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Subacute Necrotizing Myelopathy: MR Imaging in Four Pathologically Proved Cases

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This report describes the MR and correlative imaging findings of four histologically proved cases of subacute necrotizing myelopathy in which there was no evidence of a spinal dural arteriovenous fistula. Subacute necrotizing myelopathy is characterized clinically by progressive motor and sensory deterioration, and pathologically by necrosis in the spinal cord. Initial MR imaging showed focal enlargement of the spinal cord and nonspecific T1 and T2 lengthening. Rimlike enhancement was demonstrated in one case. Clinically, steroid therapy failed in all four patients. Follow-up MR scans showed two slightly enlarged lesions, one stable thoracolumbar lesion, and atrophy of a cervical lesion. Open spinal cord biopsies revealed foci of necrosis and abnormal parenchymal vessels with thickened hyalinized walls. A prolonged course distinguishes subacute necrotizing myelopathy from acute transverse myelitis, but the clinical course and imaging appearance are similar to those of intramedullary tumor. Rimlike rather than solid contrast enhancement may be a distinguishing feature.

In the absence of a demonstrable spinal dural arteriovenous fistula, the radiologic differentiation of subacute necrotizing myelopathy from tumor is probably impossible, and biopsy establishes the correct diagnosis.

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Despite the now prevalent use of MR imaging as the primary spinal investigative technique [1–11], there is only occasional mention or reference to subacute necrotizing myelopathy (SNM) [7, 8, 11]. We report the clinical findings, MR and correlative imaging results, and pathology in four proved cases. Clinically and radiologically, our group of patients resembled patients with intramedullary tumors. The radiologic and pathologic findings, possible etiology, and important roles of enhanced MR and imaging follow-up are discussed.

Materials and Methods

The MR, CT, and myelographic findings in four patients with pathologic diagnoses of SNM were reviewed retrospectively. All the patients had a history of gradual motor and sensory deterioration in the lower limbs and impaired bowel and bladder function; one patient also had a deficit in her arms. Each patient experienced acute clinical worsening preceding hospital admission.

Three patients underwent myelography with water-soluble contrast material followed by CT sections through the affected areas; 5-mm collimation was used. MR imaging (1.5-T superconducting magnet) was performed in all four patients, and was done with a rectangular 18- by 30-cm surface coil. All patients had unenhanced MR scans consisting of spin-echo T1-weighted sagittal scans, T2-weighted sagittal scans, and T1-weighted axial scans. The T1-weighted sequence was 600/20/4 (TR/TE/excitations) and the T2-weighted sequence (done with cardiac gating) was approximately 2000/70/2 (TR/TE/excitations). Gradient-moment nulling techniques were used in all patients to decrease CSF flow artifacts. For the T1-weighted sagittal sequences, 3-mm sections with an interslice gap of 0.6 mm were obtained; the gap was increased to 1 mm for the T2-weighted sequences. Five-millimeter-

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thick T1-weighted axial scans with an interslice gap of 1 mm were obtained through the region of interest. The field of view was 24 cm for sagittal scans and 20 cm for axial scans; the matrix was 256 × 256 for each case. After the preliminary sequences, one patient (case 4) received IV gadopentetate dimeglumine at a dose of 0.1 mmol/kg followed immediately by a T1-weighted sequence. A follow-up MR scan was obtained in each patient 1–7 months after the initial study.

Results

The clinical histories, laboratory values, radiologic findings, surgical appearance, biopsy results, and postoperative courses for each of the patients are summarized in Table 1.

The clinical histories of all four patients were similar and consisted of progressive motor and sensory deterioration punctuated by abrupt worsening of symptoms. Two patients (cases 1 and 2) had impaired motor and sensory function for 1–2 years before a relatively abrupt onset of paraplegia and loss of bowel and bladder control. Following acute deterioration (approximately at the time of the initial MR), each patient received steroid therapy. During the interval between MR studies (range, 1–7 months), no patients showed significant clinical improvement.

