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# Acute Spontaneous Spinal Epidural Hematomas

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***BACKGROUND AND PURPOSE:*** **Although previous reports have characterized MR imaging features of spinal epidural hematomas (EDH), few cases have been reported during the acute or hyperacute phase within the first 48 hours. Our goal in this investigation was to correlate the MR imaging features of acute (≤48 hours) spontaneous EDH with clinical management and outcome.**

***METHODS:*** **Eight patients with acute spontaneous EDH (five men and three women; age range, 31−81 years) underwent MR imaging at 1.5 T (T1-weighted, n = 8; T1-weighted after the administration of 0.1 mmol/kg contrast material, n = 6; T2-weighted, n = 8; and T2*-weighted, n = 4). The interval from symptom onset to hospital admission ranged from immediate to 5 days. Two neuroradiologists reviewed the MR images for signal characteristics, contrast enhancement, and cord compression. Treatment and clinical outcome were correlated with the imaging findings.**

***RESULTS:*** **The EDH were located in the cervical (n = 3), cervicothoracic (n = 2), thoracolumbar (n = 2), and lumbar (n = 1) regions. On T1-weighted images, the signal intensity of the EDH was isointense to spinal cord in five cases, hyperintense in two cases, and hypointense in one case and did not correlate with time to imaging. Isointensity on T1-weighted images persisted for 5 days in one case. On T2-weighted images, all EDHs were hyperintense with focal, heterogeneous hypointensity. Cord compression was severe in six patients, moderate in one patient, and minimal in one patient. Four cases were treated conservatively with complete resolution or improvement of symptoms within 1 to 3 weeks.**

***CONCLUSION:*** **MR imaging findings were useful in establishing the diagnosis of EDH but did not influence management or predict outcome in this series. Heterogeneous hyperintensity to cord with focal hypointensity on T2-weighted images should suggest the diagnosis of acute spinal EDH. Severity of neurologic impairment had the greatest impact on management and outcome. Nonoperative treatment may be successful in cases with minimal neurologic deficits, despite cord compression revealed by MR imaging.**

Spontaneous spinal epidural hematoma (EDH) is a rare condition requiring urgent diagnosis (1−4). Patients with spontaneous spinal EDH typically present with acute onset of severe back pain and rapidly develop signs of compression of the spinal cord or cauda equina (5). Spinal EDH occurring spontaneously or after minimal trauma has been attributed most often to a venous source (5−7). Predisposing factors include coagulopathy, anticoagulation, vascular anomaly, disk herniation, Paget disease of bone, Valsalva maneuver, and, possibly, hypertension (1, 5, 8−13).

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MR imaging is considered to be the technique of choice for diagnosis (10, 14−17). Signal characteristics of subacute and chronic spinal EDH have been described (1, 10, 15, 16), but few series have focused on the MR imaging features of acute spinal EDH within the first 48 hours of presentation, when management decisions are critical (18, 19).

Increasingly, reports suggest that favorable outcome is possible with conservative management of spinal EDH (1, 9, 15, 20−23). The option of conservative management warrants reassessment of the role of MR imaging in the management of spinal EDH in the acute phase. Our aim was to correlate the MR imaging features of eight cases of acute spinal EDH occurring spontaneously or in association with minimal trauma with clinical management and outcome.

## Methods

### *Patients*

A search of the electronic medical records from 1992 to 1997 revealed eight cases of acute spinal EDH (five men and

**TABLE 1: Acute spinal EDH: clinical data.**

| Case # | Age (yrs)/sex | Neurologic Findings | Time from symptom onset to operation (hrs) | Treatment | Outcome Residual Deficit/Pain |
|---|---|---|---|---|---|
| 1 | 73M | Severe sensorimotor deficit LE | 23 | Laminectomy | Moderate residual deficit |
| 2 | 40M | Paraplegia; incontinence | 50 | Laminectomy | Minimal residual deficit |
| 3 | 42M | Neck pain | | Conservative | Pain resolved |
| 4 | 81F | Sensorimotor deficit LE pain; urinary retention | 26 | Laminectomy | No residual deficit |
| 5 | 54F | Minimal sensorimotor deficit LE; interscapular pain | | Nonoperative | No residual deficit |
| 6 | 76M | Neck pain | | Conservative | Pain resolved |
| 7 | 31F | Headache; neck pain | | Conservative | Pain resolved |
| 8 | 67M | Quadriparesis; neck pain | 10 | Laminectomy | Minimal residual deficit Pain resolved |

Note.—LE, lower extremity; M, male; F, female.

