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Case Report

Nonenhancing Spinal Subdural Metastatic Tumor

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Summary: We describe a case of a spinal subdural metastatic tumor that became rapidly symptomatic after a minor trauma, as a result of severe cord compression and cord hemorrhage. On MR imaging, the lesion was oval, hyperintense with a dark rim on T2-weighted fast spin-echo images, isointense to the cord on T1-weighted images, and had dark and bright areas on gradient-echo T2*-weighted images, consistent with a hyperacute-to-acute hematoma. The hemorrhagic tumor showed no evidence of contrast enhancement.

Spinal subdural hematomas are most commonly caused by anticoagulant therapy, lumbar puncture, blood dyscrasias, spinal trauma, or spinal vascular malformations (1). Subdural metastatic tumors are very uncommon, and their presentation as spinal subdural hematomas is exceedingly rare.

Case Report

A 59-year-old woman presented with quadriparesis 2 days after a minor fall on her hip in the bathroom. At the time of the injury, she felt moderate neck pain, which, the next day, progressed to paresthesias and weakness of the right extremities and then rapidly evolved to quadriplegia during next 24 hours. Before this incident, the patient was in good health and she was not taking anticoagulants or aspirin.

MR imaging revealed a well-defined oval intradural extramedullary mass, anterior and to the left of the spinal cord at the C4 and C5 levels. A rounded and localized appearance of the mass (Fig 1*A*) favored subdural over subarachnoid space location. The lesion showed high T2 signal intensity centrally, with peripheral hypointensity, and was predominantly dark on gradient-echo T2*-weighted images (Fig 1*A–C*). The mass severely compressed the cord, with evidence of intramedullary hemorrhage (Fig 1*D*). On T1-weighted images, the subdural lesion was isointense to the cord (Fig 1*E*). This signal-intensity pattern was considered consistent with hyperacute-to-acute hemorrhage, corresponding to intracellular oxyhemoglobin and deoxyhemoglobin and early clot formation. The abnormality showed no enhancement on postcontrast images (Fig 1*F, -G*). MR imaging of the brain was performed at the same time, and the findings were normal. Results of a basic laboratory work-up showed no evidence of coagulopathy.

The patient was immediately taken to surgery for C4 and C5 laminectomy and removal of the mass via a posterior approach. On opening the dura, we visualized a lesion, measuring 2 × 1 cm, in the left anterior subdural space, between the dura and the arachnoid membrane. This mass had the appearance of a well-circumscribed hematoma that was compressing and displacing the spinal cord. The mass was also surrounding the left C5 nerve roots, but no attachment of the lesion to the meninges or the spinal cord could be seen intraoperatively. The mass was completely resected in a microneurosurgical manner, followed by exact closure of the dura. There were no signs of neurological recovery in the immediate postoperative period.

Histologic examination of the hematoma revealed a metastatic tumor (Fig 2*A*), and immunohistochemistry was positive for carcinoembryonic antigen (Fig 2*B*), cytokeratin 7, and epithelial membrane antigen, indicating a probable primary breast cancer. On further questioning, we found no history of malignant neoplasms. The work-up for possible metastatic breast cancer was scheduled, and CT findings of the chest, abdomen, and pelvis were normal. Before the other diagnostic tests could be completed, the patient requested a transfer to another institution, where her condition further deteriorated. She eventually died after repeated episodes of antibiotic-resistant pneumonia and pulmonary embolism, and her family refused an autopsy.

Discussion

Spinal hematomas are rare and usually severe neurologic disorders that without adequate treatment, frequently lead to death or permanent neurologic deficit. Most cases of spinal hematoma have a multifactorial cause, and in up to one third of patients, no factor can be identified as the cause of the bleeding (2). After idiopathic spinal hematoma, cases related to anticoagulant therapy and vascular malformations represent the second and third most common categories. Spinal and epidural anesthetic procedures in combination with anticoagulant therapy are also a relatively common causative group (2). The most frequent causes of spinal subdural hematomas are anticoagulant therapy, lumbar puncture, blood dyscrasias, spinal trauma, and vascular malformations (1). Spinal subdural hematomas may sometimes be associated with intracranial hematomas as a result of traumatic intracranial injury (1). Spontaneous subdural hematomas, in which the primary cause of the hematoma cannot be detected, have occasionally been reported (3).

