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Acute Transverse Myelopathy: Spinal and Cranial MR Study with Clinical Follow-up

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PURPOSE: To evaluate the contribution of MR in determining the cause of acute transverse myelopathy, to determine the frequency and types of the intracranial lesions detectable on MR at the onset of the disease, and to monitor clinical and MR evolution of the disease. **METHODS:** Spinal and cranial MR images were obtained for 30 patients with acute transverse myelopathy. Gadopentetate dimeglumine was administered in 10 patients. Mean follow-up time was 18 months. **RESULTS:** Spinal cord MR findings were abnormal in 14 of 30 patients. The abnormal MR can be divided into group A, in which one segment was involved (8 patients), and group B, in which more than one segment was involved (6 patients). In both groups there were 2 patients with enhancing lesions. Enhancement was less homogeneous in the group B patients. Enhancement did not change with increased length of lesion. At follow-up, the diagnostic categories of the patients were multiple sclerosis (8 patients), encephalomyelitis (1 patient), viral myelitis (3 patients), and myelopathy of unknown cause (18 patients). After the episode of acute transverse myelopathy, in 4 of 8 patients in group A and in 4 of 5 patients with normal spinal MR but abnormal brain MR findings clinical signs of multiple sclerosis developed. In no patients in group B did multiple sclerosis develop. The final diagnoses for the 4 patients with gadolinium-enhancing spinal lesions were myelopathy of unknown cause (2 patients), multiple sclerosis (1 patient), and viral myelitis (1 patient). **CONCLUSION:** MR contributed to establishing the diagnosis in 40% of our cases.

Index terms: Spinal cord, myelopathy; Spinal cord, magnetic resonance; Sclerosis, multiple

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In acute transverse myelopathy (ATM), acute motor, sensory, and autonomic dysfunction of the spinal cord develop in the absence of preexisting neurologic diseases and spinal cord compression. The term *ATM* can indicate either bilateral inflammatory spinal cord disease or partial, unilateral, and noninflammatory disease. The cause of ATM is often obscure or uncertain (1). Previous studies have indicated that it is correlated with viral diseases (2), vaccinations (3), systemic lupus erythematosus (4), multiple sclerosis (MS) (5), vascular insults (6), spinal arteriovenous malformations (7), and acute disseminated encephalomyelitis (8). Its pathogenesis, clinical presentation, and course also vary greatly (9). The reported percentage of patients in whom ATM develops into MS varies from 0% to 80% (9–11).

The aims of this study were (*a*) to evaluate the contribution of magnetic resonance (MR) in determining the cause of ATM; (*b*) to determine the frequency and types of intracranial lesions detectable on MR at the onset of myelopathy; and (*c*) to monitor clinical and MR evolution of the disease.

Patients and Methods

ATM was defined as a spinal cord syndrome fully developed within 3 days after onset in patients with: acute, complete, or partial involvement of the spinal cord, with motor and sensory system dysfunction; no previous neurologic symptoms; and no previous evidence of spinal cord compression or lesions.

Between January 1987 and December 1992, 30 patients (20 women and 10 men) with symptoms compatible with ATM were admitted to the neurologic department of

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TABLE 1: ATM: number of patients with normal and abnormal spinal and brain MR findings

		Spinal MR		Total
		+	−	
Brain MR	+	6	5	**11 (36.6%)**
	−	8	11	**19 (63.3%)**
Total		**14 (46.6%)**	**16 (53.3%)**	**30 (100%)**

Note.—+ indicates abnormal; −, normal.

our hospital. The mean age was 40 years (range, 15 to 65 years). Eighteen patients had complete spinal cord syndromes with bilateral signs and symptoms, and 12 had partial or incomplete spinal cord syndromes. Clinically the spinal cord levels were cervical for 8 patients, thoracic for 19, and conus for 3.

