

Cerebral Metastasis Mimicking A Subdural Hematoma in Cervical Carcinoma: A Case Report

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ABSTRACT

Objective:

To describe an unusual intracranial metastasis from cervical carcinoma that radiologically mimicked a subdural hematoma and to emphasize the diagnostic challenges associated with this presentation.

Case description:

A 58-year-old woman with previously treated poorly differentiated cervical carcinoma presented with progressive decline in consciousness, dysarthria, headache, and right-sided weakness. Contrast-enhanced computed tomography of the head demonstrated subdural-appearing lesions in the left parieto-occipital and right frontotemporal regions, accompanied by perilesional edema and an approximately eight-millimeter rightward midline shift. A lytic lesion of the left parieto-occipital calvarium and cortical thinning of the right temporal bone raised suspicion of metastatic involvement. Because of neurological deterioration and significant mass effect, urgent craniotomy with tumor excision was performed. Histopathological examination demonstrated metastatic carcinoma, and subsequent morphological and immunohistochemical evaluation supported a diagnosis of small-cell neuroendocrine carcinoma of cervical origin.

Comments:

Intracranial metastasis should be considered in the differential diagnosis of a subdural-appearing lesion in patients with a history of malignancy, particularly when perilesional edema, bone abnormalities, or significant mass effect are present. Contrast-enhanced computed tomography is valuable for rapid emergency assessment, whereas magnetic resonance imaging may provide superior lesion characterization in clinically stable patients. Histopathological examination remains essential for establishing the definitive diagnosis.

Keywords: Uterine Cervical Neoplasms; Neoplasm Metastasis; Hematoma, Subdural; Carcinoma, Neuroendocrine.

INTRODUCTION

Intracranial metastases from cervical carcinoma are uncommon. Brain metastases most frequently originate from breast cancer, lung cancer, and malignant melanoma, which together account for more than 75% of all reported cases of brain metastasis.¹ Dural metastases have been identified at autopsy in approximately 8–9% of patients who died from or with metastatic malignancies; however, only 198 cases of dural metastasis were described in the medical literature between 1904 and 2003.² Over the past half-century, approximately 700 cases of brain metastasis from cervical cancer and 1,100 cases from endometrial cancer have been reported.¹ The risk of brain metastasis may be increased in tumors with poorly differentiated histology. Intracranial lesions are frequently located in the supratentorial compartment, potentially reflecting the vascular supply and anatomical characteristics of this region. Once intracranial metastasis occurs, the prognosis is generally poor, with a reported median survival of approximately 2–8 months.³

Imaging plays a pivotal role in the evaluation of cervical carcinoma, including assessment of disease extent, detection of recurrence, treatment planning, and management of advanced-stage disease. Brain metastases from cervical carcinoma are most commonly located in the supratentorial region and may present as either solitary (50.6%) or multiple (49.4%) lesions. On magnetic resonance imaging (MRI), these metastases typically appear as solid lesions with variable degrees of contrast enhancement and surrounding perilesional edema.⁴ Leptomeningeal involvement has also been reported, although it remains uncommon.⁵ Although subdural hematoma (SDH) is generally associated with trauma or coagulation abnormalities, metastatic intracranial disease may occasionally produce radiological findings that resemble an SDH, potentially complicating the initial diagnosis. Here, we report an unusual case of a 58-year-old woman with cervical carcinoma who developed intracranial metastasis that radiologically mimicked an SDH on contrast-enhanced computed tomography (CT) of the head.

CASE PRESENTATION

A 58-year-old woman had been diagnosed with cervical carcinoma in March 2023. She subsequently received four cycles of cisplatin-based chemotherapy between May and September 2023 and one session of radiotherapy. Histopathological and immunohistochemical examinations performed in March 2023 demonstrated poorly differentiated adenocarcinoma of cervical origin with negative p16 expression. There was no history of head trauma.

In February 2024, the patient was brought to the hospital because of a gradually progressive decline in consciousness, incoherent speech, and difficulty communicating. She had experienced a progressively worsening headache for approximately one month. One day before admission, she developed weakness of the right side of the body. Neurological examination revealed right-sided motor weakness and hypoglossal nerve (cranial nerve XII) palsy manifested by lingual dysarthria.

Complete blood count revealed a hemoglobin level of 9.9, a leukocyte count of 9,300, and a platelet count of 252,000. Liver function tests showed AST (SGOT) of 29, ALT (SGPT) of 22, and albumin of 3.06, with no evidence of significant hepatic dysfunction. Renal function was also preserved, with a blood urea nitrogen level of 10 and serum creatinine of 0.9. The coagulation profile was within normal limits, with a prothrombin time of 12.0 and an activated partial thromboplastin time of 21.8.