Epidural and subdural spinal hematomas typically present with intense sharp pain at the location of the hemorrhage, which may be followed by a pain-free interval of minutes to days, after which there is progressive paralysis below the affected spinal level, as seen in the patient we presented. According to a recent literature survey, most patients are between 55 and 70 years of age at presentation, and more than one third of them experience complete recovery (2). The less severe the preoperative symptoms are and the more quickly surgical decompression is performed, the better are the chances for recovery. Most

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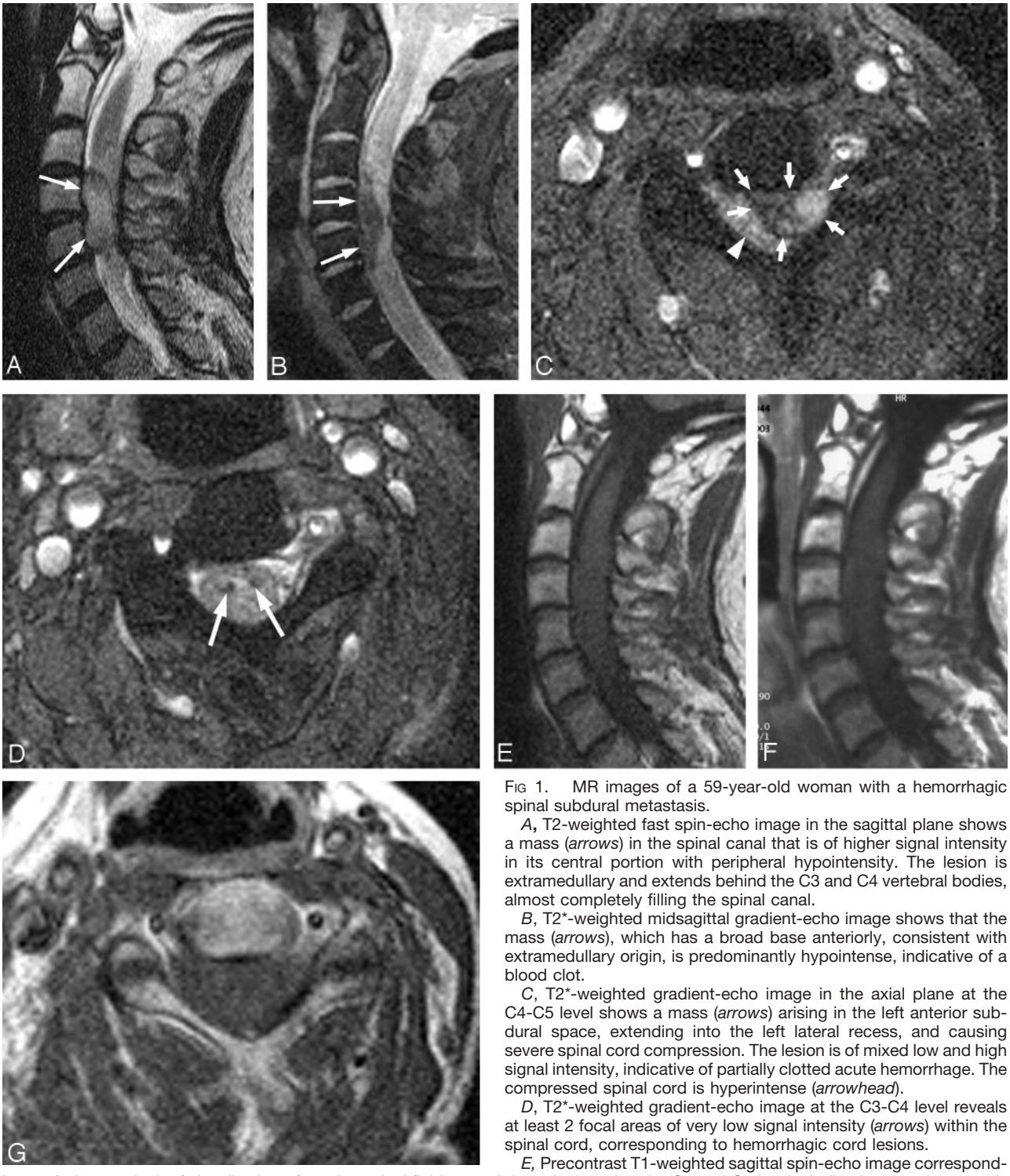

FIG 1. MR images of a 59-year-old woman with a hemorrhagic spinal subdural metastasis.