Spinal MR studies were performed for all patients within 48 hours after the onset of symptoms. All patients had cranial MR within 10 days after the onset of the myelopathy. A 1.5-T system was used. The spinal cord was imaged with surface coils. Five- or 4-mm-thick multisection images were obtained in the sagittal plane, using the following sequences: T1-weighted spin echo, 600/17 (repetition time/echo time) or 500/15; T2-weighted spin echo, 2200/20–80; 2000/35–90; or 2400/25–90. For 4 patients, axial spin-echo T2-weighted images and for 1, axial gradient-echo (400/18, 15° flip angle) were also obtained. Gadopentetate dimeglumine (0.1 mmol/kg) was administered to 10 patients. Postgadolinium scans were obtained in the sagittal plane for all cases, and for one case axial sections were also obtained. Ten subjects underwent a second spinal MR about 3 weeks after the onset. One patient was also examined after 2, 7, and 36 months.

Brain MR images were obtained with a spin-echo multisection sequence (spin echo 2400/25–90), 5- or 6-mm sections in the axial plane, and 4-mm sections in the sagittal plane.

The cerebrospinal fluid (CSF) was examined within the first 7 days with electrophoresis to detect the oligoclonal bands.

Mean duration of the clinical follow-up was 18 months (range, 4 to 50 months). Steroids were given to all patients on admission and during hospitalization.

Results

The spinal cord MR findings were abnormal in 14 (46.6%) of 30 patients (Table 1). In these 14 patients, the clinical levels agreed with the spinal MR findings. In detail, 5 patients had cervical cord lesions and 7 thoracic (3 of them with conus extension), 1 had involvement only of the conus medullaris, and in one case the entire spinal cord was involved (Table 2). Patients were divided into three groups according to the presence and lengths of the lesions: group A had focal involvement of spinal cord (no more than one segment involved) (8 patients); group B, involvement of more than one segment of the spinal cord (6 patients); and group C, normal spinal MR (16 patients).

In group A, the MR displayed hyperintense lesions on T2-weighted images (Figs 1A–D and 2A–B). In two patients, the lesions were enhanced after the injection of gadopentetate dimeglumine. In these two patients, the lesions had different locations: C-2 with involvement of the right median part of the cord (Fig 1E); and T-6 (Fig 2C and D) with more evident enhancement in the middle and posterior part of the cord and involvement of the posterior, lateral columns, and gray matter. All enhancing and nonenhancing lesions were oval in configuration (Fig 3A and B). Four of eight patients had multiple disseminated hyperintense lesions on T2-weighted brain MR scans. These lesions were located in the periventricular and cerebral hemispheric white matter. At follow-up, all four of these patients now had clinically definite MS (12), showing additional discrete cerebral lesions. Only two of them had oligoclonal bands in the CSF. The remaining four patients, with normal brain MR findings, had remitting or stable clinical courses. Patient 1, with positive oligoclonal bands, normal brain MR findings, and a lesion at T-11/T-12 might have been classified as having laboratorysupported probable MS (13). However, because no clinical or radiologic evolution has been observed during 48 months of follow-up, he has been classified as having myelopathy of unknown cause (Table 2). On the other hand, none of our patients in whom clinically definite MS developed had both abnormal spinal and normal brain MR findings at presentation.

In group B, hyperintense signals on T2-weighted and gradient-echo images involving more than one segment of the spinal cord were observed (Fig 4A–C). Four of six patients also had slightly enlarged cords and two of six (patients 9 and 14) had enhancing lesions (Fig 4D). In both of these patients, the enhancement of lesions was inhomogeneous. In patient 14 (Fig 4C), the lesion on gradient-echo axial images was located in the central part of the cord involving the gray and nearby surrounding white matter. In patient 9, with a lesion from T-11 to conus, the exact location of the lesion was not determined because axial images were