Considering the newly developed neurological deficits and the patient's history of malignancy, contrast-enhanced CT of the head was performed to evaluate the underlying intracranial pathology. The CT images demonstrated subdural-appearing lesions in the left parieto-occipital and right frontotemporal regions, accompanied by surrounding perifocal edema. The lesions produced substantial mass effect with an approximately 0.8-cm midline shift to the right. A lytic lesion involving the left parieto-occipital calvarium and cortical thinning of the right temporal bone were also identified and considered suspicious for metastatic involvement.

Given the presence of an intracranial space-occupying process associated with neurological deterioration and midline shift, the patient underwent urgent craniotomy with tumor excision. Histopathological examination of the intracranial specimen demonstrated metastatic carcinoma, with the differential diagnosis including poorly differentiated adenocarcinoma and neuroendocrine carcinoma.

Further immunohistochemical examination showed the following findings: chromogranin, negative in the tumor cell cytoplasm; CD56, positive in the tumor cell membrane and cytoplasm; vimentin, negative in the tumor cell cytoplasm; synaptophysin, negative in the tumor cell cytoplasm; p40, negative in the tumor cell nuclei; p16, negative in the tumor cell nuclei; estrogen receptor (ER), negative in the tumor cell nuclei; and carcinoembryonic antigen (CEA), positive in the tumor cell cytoplasm. Following pathological review, the morphological and immunohistochemical findings were considered consistent with small-cell neuroendocrine carcinoma of cervical origin with intracranial metastasis.

Following surgery, the patient was planned for palliative management consisting of supportive care, palliative chemotherapy, and radiotherapy. Additional surgical intervention would be reserved for emergency indications if clinically required.

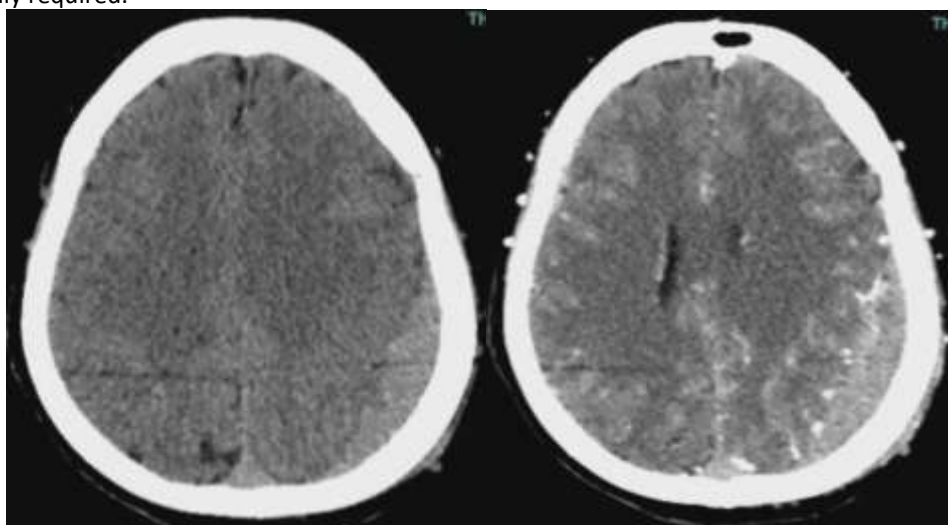


Figure 1. Axial CT images of the head demonstrating the left parieto-occipital subdural-appearing lesion. Non-contrast CT image (left) and contrast-enhanced CT image (right).

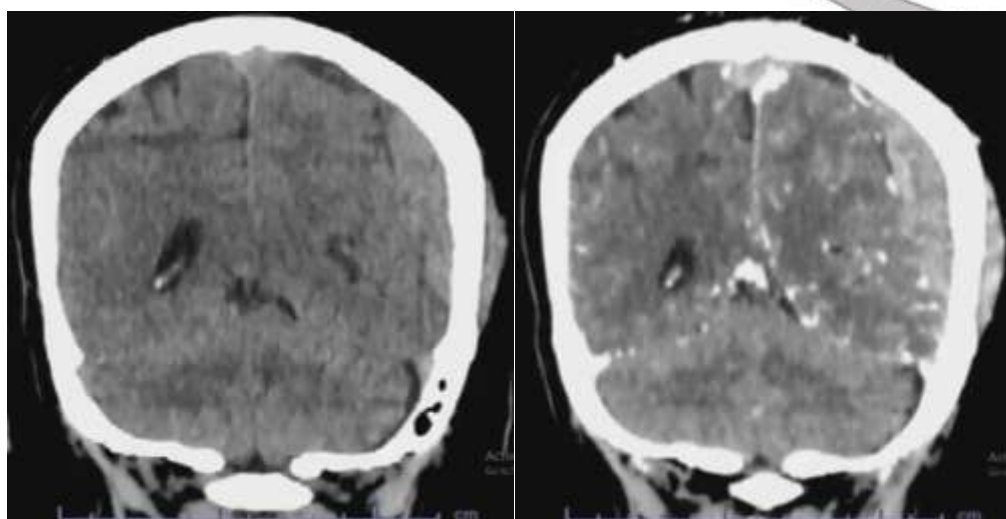


Figure 2. Coronal CT images of the head demonstrating the subdural-appearing intracranial lesion. Non-contrast CT image (left) and contrast-enhanced CT image (right).