**TABLE 2: Acute spinal EDH: MR imaging features.**

| Case # | Extent | Location | Time from Symptom Onset to Imaging (hrs) | Signal Characteristics | | | | Cord Compression Grade |
|---|---|---|---|---|---|---|---|---|
| | | | | T1 | T2 | PDW | +Gd | |
| 1 | T6-L3 | Posterior | 6 | ↑ | ↑↓ | NA | NA | +++ |
| 2 | L3-L4 | Anterior/posterior | 48 | ↑ | ↑↓ | ↑ | P | +++ |
| 3 | C2-T6 | Posterior | 120 | ↔ | ↑↓ | ↑ | M | +++ |
| 4 | T7-L3 | Posterior | 24 | ↔ | ↑↓ | NA | P | +++ |
| 5 | C6-T1 | Anterior | 14 | ↔ | ↑↓ | NA | P | +++ |
| 6 | C2-C6 | Posterior | 8 | ↓ | ↑↓ | ↑ | NA | ++ |
| 7 | C1-C3 | Anterior/lateral | 48 | ↔ | ↑↓ | NA | Cen | + |
| 8 | C3-C5 | Posterior/lateral | 8 | ↔ | ↑↓ | NA | NA | +++ |

Note.—C=cervical; T=thoracic; L=lumbar; ↔=isointense with cord; ↑=hyperintense to cord; ↓=hypointense to cord; ↑↓=hyperintense with heterogeneous hypointensity; P=peripheral; Cen=central; M=mixed; +=minimal; ++=moderate; +++=severe; NA=not available.

three women; age range, 31−81 years) (Table 1). Seven of the eight patients had undergone MR imaging within 48 hours of symptom onset. Hypertension was present in three patients initially. Two patients were treated with anticoagulative agents (one with heparin and one with coumadin); in one of these patients, removal of an epidural catheter precipitated spinal EDH. One patient (patient 8) was a hemophiliac. The interval between symptom onset and imaging was within 48 hours in seven of eight cases (Table 2). Five patients presented with acute onset of neurologic deficit, two patients presented with neck pain, and one patient (patient 7) presented with both headache and neck pain (Table 1). Patient 7 initially was suspected to have subarachnoid hemorrhage. The results of CT of the head and cerebral and spinal angiograms (initial and follow-up) were negative. MR imaging of the cervical spine then was performed for patient 7 because of persistent neck pain. The mode of treatment and the outcome were recorded in each case (Table 1).

### *MR Imaging*

All patients were studied at 1.5 T with standard T1-weighted (600/8/1.5−2 [TR/TE/number of excitations]) and fast spin-echo T2-weighted (4000/102/2−4) sequences. In addition, three patients had proton density–weighted (2400/16/2) images and four patients had gradient-echo sequences (350/11/2−3; flip angle, 20°). Six patients underwent T1-weighted imaging after the intravenous administration of 0.1 mmol/kg contrast material. Two neuroradiologists reviewed the MR images and, by consensus, determined lesion location, extent, signal characteristics, and contrast enhancement. Cord compression was graded subjectively as minimal, moderate, or severe (Table 2) according to the following criteria: minimal, hematoma abuts cord and slightly deforms it; moderate, hematoma compresses cord to more than half of its original diameter; severe, hematoma flattens cord to less than half of its original diameter.

## Results

### *Location and Extent*

The spinal EDH most often was located in the cervical or cervicothoracic region and usually extended over multiple levels (Table 2, Fig 1). The spinal EDH was usually posterior to the thecal sac (Table 2, Fig 1).

### *Signal Intensity*

On T1-weighted images, the signal intensity of the spinal EDH was isointense to spinal cord in five cases (Fig 2), hyperintense in two cases, and hypointense in one case and did not correlate with time from symptom onset to imaging (Table 2). Isointensity on T1-weighted imaging persisted for 5 days in one case (patient 3) (Fig 1). On T2-