A, T2-weighted fast spin-echo image in the sagittal plane shows a mass (*arrows*) in the spinal canal that is of higher signal intensity in its central portion with peripheral hypointensity. The lesion is extramedullary and extends behind the C3 and C4 vertebral bodies, almost completely filling the spinal canal.

B, T2*-weighted midsagittal gradient-echo image shows that the mass (*arrows*), which has a broad base anteriorly, consistent with extramedullary origin, is predominantly hypointense, indicative of a blood clot.

C, T2*-weighted gradient-echo image in the axial plane at the C4-C5 level shows a mass (*arrows*) arising in the left anterior subdural space, extending into the left lateral recess, and causing severe spinal cord compression. The lesion is of mixed low and high signal intensity, indicative of partially clotted acute hemorrhage. The compressed spinal cord is hyperintense (*arrowhead*).

D, T2*-weighted gradient-echo image at the C3-C4 level reveals at least 2 focal areas of very low signal intensity (*arrows*) within the spinal cord, corresponding to hemorrhagic cord lesions.

E, Precontrast T1-weighted sagittal spin-echo image corresponding to *A* shows a lack of visualization of cerebrospinal fluid around the spinal cord at the C4 and C5 levels, indicating that the mass is isointense with the cord. The signal intensity pattern of this lesion is consistent with a combination of oxyhemoglobin (bright on T2-weighted images) with deoxyhemoglobin and early clot formation (dark areas on T2- and especially T2*-weighted images), corresponding to hyperacute-to-acute hemorrhage.

F, Postcontrast T1-weighted sagittal spin-echo image corresponding to *E* demonstrates an absence of lesion enhancement. Note the presence of contrast enhancement in the anterior epidural space at the C1-C2 level and in the pharyngeal mucosa.

G, Postcontrast axial T1-weighted spin-echo image corresponding to *C* shows a lack of lesion enhancement. The image is from the delayed set of postcontrast T1-weighted images acquired after postcontrast scanning of the brain. The absence of enhancement may be due to the mass effect of the hemorrhage and a relatively isolated location of the lesion. Contrast enhancement could perhaps have been observed with a higher (triple) dose of contrast agent.

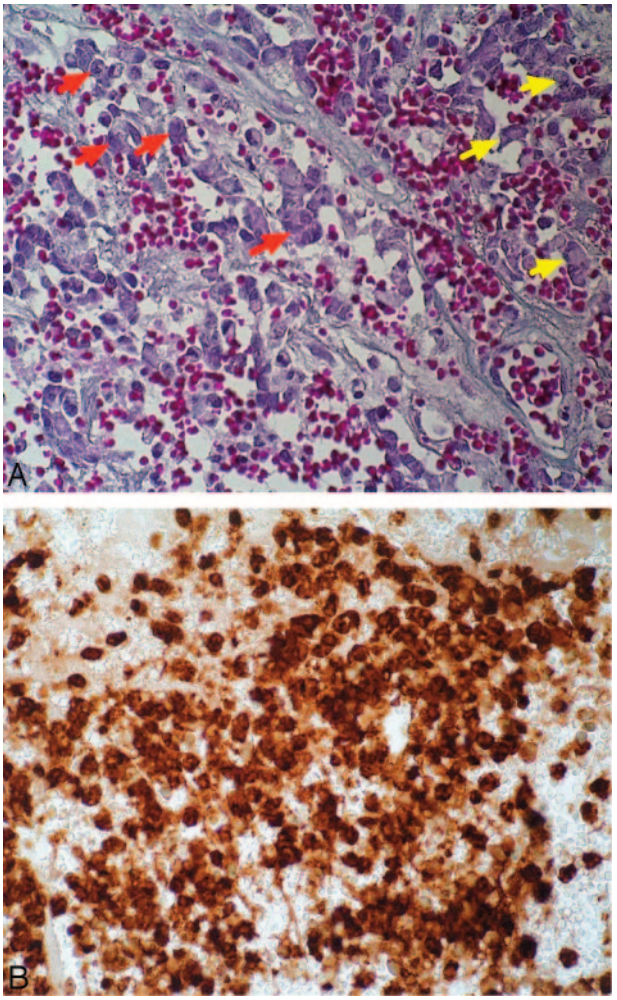

FIG 2. Histology of a hemorrhagic spinal subdural metastasis in a 59-year-old woman.

A, Photomicrograph shows numerous small anaplastic epithelial cells with nuclear polymorphism (*red arrows*). These malignant cells are also present within the blood vessels (*yellow arrows*) (Mallory, original magnification ×400).