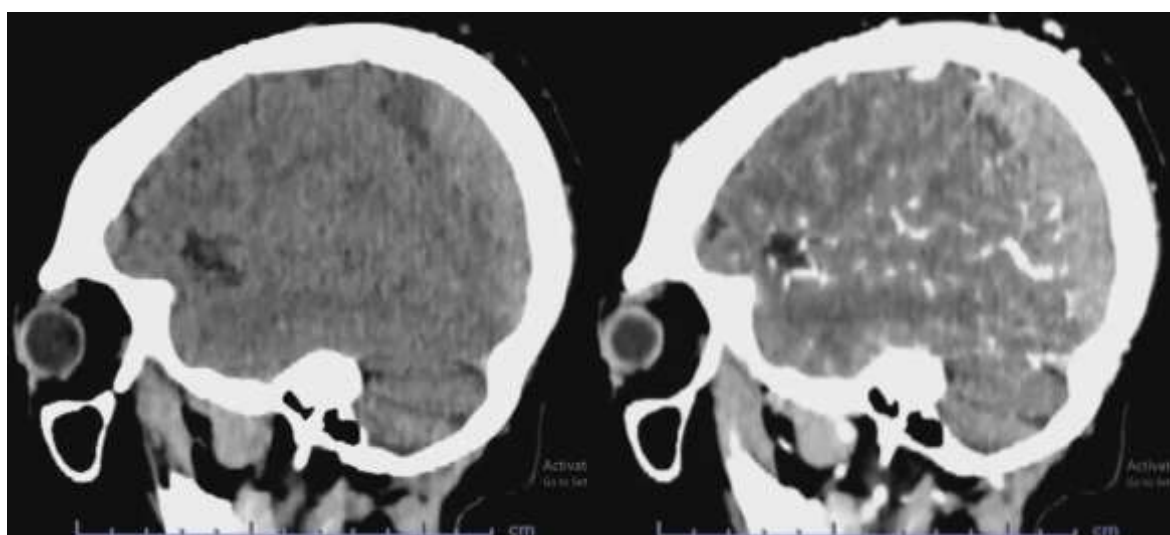


Figure 3. Sagittal CT images of the head demonstrating the subdural-appearing intracranial lesion. Non-contrast CT image (left) and contrast-enhanced CT image (right).

DISCUSSION

Hemorrhagic complications are an important concern in patients with advanced malignancy and may arise through several mechanisms. Potential causes include direct tumor-related effects, such as arterial or venous invasion, cerebral venous sinus thrombosis, leptomeningeal involvement, vascular compression, intratumoral hemorrhage, and intravascular tumor infiltration. Other contributing factors include coagulopathy, such as disseminated intravascular coagulation, thrombocytopenia, hypercoagulable states, and abnormalities of the hemostatic cascade; myelosuppressive effects of chemotherapy; infection; intracranial radiotherapy; and invasive procedures, including lumbar puncture, intrathecal chemotherapy, and surgery.⁶

In the present case, there was no history of head trauma, and hematological evaluation showed a normal platelet count and coagulation profile. Liver and renal function were also preserved. Although mild anemia was present, there were no clinical signs of systemic bleeding, such as hemoptysis, epistaxis, petechiae, purpura, hematemesis, or melena. The patient had not previously received intracranial radiotherapy, and her prior surgical procedure had been limited to cervical biopsy. These findings made trauma, clinically significant coagulopathy, and procedure-related intracranial hemorrhage less likely as the primary explanation for the subdural-appearing lesion. In this clinical context, direct involvement of the intracranial structures by an aggressive metastatic tumor was therefore considered a plausible underlying mechanism.