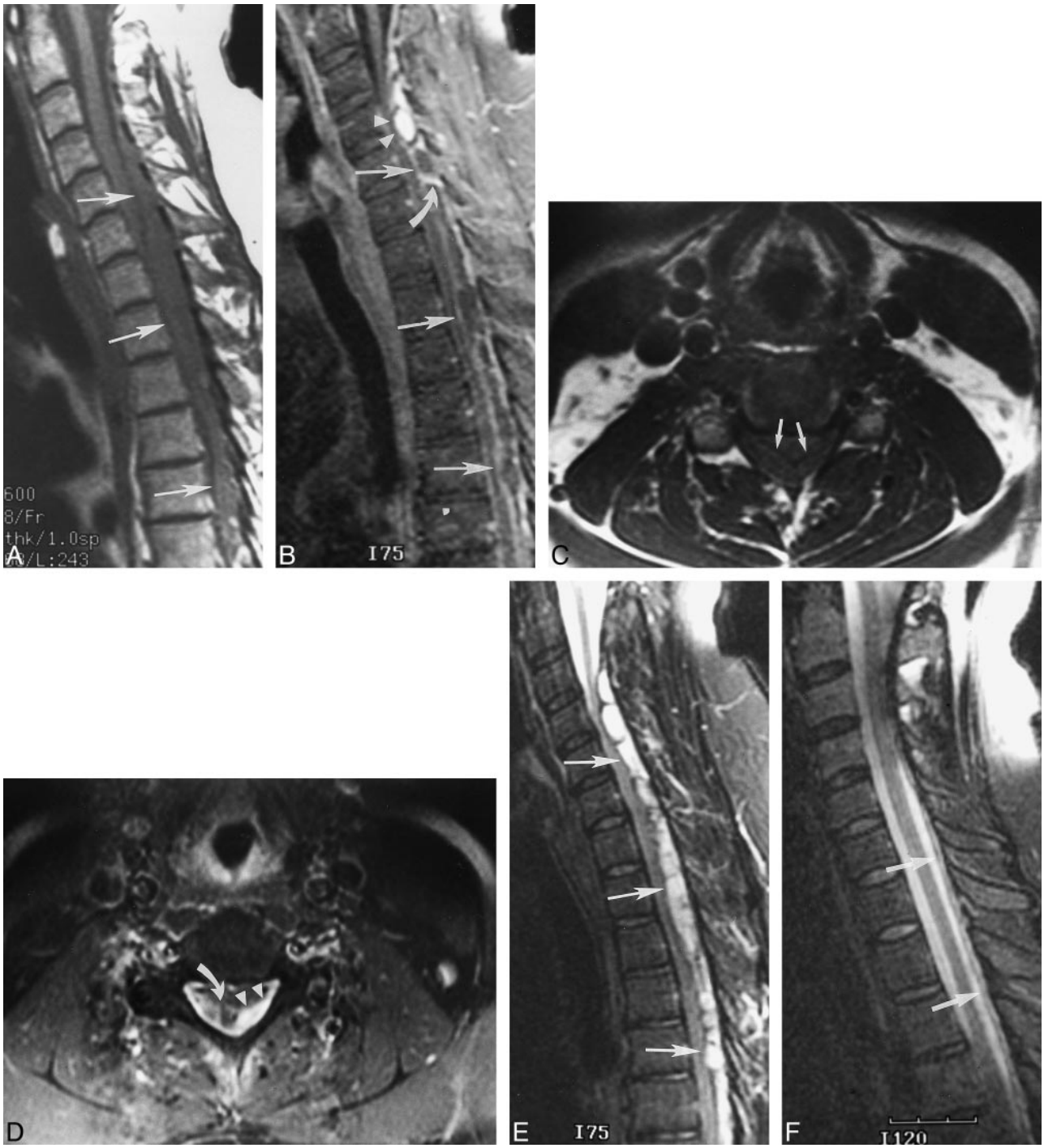


FIG 1. Patient 3.

*A,* Initial sagittal T1-weighted image (600/8/2 [TR/TE/excitations]), obtained 120 hours after symptom onset, reveals a long-segment EDH that is isointense in signal to spinal cord and is causing severe cord compression (*arrows*).

*B,* Sagittal T1-weighted image (400/8/2), obtained after the intravenous administration of contrast material, shows a mixed pattern of central (*arrowheads*) and peripheral (*straight arrows*) enhancement that likely reflects both enhancement of the hematoma itself and dural enhancement. Linear enhancement within the hematoma (*curved arrow*) may represent septa or vessels in lateral extensions of epidural fat.

*C,* Axial T1-weighted image (500/9/2), obtained at the level of the central enhancement seen in *B,* shows the absence of hyperintensity within the hematoma before the administration of contrast material (*arrows*).

*D,* Axial T1-weighted image (650/9/2), obtained after the intravenous administration of contrast material and at the level of the central enhancement seen in *B,* shows a mixed pattern of central (*arrowheads*) and peripheral (*curved arrow*) enhancement.

*E,* Sagittal T2-weighted image (4000/98/2) shows that the hematoma is hyperintense to spinal cord with focal hypointensity that may represent deoxyhemoglobin and/or fibrous septa (*arrows*).

*F,* Follow-up sagittal T2-weighted image, obtained 6 days after the initial imaging was performed, shows that nearly complete resolution of the EDH (*arrows*) was achieved with conservative management.

