Spontaneous Intracranial Epidural Hematoma Originating from Dural Metastasis of Hepatocellular Carcinoma

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Spontaneous intracranial epidural hematoma (EDH) due to dural metastasis of hepatocellular carcinoma is very rare. A 53-year-old male patient with hepatocellular carcinoma, who was admitted to the department of oncology, was referred to department of neurosurgery because of sudden mental deterioration to semicoma with papillary anisocoria and decerebrate rigidity after transarterial chemoembolization for hepatoma. Brain computed tomography (CT) revealed large amount of acute EDH with severe midline shifting. An emergent craniotomy and evacuation of EDH was performed. Active bleeding from middle cranial fossa floor was identified. There showed osteolytic change on the middle fossa floor with friable mass-like lesion spreading on the overlying dura suggesting metastasis. Pathological examination revealed anaplastic cells with sinusoidal arrangement which probably led to spontaneous hemorrhage and formation of EDH. As a rare cause of spontaneous EDH, dural metastasis from malignancy should be considered.

KEY WORDS: Spontaneous epidural hematoma • Hepatocellular carcinoma • Dural metastasis.

INTRODUCTION

Intracranial epidural hematoma (EDH) is usually a consequence of head injury. But, spontaneous intracranial EDHs can occur in patients with paracranial infections, bleeding or coagulation disorders, vascular malformation, and neoplastic condition. Spontaneous intracranial EDH originating from a metastatic hepatocellular carcinoma is very rare. To the authors’ knowledge, only 6 cases have been reported in the literature. We describe a case of spontaneous intracranial acute EDH originating from a metastatic hepatocellular carcinoma of the skull and dura mater in the middle cranial fossa.

CASE REPORT

A 53-year-old male patient with hepatocellular carcinoma, who was admitted to the department of oncology, was referred to department of neurosurgery because of sudden mental deterioration to semicoma with papillary anisocoria and decerebrate rigidity after transarterial chemoembolization for hepatoma. Brain computed tomography (CT) revealed large amount of acute EDH with severe midline shifting (Fig. 1). He had no history of trauma and coagulopathy even though he had slightly prolonged prothrombin time (INR 1.36). An emergent craniotomy and evacuation of EDH was performed. There was no evidence of trauma on the scalp, subgaleal

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Fig. 1. Brain CT scans showing a huge epidural hematoma from the right temporal base to the cerebral convexity with severe mass effect.
space and skull bone. Thick hematoma was seen on the epidural space, mostly on the temporal base displacing the brain. There showed an osteolytic change on the middle cranial fossa floor (Fig. 2). Active bleeding from the middle meningeal artery and osteolytic bone on the middle cranial fossa floor was controlled with bipolar cautery and application of bone wax. Curettage of friable mass-like lesions on the overlying dura and skull was performed, which suggested metastasis to the dura and skull. Pathological examination revealed anaplastic cells with sinusoidal arrangement which probably led to spontaneous hemorrhage and formation of EDH (Fig. 3). Despite of the rapid evacuation of EDH, he is in vegetative state after surgery.

**DISCUSSION**

EDH is usually a consequence of head injury. In-bending of skull at the time of traumatic impact on the head makes the skull fracture, which can cause epidural bleeding from lacerated meningeal vessels, fractured edges, torn venous sinuses or diploic veins. But, spontaneous or non-traumatic intracranial EDHs are very rare. Main possible causes of this unusual phenomenon are infection from frontal sinusitis, maxillary sinusitis, otitis media and craniofacial infections, and coagulation disorders, vascular malformation of dura, and malignancy. Intracranial hemorrhage from distant metastasis of primary cancer is rare and its incidence accounts for 0.9-11% (28). The location is usually intratumoral or intracerebral, and very rarely epidural. Spontaneous EDHs resulting from dural metastasis of primary malignancy have been reported only 16 cases (29), which include lung cancer (20,22,24), hepatocellular carcinoma (5,10,12,15-17), ovarian cancer (9), eosinophilic granuloma (4,14), Ewing’s sarcoma (2,26), malignant fibrous histiocytoma (13), and Langerhans’ cell histiocytosis (3). Spontaneous EDH due to dural metastasis of hepatocellular carcinoma is very rare and to the authors’ knowledge, it has been reported only 6 cases in the literature (5,10,12,15-17). It has been well known that hepatocellular carcinoma has a characteristic of high risk of bleeding tendency due to hepatic failure and formation of neoangiogenesis (6). It usually spreads to regional lymph nodes around the porta hepatitis via lymphatics, but it can also spread hematogenously via intrahepatic vein, portal vein, and hepatic artery. It commonly metastasizes to the lung, regional lymph nodes, peritoneum, and adrenal glands, but, rarely spread to the brain. Secondary intracranial hepatic carcinomas were 1.3-2.9% among intracranial metastatic tumors (29). In most cases of spontaneous EDH of hepatocellular carcinoma, bleeding