Histopathological examination of the intracranial specimen demonstrated metastatic carcinoma, and subsequent morphological and immunohistochemical review was considered consistent with small-cell neuroendocrine carcinoma of cervical origin. Brain metastases from cervical carcinoma have been reported more frequently in tumors with poorly differentiated histology, while non-squamous histological subtypes account for a substantial proportion of reported intracranial metastases.^{7,8} Small-cell neuroendocrine carcinoma of the cervix is an uncommon but highly aggressive malignancy characterized by an increased propensity for early distant dissemination and an unfavorable prognosis.⁹ The aggressive histopathological characteristics identified in this patient may therefore have contributed to the development of intracranial metastatic disease. Several molecular processes have been proposed to facilitate tumor invasion and distant metastasis. Expression of variant isoforms of the cell adhesion molecule CD44 (CD44v) has been associated with an enhanced metastatic phenotype in several malignancies.¹⁰ CD44 may also interact with matrix metalloproteinases (MMPs), including MMP-9, promoting extracellular matrix degradation and facilitating tumor invasion.¹¹ In addition, epithelial-mesenchymal transition (EMT) enables malignant epithelial cells to lose cell-to-cell adhesion and cellular polarity while acquiring increased motility and invasive capacity. MMP-mediated cleavage of E-cadherin may further facilitate tumor-cell migration and invasion.^{11,12} These mechanisms provide a biological framework for the aggressive metastatic behavior of poorly differentiated tumors; however, they were not directly evaluated in the present patient and should therefore be regarded as possible rather than proven mechanisms in this case. Tumor angiogenesis may further contribute to the risk of hemorrhage. Unlike normal vascular networks, tumor-associated vessels are frequently immature, tortuous, disorganized, and hyperpermeable because of an imbalance between proangiogenic and antiangiogenic signaling.¹³ These abnormal vessels may have incomplete endothelial lining, deficient smooth-muscle support, and abnormal basement membranes. Increased permeability, impaired perfusion, and regional hypoxia may further compromise vascular integrity. Consequently, tumor-associated vessels can be susceptible to thrombosis, aneurysmal change, and rupture, providing another potential mechanism for hemorrhage associated with intracranial metastatic disease.^{11,13} Cervical carcinoma may reach the brain through hematogenous dissemination. Tumor cells may enter the systemic circulation through the inferior vena cava and pulmonary circulation before reaching the left heart, aorta, and cerebral circulation. An alternative pathway may involve dissemination from the pelvic veins through the paravertebral venous plexus and subsequently into the intracranial venous system.¹ In patients with malignancy, SDH is more commonly associated with trauma or coagulation disorders; however, dural metastatic involvement may occasionally be accompanied by an SDH-like appearance. Proposed mechanisms include rupture of vessels within metastatic tissue, erosion of adjacent vessels by tumor infiltration, and rupture of inner dural vessels secondary to obstruction or congestion of the outer dural vasculature.⁶ Previous reports indicate that SDH occurs in only a subset of patients with dural metastases, estimated at approximately 15–40%.⁶ Distinguishing a true SDH from dural or other intracranial metastatic disease may be challenging on CT imaging alone. Dural metastases may appear hyperdense, isodense, or heterogeneous on CT, and subtle nodularity may be difficult to recognize, particularly when the lesion is predominantly isodense.¹⁴ In the present case, the initial contrast-enhanced CT demonstrated subdural-appearing lesions with marked perifocal edema and an approximately 8-mm rightward midline shift. Importantly, a lytic lesion of the left parieto-occipital calvarium and cortical thinning of the right temporal bone were also observed. In a patient with a known malignancy, these osseous abnormalities increased the suspicion of metastatic involvement rather than an uncomplicated SDH.¹⁴ Contrast-enhanced MRI generally provides superior soft-tissue characterization and can more clearly differentiate hematoma from dural or intracranial tumor.¹⁴ Nevertheless, imaging decisions must be considered within the clinical context. In this patient, progressive neurological deterioration, focal neurological deficits, substantial mass effect, and an approximately 8-mm midline shift indicated an intracranial space-occupying process requiring urgent intervention. Therefore, surgical decompression and tumor excision were prioritized rather than delaying treatment to obtain additional MRI characterization. Histopathological examination subsequently established the metastatic nature of the lesion. This case therefore illustrates that an apparent SDH in a patient with a known malignancy, particularly when accompanied by disproportionate edema, bone destruction, or other atypical imaging features, should prompt consideration of metastatic disease.

CONCLUSION

Intracranial metastasis should be included in the differential diagnosis of a subdural-appearing lesion in patients with a history of malignancy, particularly when new focal neurological deficits, progressive deterioration of consciousness, marked perifocal edema, bone abnormalities, or significant mass effect are present. Contrast-enhanced CT is valuable for rapid assessment of intracranial lesions, hemorrhage, mass effect, and midline shift in emergency settings. Although contrast-enhanced MRI provides superior characterization and may help

distinguish hematoma from metastatic disease, additional MRI should not delay urgent surgical management when clinical and CT findings indicate neurological deterioration and significant intracranial mass effect. Histopathological examination remains essential for establishing the definitive diagnosis in diagnostically ambiguous cases.

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