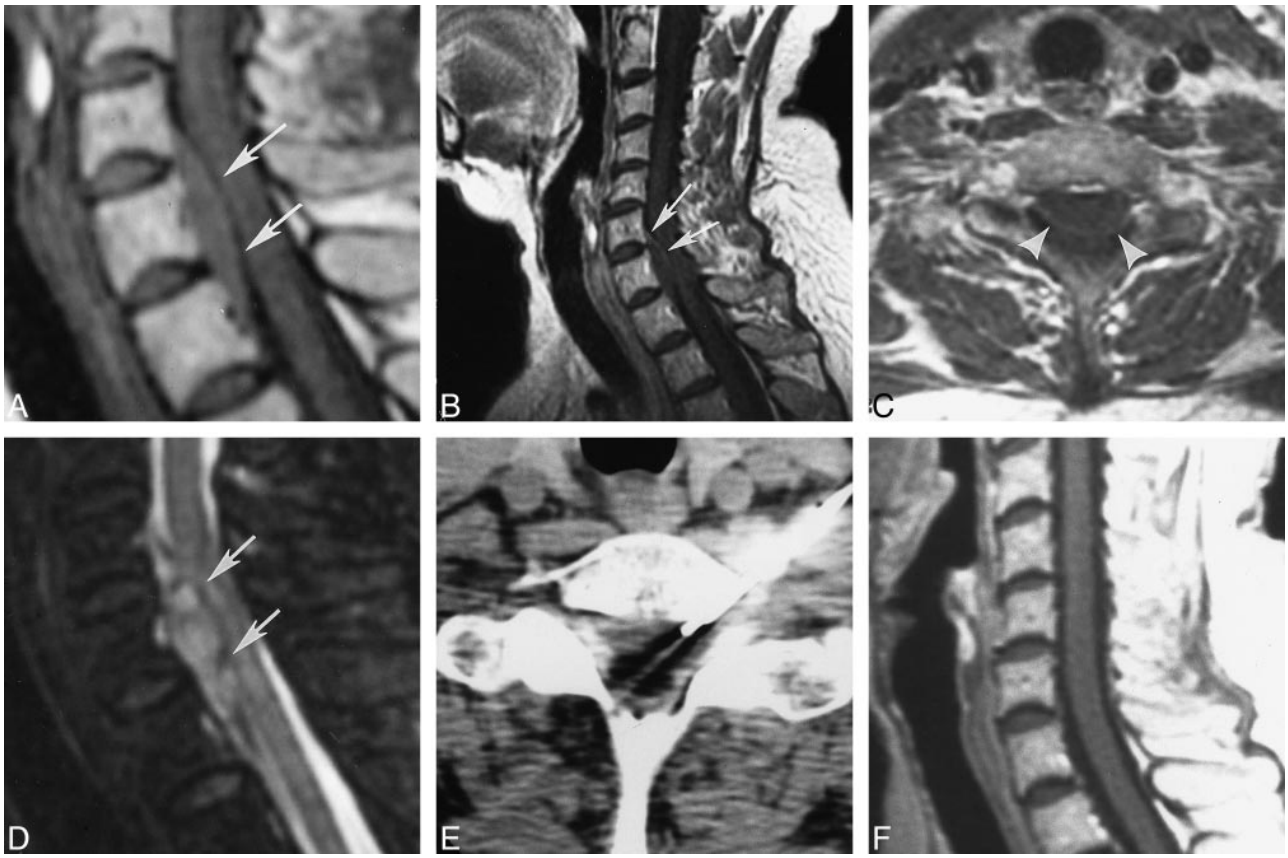


FIG 2. Patient 5.

*A,* Initial sagittal T1-weighted image (483/15/23), obtained 14 hours after the ictus, reveals a short-segment EDH that is isointense to spinal cord (*arrows*).

*B,* Sagittal T1-weighted image (400/15/2), obtained after the administration of contrast material, shows faint, peripheral enhancement (*arrows*).

*C,* Enhanced axial T1-weighted image (433/17/2) shows minimal peripheral enhancement and severe cord compression (*arrowheads*).

*D,* Sagittal T2-weighted image shows that the hematoma is slightly hyperintense to cord with focal hypointensity (*arrows*).

*E,* Axial CT image, obtained during percutaneous sampling of the epidural mass (described in the methods), shows the tip of the needle within the epidural collection.

*F,* Follow-up sagittal T1-weighted image (500/10/2), obtained 22 days after the initial imaging and percutaneous aspiration were performed, shows complete resolution of the hematoma.

weighted images, six of the eight cases of spinal EDH were heterogeneously hyperintense to spinal cord with foci of hypointensity (Table 2, Fig 1).

### *Enhancement*

Enhancement was observed in all five cases in which contrast material was administered. Peripheral enhancement was the most common pattern (Table 2, Fig 2). The peripheral pattern included enhancement that was linear along the borders of the hematoma but also had a septated appearance. A mixed pattern of central and peripheral enhancement was seen in one case (Fig 1). Purely central enhancement was observed in only one case (Fig 3). The patterns of mixed and central enhancement were seen at 48 hours or more after symptom onset.

### *Cord Compression*

Spinal cord compression was severe in six cases (Fig 1), moderate in one case, and minimal in the remaining case (Table 2).