TABLE 2: ATM: clinical data, MR findings, and final diagnoses

Patient/Age/Sex	Clinical Level at Onset	Spinal MR (hyperintense T2-weighted images)	Brain MR	Oligoclonal Bands	Clinical Evolution	Final Diagnosis
Group A (8 patients)						
1/19/F	T-11/T-12	T-11/T-12 GD(−)	−	+	R	M
2/65/F	Lumbosacral	Conus GD(−)	−	−	pR	M
3/49/F	Cervical	C-3/C-4 GD(ng)	+	−	pR	MS
4/58/F	High thoracic	T-2/T-3 GD(ng)	−	−	S	M
5/32/M	Cervical	C-4/C-5 GD(ng)	+	+	R	MS
6/36/F	Thoracic	T-6 GD(+)	−	−	R	M
7/21/M	Cervical	C-2 GD(+)	+	−	pR	MS
8/41/M	Cervical	C-2/C-3 GD(ng)	+	+	pR	MS
Group B (6 patients)						
9/57/M	T-10/T-11	T-11/conus GD(+), increased in size	−	−	pR	VM
10/25/F	T-8 to T-10	T-11/conus GD(ng)	−	−	R	VM
11/15/M	T-7	T-12/conus GD(ng), increase in size	−	−	pR	M
12/60/F	C-5	C-2 to C-7 GD(ng)	+	−	W	E
13/32/F	Cervical	Entire spinal cord, GD(ng), increase in size	+	−	E	M
14/24/F	Thoracic	T-6 to T-8 GD(+)	−	−	pR	M
Group C (16 patients)						
15/47/F	T-11/T-12	− GD(−)	−	−	R	M
16/33/F	Low cervical	− GD(ng)	−	−	pR	M
17/51/F	T-7/T-8	− GD(−)	+	+	W	MS
18/29/M	Lumbosacral	− GD(−)	−	−	R	M
19/25/F	T-12	− GD(ng)	+	+	R	MS
20/46/F	T-10/T-11	− GD(−)	−	−	R	M
21/21/M	T-10	− GD(ng)	+	+	pR	MS
22/56/F	T-5	− GD(ng)	−	−	pR	VM
23/23/M	L-2	− GD(ng)	+	+	R	MS
24/33/M	T-8	− GD(ng)	−	−	R	M
25/37/F	T-10	− GD(ng)	−	−	R	M
26/17/F	Cervical	− GD(ng)	−	−	E	M
27/38/F	T-11	− GD(ng)	−	−	pR	M
28/62/F	T-11	− GD(ng)	−	−	pR	M
29/58/F	T-5	− GD(ng)	+	−	pR	M
30/34/M	Thoracic	− GD(ng)	−	−	R	M

Note.— + indicates abnormal; −, normal; R, remission; pR, partial remission; W, worsening; E, exacerbation; M, acute transverse myelopathy of unknown cause; VM, viral myelitis; MS, multiple sclerosis; E, encephalomyelitis; GD(+), enhancing lesion; GD(−), no enhancement; and GD(ng), gadopentetate dimeglumine not given.

not obtained. No real differences in distribution of lesions in white or gray matter of patients with enhancing lesions (4 patients) were observed for the two A and B subgroups. In one case (patient 13), the spinal cord displayed an abnormal signal and there was slight enlargement from the cervicomedullary junction extending along the entire spinal cord, particularly evident at the thoracic segments (Fig 5A and B). In the follow-up MR after 7 months, this abnormality had partially improved (Fig 5C and D), and after 36 months, when tetraparesis had worsened, spinal MR showed persistence of only a slightly abnormal signal (Fig 5E). Cranial MR findings were abnormal and had not changed at follow-up. This patient remained clinically unclassified. Two of the remaining patients (cases 9 and 10) were diagnosed as having viral myelitis (high antibody titers for respiratory viruses in the sera and elevated cells and proteins in the CSF), and one (patient 12) as having acute disseminated encephalomyelitis (abnormal spinal and cranial MR, no other laboratory abnormalities, and no further evolution). Patients 11

Fig 1. Patient 7: multiple sclerosis.

A, Sagittal proton density–weighted image (2400/25).

B, Sagittal T2-weighted image (2400/90). Hyperintense oval lesion (*arrow*) at the C-2 level.

C, Axial proton density–weighted image (2400/25).

D, Axial T2-weighted image (2400/90). The lesion (*arrows*) is located in the right median part of the spinal cord, with involvement of the lateral and dorsal columns but also of the right gray matter.