CSF analyses from all patients showed elevated protein levels ranging from three to five times normal (normal, 15–45 mg/dl) and lymphocytosis. Electromyograms (obtained in three patients) in conjunction with imaging studies and clinical findings suggested a thoracic cord tumor in case 1, a lower motor neuron lesion and cauda equina compression (in keeping with a herniated disk or extramedullary tumor) in case 2, and myelitis in case 3. Focal cord expansion was recognized in two of three patients who underwent CT myelography.

The initial MR evaluation in each case revealed focal intramedullary expansion of the spinal cord (three thoracolumbar and one cervical) but no evidence of a spinal dural arteriovenous fistula (AVF). The lesions (each expanded segment) exhibited isointense or decreased signal on T1-weighted images and corresponding increased signal on T2-weighted images when compared with the normal spinal cord (Figs. 1A–1D and 2A–2C).

On interval follow-up 1–7 months later, two lesions (cases 1 and 3) showed further slight enlargement (Figs. 1E–1I), one lesion (case 2) was unchanged, and one lesion (case 4) was smaller. Enhanced MR images in case 4 initially showed rimlike enhancement and central decreased signal (Figs. 2D and 2E). Four months later, the intramedullary lesion was slightly smaller and showed no enhancement after the administration of gadopentetate dimeglumine (Figs. 2F–2H).

All four patients underwent open surgical biopsy. In each case, yellowish discoloration and focal enlargement of the cord was identified. In two cases (cases 1 and 2), prominent veins were found on the surface of the cord, but there was no other evidence of a spinal dural AVF and no hemorrhage or thrombosis. In every case, characteristic pathologic findings of SNM were identified (Fig. 3), including abnormal intraparenchymal vessels with thickened hyalinized walls, central coagulative necrosis, demyelination, lipid-laden macrophages, and gliosis. There was no evidence of tumor or infection (gram stains and cultures were all negative) in the biopsies. Initially, gliosis was misinterpreted as low-grade astrocytoma in two biopsies (cases 1 and 4).

TABLE 1: Summary of Four Pathologically Proved Cases of Subacute Necrotizing Myelopathy

Case No.	Age (yr)	Sex	Clinical Findings	CSF	Electromyography	CT Myelography	Spinal Cord MR	Surgical Findings	Postbiopsy Course
1	58	M	Progressive P & P for 1 yr; acute paraplegia; loss of bowel & bladder function	Increased protein; lymphocytosis	Thoracic cord lesion, probably tumor	Not done	Thoracic FFE; increased T2 signal from T4 to conus; 7 mo later, slight enlargement; no spinal dural AVF	Vein adherent to enlarged spinal cord; sonography suggested foci of necrosis or small cysts; quick-section histology initially suggested low-grade astrocytoma	No change
2	62	M	Progressive P & P for 2 yr; acute paraplegia	Increased protein; lymphocytosis	Radiculopathy; disk herniation or extramedullary tumor	Enlarged conus medullaris	Conus FFE; decreased T1 signal; increased T2 signal; 1 mo later, no change; no spinal dural AVF	Vein adherent to enlarged conus; myelotomy yielded necrotic material	No improvement; bladder became atonic
3	77	F	Progressive P & P over 3 mo; acute paraplegia; loss of bowel & bladder function	Increased protein; lymphocytosis	Supported inflammatory process/ myelitis	Normal	Conus FFE with increased T2 signal; 2 mo later, slight FEE, decreased T1 signal in conus; no spinal dural AVF	Enlarged conus; myelotomy yielded necrotic material	No change
4	47	F	Brown-Séquard syndrome (C4 level) developed over 3 wk; acute worsening of right-sided paresis	Increased protein; lymphocytosis	Not done	Enlarged cervical cord	Cervical FEE, right > left; increased T2 signal, rim enhancement; 4 mo later, lesion smaller & no enhancement; no spinal dural AVF	Asymmetric enlarged cervical cord; myelotomy yielded white friable material; quick-section histology initially suggested low-grade astrocytoma	No change

Note.—P & P = paresis and paresthesias; FFE = focal fusiform enlargement; AVF = arteriovenous fistula.