**Fig. 2.** Brain CT scans showing osteolytic bone lesion on the middle cranial fossa floor. Axial (A) and sagittal (B) views of bone window level show eroded right temporal base (open arrows). Bony defect (black arrow) is well visualized on CT window image (C).

**Fig. 3.** Photomicrographs obtained from the thickened dura matter showing metastatic hepatocellular carcinoma. A: Sinusoidal pattern of anaplastic cells is demonstrated in dural layer (H & E, X100). B: Sinusoidal blood vessels (H & E, X400). C: Hepatocyte antibody immunostain showing metastatic poorly differentiated hepatocyte with mitosis.
Table 1. Summary of patients in the literature who were diagnosed as spontaneous epidural hematoma from hepatocellular carcinoma metastasis

| Authors, Year | Age/Sex | Clinical features | Preop Dx of HCC | Surgical outcomes* |
|---------------|---------|------------------|-----------------|--------------------|
| Nakao, 1992   | 58/M    | Scalp and bone mass | Yes             | Discharged without ND |
| Nakagawa, 1992| 52/M    | Semicoma          | No              | 1st operation due to recurrent bone mass |
| Lee, 1996     | 65/F    | Semicoma, ascites, liver mass | No          | 1 month later, died of liver failure |
| Endo, 1999    | 44/F    | Hemiparesis, coma one week later, combined SDH | Yes | Died of liver failure |
| Hayashi, 2000  | 70/M    | Headache, hemiparesis | Yes | 2 month later, died of liver failure and pneumonia |
| McIver, 2001  | 50/M    | Headache, hemiparesis | No | No neurologic deficit. Underwent palliative C Tx |
| This study    | 53/M    | Semicoma          | Yes | Persistent vegetative state |

*All patients underwent surgery. CTx: chemotherapy, Dx: diagnosis, F: female, HCC: hepatocellular carcinoma, M: male, ND: neurologic deficit, Preop: preoperative, SDH: subdural hematoma

arise from middle meningeal artery, emissary vein, and venous sinus with regional bone invasion. Histological feature shows many sinusoid-like blood vessels. In our case, the bleeding had arisen from the eroded middle meningeal artery and osteolytic bone. Spontaneous EDHs of hepatocellular carcinoma may be spread via hematogenous route, but it is uncertain. Although dura acts as a barrier of tumor invasion, hepatocellular carcinoma has a high potency of dural invasion and destruction of neighboring bone.

To elucidate the cause of spontaneous EDH, it is necessary to examine the parasanal sinus and ear to rule out infection, chest X-ray for lung mass, abdominal sonography or CT scan for hepatocellular carcinoma, laboratory finding for coagulopathy and image studies for vascular abnormality8). In particular, the patient with hepatocellular carcinoma and lung metastasis should be evaluated for brain metastasis because intracranial metastasis of hepatocellular carcinoma often combined lung metastasis9,10,11. In addition, biopsy of dura mater and skull are needed during the operation.

Clinical outcomes of spontaneous EDH with hepatocellular carcinoma were usually poor (Table 1). All of these patients (reported 6 and ours 1) underwent surgery. Five were dead because of liver failure, ascites, coagulopathy and pneumonia, one became vegetative state, and one survived. These poor outcomes might be caused by its own poor prognosis of terminal stage hepatocellular carcinoma, combined coagulopathy and liver failure, and high mortality rate of EDH.

CONCLUSION

The authors report a rare case of spontaneous intracranial EDH originating from a metastatic hepatocellular carcinoma of the skull and dura mater on the middle cranial fossa. In patients who have spontaneous EDH, metastatic hepatocellular carcinoma should be included in the differential diagnosis as a rare cause of spontaneous EDH.

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