### *Treatment*

Four patients underwent decompressive laminectomy within 50 hours (range, 10−50 hours; mean, 27.3 hours) of MR imaging (Table 1). All four patients who were not treated surgically underwent follow-up MR imaging (Fig 1). One patient (patient 5) underwent percutaneous needle biopsy of the epidural lesion because of a history of breast carcinoma, but was treated without surgery. The approach for the aspiration of the EDH in patient 5 was transforaminal; an 18-gauge spinal needle was advanced through the C6−C7 neural foramen, and a 22-gauge Ciba needle was coaxially placed to aspirate the specimen, which proved to be blood (Fig

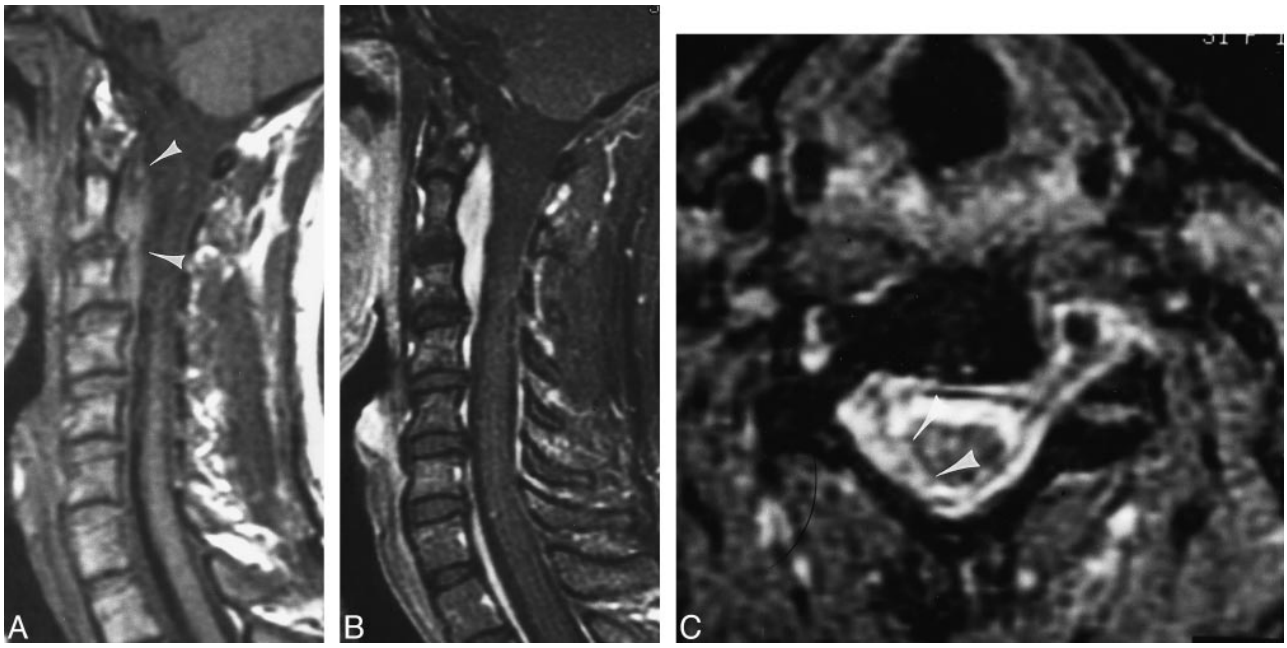


FIG 3. Patient 8.

*A,* Initial sagittal T1-weighted image (400/14/3), obtained 48 hours after the onset of headache and neck pain that clinically was suspicious for subarachnoid hemorrhage, reveals an EDH at C2–C3 (*arrowheads*) that is isointense to spinal cord.

*B,* Sagittal T1-weighted image (733/20/2), obtained after the administration of contrast material, shows striking central enhancement.

*C,* Axial gradient-echo image (500/12/2; flip angle, 20°), obtained at the C5 level, shows minimal deformity of the spinal cord from the posterolateral EDH (*arrowheads*).

2E). This procedure was performed because of the patient's history of breast cancer. The patient had no other evidence of disseminated disease and was intensely interested in knowing whether this was tumor. In three of four patients who were treated conservatively, there was nearly complete resolution of spinal EDH by 2 weeks; in one of four, there was a substantial reduction in the size of spinal EDH (Table 1).

## Discussion

### *MR Imaging*

Our primary focus in this report is the MR imaging features and management of acute (≤48 hours) spinal EDH (17). Despite numerous reports of the clinical features and management of spinal EDH, there are relatively few series that include MR imaging findings in the acute phase. In a series of 18 surgically confirmed cases of lumbar EDH, Gundry and Heithoff (10) described signal characteristics of spinal EDH an average of 169 days after presentation on long TR imaging but did not discuss T1-weighted signal characteristics. The signal characteristics of subacute and chronic spinal EDH have been described as usually hyperintense (1, 11, 12, 15, 16, 19, 20, 24, 25), and less often as isointense (1, 16, 19), to the spinal cord on T1-weighted images. The diagnosis of hematoma is obvious when hyperintense, subacute blood is present on T1-weighted images. Hyperintensity was present initially, however, in only two of our patients (patients 1 and 2), at 6 and 48 hours, respectively. The majority of the patients (five of eight patients) presented within 24 hours, and all except one showed hypointense to isointense spinal EDH on T1-weighted images (Table 1). Of the remaining three patients who presented more than 24 hours after symptom onset, the spinal EDH was isointense to spinal cord in two cases and hyperintense in one case on T1-weighted images.