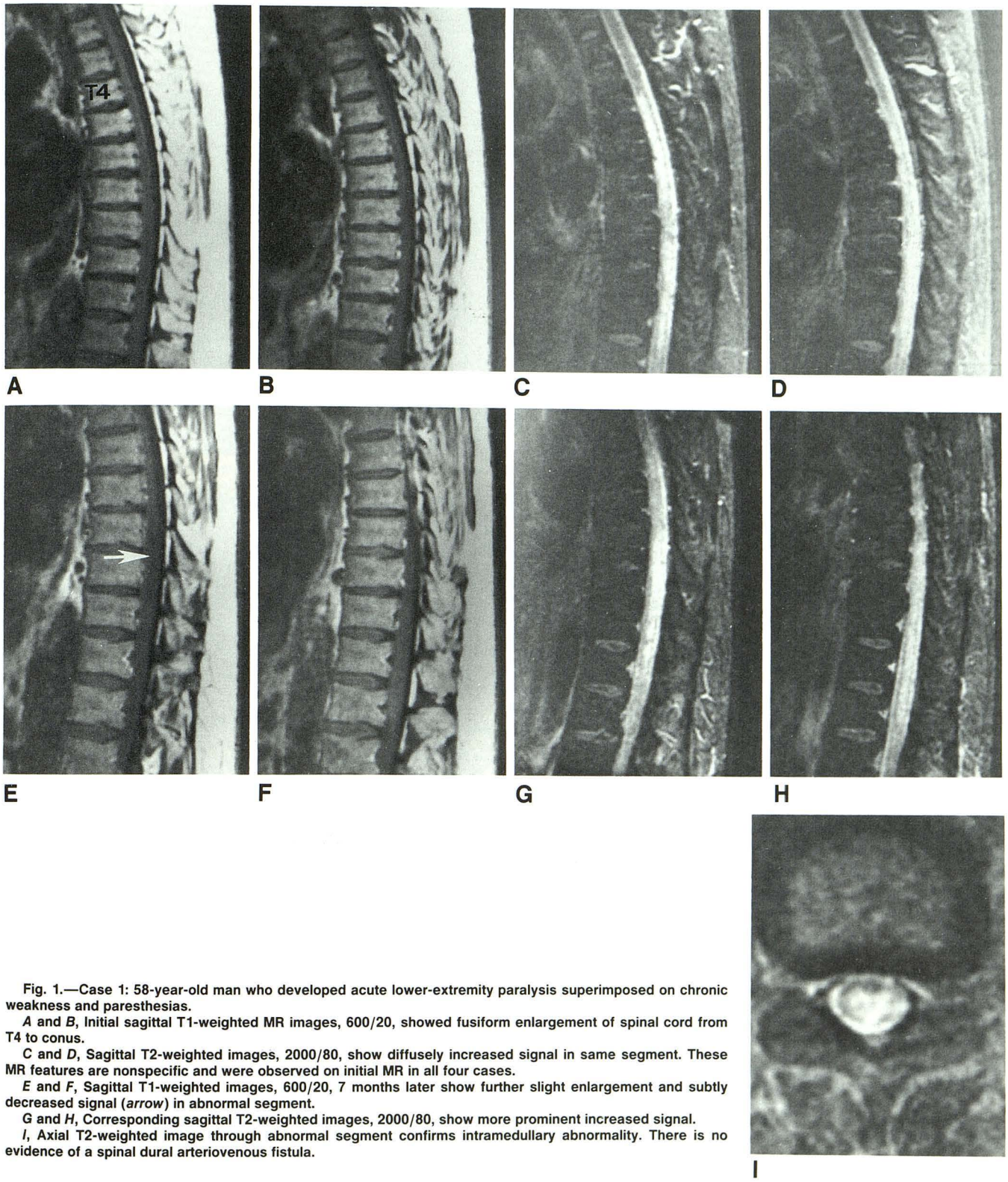


Fig. 1.—Case 1: 58-year-old man who developed acute lower-extremity paralysis superimposed on chronic weakness and paresthesias.

