the frequent manifestation of coagulopathies due to hepatic dysfunction in HCC patients, the propensity for tumoral bleeding is substantially increased. The rapid and aggressive proliferation of metastatic HCC within the skull can induce osteolysis, subsequently disrupting the vascular architecture within the peritumoral tissue.[17] The majority of AEDH instances are attributed to arterial bleeding (85%), predominantly within the dura mater. Conversely, hematomas of the vertex, anterior middle fossa, and posterior fossa can originate from injuries to the dural sinus, arachnoid granulations, and diploic emissary veins.[10] In the 8 cases reviewed, 7 exhibited metastatic lesions proximate to a large venous sinus, with 5 of these cases presenting osteolytic lesions (**TABLE 1**). In the presented case, an osteolytic lesion was located in the area of the torcular herophili, the confluence of the superior sagittal sinus and the transverse sinus; the surgical findings revealed no active arterial bleeding focus. Rather, venous hemorrhaging within the metastatic tumor and its surrounding regions was confirmed as the etiological factor of the AEDH.

CONCLUSION

Spontaneous occurrences of AEDH precipitated by metastatic skull lesions in HCC patients are uncommon, often leading to the absence of routine diagnostic procedures. The inability of the patient to recuperate and the ensuing mortality can be ascribed to 2 primary determinants: the inherent pathology and the postponement of surgical intervention. Consequently, preemptive diagnosis prior to the manifestation of hemorrhage assumes paramount importance. Swift observation and intervention, rather than awaiting the advancement of symptoms such as headaches, trauma, or other novel presentations, is vital for timely therapeutic management. Therefore, accurate identification of tumoral bleeding via CT scans, followed by the swift initiation of appropriate treatment, remains a necessity.

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