Few other series have reported MR imaging findings of spinal EDH within 48 hours. Rothfus et al (16) reported three cases of spontaneous spinal EDH within 48 (n = 2) and 72 (n = 1) hours. Of these three cases, findings on T1-weighted images were described as hyperintense in one, isointense in one, and heterogeneous in one. Tarr et al (26) reported MR imaging findings in a series of 14 patients after recent spinal trauma. They included patients who had undergone MR imaging within 2 1/2 weeks of injury. The EDH (n = 3) in that series were imaged 1 week (n = 1) or 2 days (n = 2) after trauma, and therefore, only two of their cases were in the acute time frame (≤48 hours) on which our series was focused. The signal characteristics of the hematomas were described as increased signal intensity on T1- and T2-weighted images as early as 24 hours (26). It is possible that posttraumatic spinal EDH, being different in origin from spontaneous spinal EDH, may have slightly different imaging features or evolution of signal characteristics. A series of spi-

nal EDH by Holtås et al (1) studied 13 patients at 0.3 and 1.5 T and reported isointensity on T1-weighted images in only two of six cases examined within 2 days after the onset of symptoms. These authors suggested, ''T1 weighted MR images were most valuable because of the pathognomonic signal shift from isointensity with the cord in the early period to hyperintensity in the intermediate stage.'' Interestingly, however, in our series, the shift to hyperintensity on T1-weighted images with increased age of the hematoma was not useful in making the initial diagnosis, because we observed persistent isointensity as late as 120 hours after symptom onset (Fig 1, patient 3; Table 2). Because spinal EDH requires urgent diagnosis and possibly urgent surgery, follow-up imaging to confirm the diagnosis will be useful only for patients who are going to be treated conservatively. Isointensity or hypointensity on T1-weighted images is nonspecific and can be seen in association with other conditions, including lymphoma and metastasis (27); lymphoproliferative disorder or neoplasm was the initial clinical suspicion in patients 2 and 5 because those patients had histories of renal transplant and breast carcinoma, respectively. Thus, isointensity or hypointensity on T1-weighted images is a feature that may confound the diagnosis of acute spinal EDH in cases of complicated clinical history.

Reports of contrast enhancement in cases of spinal EDH are scarce. To our knowledge, only three cases, two with central and one with peripheral enhancement, have been described (11, 24, 28). The majority of the patients in our series (five of eight patients) underwent imaging after the administration of contrast material, and all except one of these had peripheral contrast enhancement. Hyperemia of dura mater resulting in thickening of adjacent meninges has been postulated as a cause of peripheral enhancement of spinal EDH (11, 24). A mixed pattern of central and peripheral septated enhancement was seen on the images of patient 3 (Fig 1). Knowledge of the anatomy of the lumbar epidural space, as reported by Hogan (29) on cryomicrotome section, may be helpful in clarifying the enhancement patterns that we observed. Hogan (29) described the posterior epidural fat as being free of septation or internal structure, except in the midline, where it is attached to the ligamenta flava by a vascular pedicle. Epidural injections that enter the posterior fat directly loculate rather than dissipate (29). The lateral epidural fat, however, is lobulated by septa (29). The linear enhancement that we observed within the hematoma may represent septa or vessels in lateral extensions of epidural fat.

In contrast, in the absence of an underlying lesion, the central pattern of enhancement may result from extravasation of contrast material by leaking vessels (28). The pattern of contrast enhancement may be useful in the differential diagnosis, because central contrast enhancement also has been attributed to tumor, aneurysm, and vascular malformation (28, 30). Unlike other reported series, most of our patients received contrast material because of complex histories, such as the presence of primary malignancy. In two of these patients (patients 2 and 5), peripheral rather than central enhancement supported the diagnosis of spinal EDH and helped to exclude an underlying cause of hemorrhage.

The signal characteristics of acute spinal EDH that we report on T2-weighted images (Table 2) in our series are in accordance with those of other series (1, 10, 16). Striking focal hypointense foci within a primarily hyperintense lesion on T2-weighted spin-echo or gradient-echo images are characteristic and were seen on the images of seven of eight of our patients. Possible causes of heterogeneity on T2-weighted imaging included deoxyhemoglobin and the fibrous septa that attach the dura to the walls of the spinal canal (17). T2-weighted pulse sequences added important diagnostic information at the time of presentation of spinal EDH when characteristic hyperintensity is usually lacking on T1-weighted images. In contrast to T2-weighted images, relatively proton density–weighted images showed diffuse hyperintensity, which is nonspecific because it also is seen frequently in other lesions, including metastasis and lymphoma (27).