***A* and *B*, Initial sagittal T1-weighted MR images, 600/20, showed fusiform enlargement of spinal cord from T4 to conus.**

***C* and *D*, Sagittal T2-weighted images, 2000/80, show diffusely increased signal in same segment. These MR features are nonspecific and were observed on initial MR in all four cases.**

***E* and *F*, Sagittal T1-weighted images, 600/20, 7 months later show further slight enlargement and subtly decreased signal (*arrow*) in abnormal segment.**

***G* and *H*, Corresponding sagittal T2-weighted images, 2000/80, show more prominent increased signal.**

***I*, Axial T2-weighted image through abnormal segment confirms intramedullary abnormality. There is no evidence of a spinal dural arteriovenous fistula.**

Discussion

SNM is an uncommon disease process characterized clinically by progressive neurologic deterioration and pathologically by necrosis in the spinal cord. In most patients, the underlying abnormality is now believed to be a spinal dural AVF [6, 10–13]. The fistula causes stagnation (slow flow) in the spinal medullary veins leading to impaired spinal cord

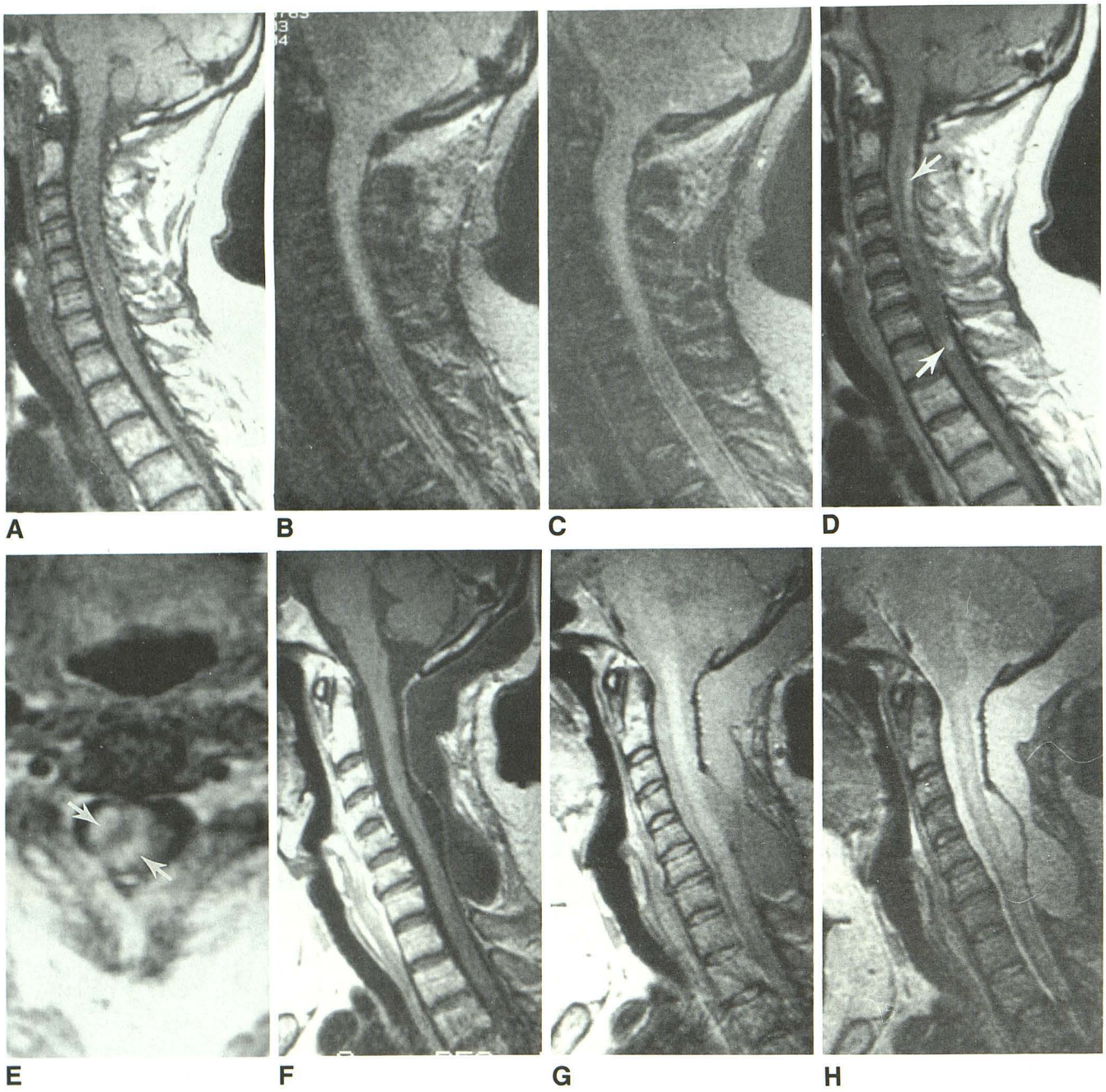


Fig. 2.—Case 4: 47-year-old woman with Brown-Séquard syndrome.

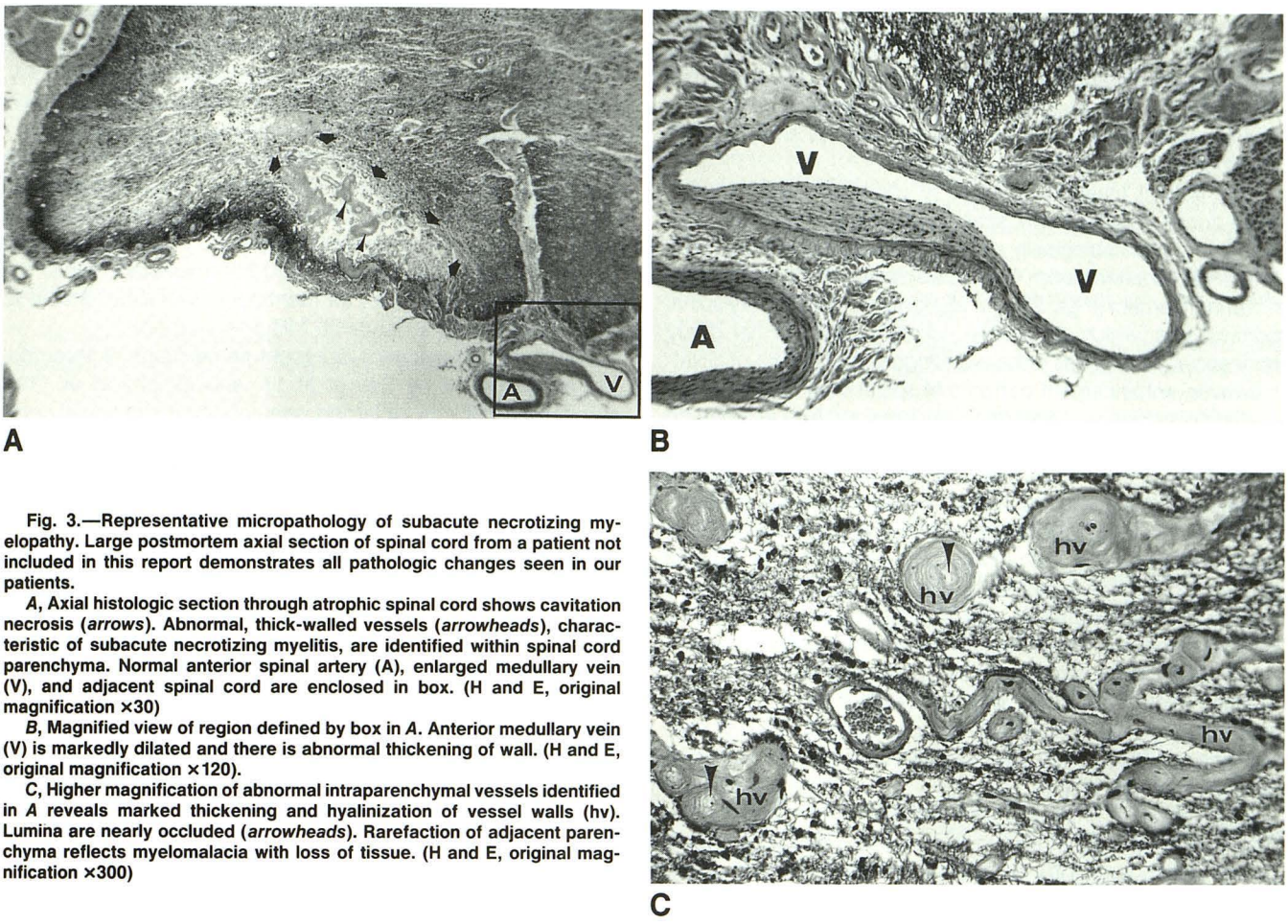
***A–C*, Sagittal T1-weighted image, 600/20 (*A*), shows fusiform enlargement of cervical spinal cord and central decreased signal corresponding to abnormally increased signal on sagittal T2-weighted images 2000/70 (*B* and *C*).**