B, Photomicrograph demonstrates strong carcinoembryonic antigen (CEA) reactivity in metastatic carcinoma cells (Immunohistochemistry for CEA, original magnification ×400).

spinal hematomas are localized dorsally to the spinal cord at the level of the cervicothoracic and thoracolumbar regions, and subarachnoid hematomas can extend along the entire length of the spinal subarachnoid space (2). An unusual feature of hematoma in the patient we described was its appearance as a focal mass on MR imaging, instead of a typical linear lesion extending over multiple vertebral segments.

The generally accepted model for the appearance of intracerebral (or intramedullary) hemorrhage on MR images attributes the changes in the signal-intensity pattern in evolving hematoma to the products of iron metabolism and to the integrity of the red blood cell. This model is not directly applicable to extraaxial hemorrhages, and acute spinal subdural hematomas are typically of low signal intensity on T2-weighted spin-echo and T2*-weighted gradient-echo sequences (4) and are frequently of heterogeneous T2 signal intensity (3).

We think that the secluded location of the hematoma in the presented patient may explain the imaging findings that were more similar to hyperacute-to-acute intraparenchymal hemorrhage. The appearance of intracerebral blood during the hyperacute phase (within the first 6 hours) on conventional MR images is predominantly determined by the water and protein content of blood, without significant influence of the hemoglobin molecules (5, 6). Hyperacute hematomas are therefore hyperintense on T2-weighted sequences and hypo- to isointense to brain and spinal cord on T1-weighted images (5–7). This appearance is followed by clot formation during the period of acute hemorrhage, when oxyhemoglobin molecules rapidly deoxygenate, yielding deoxyhemoglobin within the red blood cells. Deoxyhemoglobin is a paramagnetic substance with high susceptibility that generates a strong local magnetic field, much stronger than that in the surrounding extracellular space. The protons within red blood cells therefore precess faster from their counterparts on the other side of the cell membrane, causing rapid loss of transverse magnetization and very low signal intensity of acute clots on T2-weighted images. This loss of signal intensity is even more prominent on gradient-echo T2*-weighted images than on spin-echo T2-weighted sequences because of their high sensitivity for susceptibility effects.

Heterogeneous internal clot structure further contributes to susceptibility effects within voxels and is largely responsible for T2-signal-intensity loss of acute hematomas. "Normal" clots containing large islands of red blood cells surrounded by lakes of serum are very dark on T2 images, whereas settled blood and serum-poor homogeneous clots remain iso- to hyperintense (8). Reports in patients and studies in animal models indicate that the signal-intensity loss on T2*-weighted images may be observed almost immediately after the onset of hemorrhage (7, 9, 10). Early clot formation and the intratumoral location of hemorrhage, where it was isolated from the oxygen-rich cerebrospinal fluid, were likely responsible for areas of low signal intensity on T2-weighted images in the patient we reported. The bleeding was most likely prolonged, which explains predominantly high signal intensity on fast spin-echo T2-weighted images, consistent with presence of hyperacute hemorrhage.

Spinal metastatic disease from systemic cancer is most commonly found in the extradural compartment, typically involving the vertebrae. Leptomeningeal metastases, with a characteristic appearance of linear and nodular areas of contrast enhancement along the surface of the spinal cord, are less common and are found in patients with primary central nervous system tumors or metastatic systemic cancer (11). Even less frequent is intramedullary metastatic disease, which accounts for only approximately 1% of all metastatic lesions (12). Metastatic neoplasms to the central nervous system almost invariably demonstrate strong contrast enhancement, which may occasionally be minimal or absent if the tumors are hemorrhagic. One study found an absence of contrast enhancement in as many as 8% of intramedullary and

leptomeningeal metastases (13), whereas in a more recent publication, all the intradural extramedullary secondary neoplasms showed prominent contrast enhancement (14).

Reports that evaluated the detection of cerebral metastases found a slight increase in lesion detection on delayed (10 and 20 minutes) studies, which was, however, clearly inferior to the administration of an additional dose of contrast agent (15, 16). We believe that the absence of contrast enhancement in the patient we presented was due to the development of hemorrhage and associated mass effect, as well as to a fairly isolated and secluded location of the metastatic lesion. Even the delayed (for ≈15 minutes) postcontrast imaging did not demonstrate any contrast enhancement of the metastasis. On the basis of the studies of cerebral metastases, we believe that the lesion might have shown some contrast enhancement with a triple dose of gadolinium.

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