### *Clinical Presentation*

The most common clinical presentation in our series was localized pain with neurologic deficit ranging from none to severe. Interestingly, two of our patients (patients 7 and 8) presented with clinical features typical of subarachnoid hemorrhage. Both of these patients who had subarachnoid hemorrhage revealed by lumbar puncture were investigated initially with CT of the head and cerebral angiography. The results of these studies and subsequent spinal angiography were also negative. One of these patients (patient 6) had a history of resection of a frontal arteriovenous malformation, but no residual arteriovenous malformation or aneurysm was detected by angiography at presentation. Persistent neck pain prompted MR imaging of the cervical spine. To our knowledge, this association of small cervical spontaneous EDH, subarachnoid hemorrhage, and presentation typical of subarachnoid hemorrhage has not been reported.

### *Treatment and Outcome*

Outcome after surgery is somewhat variable. Several authors recommend early decompressive surgery and suggest that if the operation is performed before interference with blood supply to the cord occurs, the prognosis is favorable (2−4). The neurologic recovery after surgery varies with the severity of the preoperative impairment (2) and the interval between presentation and operation (3). In our series, the patients who had the most severe neurologic deficits (patients 1, 2, 4, and 8) were

treated with laminectomy within 50 hours of ictus and all achieved excellent recovery except one patient (patient 1) in whom minimal residual neurologic deficit persisted (Table 1). These results are in accord with those of other series, because this patient (patient 1) had the most severe preoperative deficit (2). Foo and Rossier (2) reviewed the outcomes of 158 cases of spinal EDH treated surgically. Ninety-five percent of the patients who had incomplete sensorimotor deficit, in contrast with 45.3% of the patients with complete deficit, returned to baseline neurologic function after surgery (2). Surgical outcome is more favorable if spinal EDH is located in the lumbosacral region, compared with the thoracic region, and is localized to one bony level (2). Because there is a tendency for neurologic progression, most patients are treated by prompt decompressive surgery within 36 hours (3). Therefore, reliable diagnostic features within the acute (≤48 hours) phase are necessary to guide surgery, when indicated.

There are increasing numbers of reports, however, describing the resolution of spinal EDH with nonoperative management (1, 9, 15, 20−23). Two of our patients (patients 3 and 5) with severe cord compression revealed by MR imaging but with minimal sensory deficits were not treated surgically, resulting in excellent outcomes (Table 1). The spinal EDH in these cases extended over several vertebral levels and were located in the cervicothoracic and cervical regions. In one patient (patient 5), the spinal EDH had almost completely resolved within 1 week, indicating that rapid resolution of spinal EDH is possible. Interestingly, because the same patient (patient 5) had a history of breast cancer, the epidural lesion was sampled percutaneously (Fig 2E); this procedure may have both partially removed the hematoma and provided a pathway for the egress of the residual blood. This observation of resolution of EDH after needle placement has been made previously but remains unexplored as a potential treatment for spinal EDH (21, 23). In this series, we were unable to discern consistent MR imaging characteristics predictive of successful conservative treatment. Although MR imaging features, including degree of cord compression, were not predictive of outcome, MR imaging served an important role in confirming the diagnosis and resolution of the hematoma.

## Conclusion

The clinical presentation as well as the MR imaging findings may be misleading in the diagnosis of acute (≤48 hours) spinal EDH. Small spontaneous spinal EDH may mimic subarachnoid hemorrhage clinically, in which case, MR imaging plays an especially important diagnostic role. Isointensity to spinal cord on T1-weighted imaging occurs frequently in cases of acute spinal EDH and may persist for 5 days, confounding the diagnosis. Peripheral enhancement was common in our series and may exclude underlying neoplasm in cases with complex histories. Heterogeneous hyperintensity to cord with focal hypointensity on T2-weighted images should suggest the diagnosis of acute spinal EDH. Neurologic status will likely continue to dictate management, because nonoperative treatment may be successful in cases with minimal neurologic deficits, despite cord compression on MR imaging.