***D* and *E*, Sagittal (*D*) and axial (*E*) T1-weighted enhanced images, 600/20, show peripheral rimlike enhancement (*arrows*). There is more disease in right half of spinal cord, correlating with clinical findings. There is no evidence of a spinal dural arteriovenous fistula. In preparation for surgical resection, laminectomies were performed from C2 through C6; however, biopsy yielded subacute necrotizing myelopathy.**

***F*, Follow-up T1-weighted enhanced image, 600/20, 4 months later shows cervical cord atrophy and no abnormal enhancement.**

***G* and *H*, Corresponding proton density, 2000/30 (*G*), and T2-weighted, 2000/70 (*H*), sagittal images show diffuse, abnormally increased signal. MR findings are consistent with sequelae of long-standing ischemia and infarction. Biopsy, although small, may have contributed to final MR appearance.**

venous drainage and perfusion [12–16]. Subacute clinical deterioration probably reflects increasing venous pressure (or decreased arterial pressure), which further impairs spinal cord perfusion and leads to infarction. These patients may be considered to have the Foix-Alajouanine syndrome. This syndrome was originally described by Foix and Alajouanine [17] in 1926. Previous reports investigating this syndrome implicated infection, thrombophlebitis, spontaneous thrombosis of a vascular malformation, and a specific degenerative disease of spinal cord vessels as possible causes [17–21].



A B C

Fig. 3.—Representative micropathology of subacute necrotizing myelopathy. Large postmortem axial section of spinal cord from a patient not included in this report demonstrates all pathologic changes seen in our patients.

***A*, Axial histologic section through atrophic spinal cord shows cavitation necrosis (*arrows*). Abnormal, thick-walled vessels (*arrowheads*), characteristic of subacute necrotizing myelitis, are identified within spinal cord parenchyma. Normal anterior spinal artery (A), enlarged medullary vein (V), and adjacent spinal cord are enclosed in box. (H and E, original magnification ×30)**

***B*, Magnified view of region defined by box in *A*. Anterior medullary vein (V) is markedly dilated and there is abnormal thickening of wall. (H and E, original magnification ×120).**

***C*, Higher magnification of abnormal intraparenchymal vessels identified in *A* reveals marked thickening and hyalinization of vessel walls (hv). Lumina are nearly occluded (*arrowheads*). Rarefaction of adjacent parenchyma reflects myelomalacia with loss of tissue. (H and E, original magnification ×300)**

Men are affected more than women, usually in the sixth and seventh decades of life, which parallels the prevalence of spinal dural AVFs [10, 12–15, 22]. Clinically, there is an initial spastic paraparesis resembling an upper motor neuron lesion; the legs later become flaccid with occasional fasciculations indicating dysfunction of the lower motor neuron unit as well. By the time of presentation, there is almost always a sensory deficit or paresthesia. This typically involves the posterior columns and spinothalamic tracts and is temporally followed by impaired bowel and bladder function.