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## References

1. Holtås S, Heiling M, Lönntoft M. **Spontaneous spinal epidural hematoma. Findings at MR imaging and clinical correlation.** *Radiology* 1996;199:409–433
2. Foo D, Rossier A. **Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas.** *Surg Neurol* 1981;15:389–401
3. McQuarrie G. **Recovery from paraplegia caused by spontaneous spinal epidural hematoma.** *Neurology* 1978;28:224–228
4. Packer N, Cummins B. **Spontaneous epidural hemorrhage. A surgical emergency.** *Lancet* 1978;1:356–358
5. Groen R, Ponssen H. **The spontaneous spinal epidural hematoma. A study of the etiology.** *J Neurol Sci* 1990;98:121–138
6. Groen RJ, Groenewegen HJ, van Alphen HA, Hoogland PV. **Morphology of the human internal vertebral venous plexus. A cadaver study after intravenous Araldite CY 221 injection.** *Anat Rec* 1997;249:285–294
7. Joseph AP, Vinen JD. **Acute spinal epidural hematoma.** *J Emerg Med* 1993;11:437–441
8. Lee K, McWhorter J, Angelo J. **Spinal epidural hematoma associated with Paget's disease.** *Surg Neurol* 1988;30:131–134
9. Brawn L, Bergval U, Davies-Jones G. **Spontaneous spinal epidural haematoma with spontaneous resolution.** *Postgrad Med J* 1981;62:885–887
10. Gundry C, Heithoff K. **Epidural hematoma of the lumbar spine. 18 surgically confirmed cases.** *Radiology* 1993;187:427–431
11. Caldemeyer K, Mocharla R, Moran C, Smith R. **Gadolinium enhancement in the center of a spinal epidural hematoma in a hemophiliac.** *J Comput Assist Tomogr* 1993;17:321–323
12. Muhonen M, Piper J, Moore S, Menezes A. **Cervical epidural hematoma secondary to an extradural vascular malformation in an infant. Case report.** *Neurosurgery* 1995;36:585–588
13. David S, Salluzzo RF, Bartfield JM, Dickinson ET. **Spontaneous cervicothoracic epidural hematoma following prolonged Valsalva secondary to trumpet playing.** *Am J Emerg Med* 1997;15:73–75
14. D'Angelo V, Bizzozero L, Talamonti G, Ferrara M, Colombo N. **Value of magnetic resonance imaging in spontaneous extradural spinal hematoma due to vascular malformation (case report).** *Surg Neurol* 1990;34:343–344
15. Saito S, Katsube H, Kobayashi Y. **Spinal epidural hematoma with spontaneous recovery demonstrated by magnetic resonance imaging.** *Spine* 1994;19:483–486
16. Rothfus W, Chedid M, Deeb Z, Abla A, Maroon J, Sherman R. **MR imaging in the diagnosis of spontaneous spinal epidural hematomas.** *J Comput Assist Tomogr* 1987;11:851–854
17. Donovan Post M, Becerra J, Madsen P, et al. **Acute spinal subdural hematoma. MR and CT findings with pathological correlates.** *Am J Neuroradiol* 1994;15:1895–1905
18. Shen C-C, Wang Y-C, Yang D-Y, Wang F-H, Shen B-H. **Brown-Séquard syndrome associated with Horner's syndrome in cervical epidural hematoma.** *Spine* 1995;20:244–247
19. Avrahami E, Tadmor R, Ram Z, Feibel M, Itzhak Y. **MR demonstration of spontaneous acute epidural hematoma of the thoracic spine.** *Neuroradiology* 1989;31:89–92
20. Bernsen P, Haan J, Vielvoye G, Peerlinck K. **Spinal epidural hematoma visualized by magnetic resonance imaging.** *Neuroradiology* 1988;30:280

21. Harik S, Raichle M, Reis DJ. **Spontaneously remitting spinal epidural hematoma in a patient on anticoagulants.** *N Engl J Med* 1971;284:1355–1357
22. Hernandez D, Vinuela F, Feasby T. **Recurrent paraplegia with total recovery from spontaneous spinal epidural hematoma.** *Ann Neurol* 1982;11:623–624
23. Priest W. **Epidural hemorrhage due to hemophilia.** *Lancet* 1935; 2:1289–1291
24. Crisi G, Sorgato P, Colombo A, Scarpa M, Falasca A, Angiari P. **Gadolinium-DTPA-enhanced MR imaging in the diagnosis of spinal epidural haematoma. Report of a case.** *Neuroradiology* 1990;32:64–66
25. Latham J, Dracopoulos G, Hall D. **Cervical epidural haematoma following minor trauma.** *Aust NZ J Surg* 1993;63:985–986
26. Tarr R, Drolshagen L, Kerner T, Allen J, Partain C, James A. **MR imaging of recent spinal trauma.** *J Comput Assist Tomogr* 1987; 11:412–417
27. Mascalchi M, Torselli P, Falaschi F, Dal Pozzo G. **MRI of spinal epidural lymphoma.** *Neuroradiology* 1995;37:303–307
28. Chen C, Ro L. **Central gadolinium enhancement of an acute spontaneous spinal epidural hematoma.** *Neuroradiology* 1996; 38:114–116
29. Hogan Q. **Lumbar epidural anatomy.** *Anesthesiology* 1991;75: 767–775
30. Sze G, Krol G, Zimmerman R, Deck M. **Malignant extradural spinal tumors. MR imaging with Gd-DTPA.** *Radiology* 1988; 167:217–223