Clinically, it may be difficult or impossible to distinguish SNM from intrinsic spinal cord tumors or transverse myelitis, as acute or subacute neurologic deterioration can be seen with all these processes. However, chronic pain and gradual worsening of the paresis over a period of several months or years would be unlikely in transverse myelitis, as would the absence of significant clinical improvement after the acute episode. Furthermore, there is often some response to trials of high-dose steroids in myelitis and other inflammatory lesions such as sarcoids [23]. Typically, and for the patients reported here, SNM does not respond to steroids.

In addition to the pathologic finding of necrotizing myelopathy, and clinical finding of subacute neurologic deterioration, the classic description of the Foix-Alajouanine syndrome includes abnormal vessels on myelography and a normal angiogram (prior to superselective angiographic techniques). In our series, three patients had myelograms, but no abnormal vessels were identified; no patients underwent spinal angiography. Therefore, from the neuroimaging standpoint, the patients reported here do not meet the classic diagnostic radiologic criteria for Foix-Alajouanine syndrome.

All four patients were investigated with MR, three after CT myelography. MR imaging demonstrated fusiform enlargement of the affected spinal cord in all patients, three in the thoracolumbar region and one in the cervical area. Enlargement was also recognized in two of three cases on CT myelography. No abnormal vessels or vascular malformations were identified on MR, but flow-sensitive sequences were not used specifically.

On T1-weighted MR images, the lesions (enlarged segments) exhibited hypointense or isointense signal (Figs. 1A, 1B, and 2A) and corresponding increased signal on T2-weighted images (Figs. 1C, 1D, 2B, and 2C) when compared with the normal spinal cord. These MR features are common to many disease processes, including (but not limited to) primary spinal cord tumor, myelitis, and sarcoidosis [1, 2, 5, 10, 23–25].

This combination of morphologic and signal abnormalities are of little differential diagnostic value. Follow-up MR scans may be diagnostically helpful. Specifically, MR scans obtained

in two patients 1 and 4 months later showed no change and decreased lesion size, respectively. We believe regression or stability of a spinal cord lesion over time supports a nonneoplastic origin. However, spinal cord tumor growth is variable, and follow-up over a few months is too short an interval to conclusively assess change in size.

One patient (case 4) received IV gadopentetate dimeglumine during the initial MR scan, and to our knowledge, this was the first pathologically proved case of SNM imaged with the MR contrast material. The lesion demonstrated peripheral rim enhancement (Figs. 2D and 2E), unlike most primary spinal cord tumors, which show solid enhancement. Further study may show this pattern to be a distinguishing feature of SNM. However, enhancement patterns of spinal cord abnormalities (including tumor) are known to be variable, and in isolation cannot be used to distinguish between diseases.

There have been few reports on imaging in SNM. Dillon et al. [8], in their series of spinal cord lesions examined before and after IV administration of gadopentetate dimeglumine, described four AVFs. Two of four cases were associated with spinal cord infarction and were well seen only on enhanced images. They attributed T1 and T2 lengthening to edema and myelomalacia and abnormal enhancement to breakdown of the blood-cord barrier. Sze et al. [7], in their series of intramedullary lesions studied with gadopentetate dimeglumine, included three patients with thrombosed spinal vascular malformations. They found focal hematomas but no abnormal vessels or enhancement.

Criscuolo et al. [13] reported five patients with "early" Foix-Alajouanine syndrome, and each was documented to have a patent spinal dural AVF. All five patients were treated with a combination of intravascular embolization and surgery, resulting in subsequent improvement of motor and sensory deficits. The undeniable value of angiography to diagnose, and intravascular embolization to treat, spinal dural AVFs is not being disputed; however, the authors did not compare the angiographic findings with MR, and cord biopsies were not performed, leaving some doubt as to whether necrotizing myelopathy was in fact present. None of our patients underwent angiography. Therefore, at best, we can only infer the presence of a spinal dural AVF and the causal relationship to SNM. None of the MR studies performed in our patients revealed spinal dural AVFs, and it is possible that other as yet undiscovered or unrecognized entities could cause the same pathologic findings.

We have shown that the recognized pathologic end stage (necrotizing myelopathy) of Foix-Alajouanine syndrome is present when MR shows a focal enlarged spinal cord with rim enhancement. Further investigation of these patients with superselective spinal angiography is controversial. Certainly, the clinical status, duration of symptoms, and MR findings (i.e., evidence of a patient spinal dural AVF) must be considered. If a patent spinal dural AVF is demonstrated, there may be a role for delayed intravascular intervention or surgery.

All four patients reported here had biopsies. The pathology was similar in all four cases, but actual necrosis was identified in only three cases. Each biopsy showed the characteristic pathology of SNM, including abnormal clusters of thick-walled hyalinized vessels within the spinal cord, demyelination, myelomalacia, and foamy lipid-laden macrophages [12, 14, 17] (Fig. 3). There was also a component of gliosis in each biopsy, which explains the initial pathologic "quick-section" diagnosis of low-grade astrocytoma in two cases.

We are in agreement with the theory of elevated venous pressure in the draining vein of a dural AVF causing impaired spinal cord perfusion and the pathologic changes of SNM. To our knowledge, no other entity causes the same pathologic picture (in particular the thickened intramedullary veins) described above. However, as mentioned, we were unable to demonstrate a dural fistula on MR or at surgery.

There was no evidence of recent hemorrhage or thrombosis, as described by Sze et al. [7] and Minami et al. [11], respectively. Slightly enlarged veins on the distal spinal cord/conus at surgery in two patients were possibly related to a spinal dural AVF beyond the operative site or one that had previously been obliterated. These veins were believed to be patent, but a biopsy was not performed nor could their entire length be examined. We believe that myelomalacia and gliosis, with or without necrosis, were responsible for T1 and T2 lengthening, and the peripheral MR enhancement observed subacutely in case 4 was secondary to ischemia/infarction and breakdown of the blood-cord barrier. Once infarcted, the involved spinal cord may become atrophic and enhancement less evident or absent (Figs. 2F–2H).

The reported prognosis for Foix-Alajouanine syndrome is dismal [11, 15, 17–19] and consistent with the development of SNM. Acutely or subacutely, there is no satisfactory noninterventional treatment [13, 14]. After irreversible ischemic damage (i.e., after cord necrosis has occurred), there is little or no chance for significant recovery. Necrosis was found in the biopsies of three of four patients, and none have shown any clinical improvement.

SNM is an unusual pathologic process occurring in the spinal cord; its *pathologic* findings are identical to those in Foix-Alajouanine syndrome. The latter is believed to represent the end stage of chronically elevated venous pressure distal to a spinal dural AVF. We think it is important to emphasize that Foix-Alajouanine syndrome and SNM are not the same entity; nor do we imply that a spinal dural AVF is the only cause of SNM. SNM may have other causes; we found no MR evidence of a spinal dural AVF in the four proved cases of SNM in this report. It could be argued, however, that we failed to exclude Foix-Alajouanine syndrome by not performing angiography.

The main clinical-radiologic differential diagnoses in SNM are transverse myelitis and primary spinal cord tumor. Considered alone, the unenhanced MR appearance and signal characteristics of SNM do not discriminate between these diagnostic possibilities. MR demonstration of spinal cord lesion stability or atrophy over months or years and peripheral rimlike enhancement after contrast administration, together with the clinical findings of gradual progressive deterioration, should raise the diagnostic possibility of SNM.

The preoperative diagnosis of SNM is difficult to determine, perhaps impossible unless a spinal dural AVF is demonstrated, and a biopsy is usually necessary to confirm the

diagnosis. The role of superselective spinal angiography in the group of patients discussed has yet to be determined. Certainly, the clinical status and MR findings should be considered before proceeding to angiography. Further study including angiography will probably be necessary to assess the sensitivity of MR for spinal dural AVFs and prior to invoking other explanations for SNM in the absence of a spinal dural AVF.

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The reader's attention is directed to the commentary on this article, which appears on the following pages.