E, Sagittal T1-weighted image (600/17) after gadopentetate dimeglumine injection. Oval homogeneous enhancing lesion. There is no cord enlargement.

and 14 had partially recovered: they were diagnosed as having myelopathy of unknown cause. The spinal MR of patient 14, performed after 12 months of follow-up, showed almost complete regression of the T-6/T-8 lesion (Fig 4E–G). Despite the higher percentage of positive CSF oligoclonal bands reported in large previous studies (14–16) in patients with acute myelopathy or early suspected MS, none of the six patients in group B had oligoclonal bands. At follow-up, MS had developed in none of them.

In group C, 5 of the 16 patients with normal spinal MR findings showed multiple lesions on brain MR. Four of them had additional neurologic symptoms involving other regions of the central nervous system. All these patients had positive cranial MR and oligoclonal bands at presentation and were classified as having clinically definite MS. In the remaining patient (case 29), additional discrete cerebral lesions developed that were judged to be ischemic.

The initial findings and follow-up enabled us to establish diagnoses for 12 patients. Final diagnoses and correlations with MR and CSF findings are illustrated in Table 3.

Discussion

In our study of ATM, we observed two spinal cord MR patterns. The pattern of group A was involvement of one segment of the spinal cord, no increase in size of the cord, and homogeneous enhancement. The pattern in group B was involvement of multiple segments, cord enlargement, and inhomogeneous enhancement. Spinal MR did not demonstrate any specific pattern for any of the final diagnostic categories, but with pattern B there seemed to be no evolution to MS.

ATM is a final diagnosis obtained after exclusion of several different disorders that can cause signal hyperintensity on T2-weighted sequences. In the patients (cases 9, 11, and 13) with involvement of more than one spinal cord segment, associated with cord enlargement at the level of the lesion, an intramedullary tumor could not be excluded (17). However, follow-up spinal MR examination about 3 weeks later demonstrated that the width of the spinal cord was normal, and the extension of the abnormal signal was reduced; these findings suggested

Fig 2. Patient 6: myelopathy of unknown cause.

A, Sagittal proton density–weighted image (2200/20).

B, Sagittal T2-weighted image (2200/90). Focal hyperintense lesion at the T-6 level (*arrow*).

C, Sagittal T1-weighted image after gadopentetate dimeglumine infusion (500/15). Homogeneous enhancement at the T-6 level (*arrow*).

D, Axial T1-weighted image after gadopentetate dimeglumine infusion (500/15). Enhancement is more evident in the middle and posterior part of the spinal cord. Involvement of posterolateral columns and gray matter (*arrows*).

that it was not tumor. Gadopentetate dimeglumine, which could have helped in diagnosis, was not available until after 1987. Granulomatous diseases that affect the cord, such as sarcoid or tuberculosis, may cause nodular enhancement. Differentiation of these conditions from a tumor can be difficult. The presence of a skip area separating multiple regions of enlarged cord is strongly suggestive of an inflammatory condition rather than a tumor. Enhancement may be diffuse, peripheral, or even punctate and speckled. These patterns are unusual for tumors (18). Dural arteriovenous fistulas may cause myelopathy, but acute symptoms were rarely present (7). The symptoms generally have a very insidious onset and subacute or chronic course and are marked by mild or moderate paraparesis or sensory disturbances. There are several possible patterns of presentation of this entity: nonspecific cord swelling, vague fuzziness or irregularity of cord margins caused by distention of small superficial vessels, definite demonstration of pathologically large vessels on noncontrast scans, or demonstration of abnormal vessels on postcontrast scans only (19, 20). Sometimes definitely abnormal vessels are not seen and myelography may be mandatory (21). Three of our patients with abnormal MR also underwent myelography, because they were middle-aged and their clinical syndromes initially seemed to progress despite steroid therapy. Myelographic findings for all these patients were normal. Abnormal high signals on T2-weighted images, edema, and cord swelling may also occur after surgery (because of obstruction of the extramedullary venous drainage system) or cord contusion (22, 23). Both events can easily be excluded by the clinical history.

Fig 3. Patient 5: multiple sclerosis.

A, Sagittal proton density–weighted image (2200/20).

B, Sagittal T2-weighted image (2200/80). C-4/C-5 oval hyperintense lesion (*arrow*).

Fig 4. Patient 14: myelopathy of unknown cause.

A, Sagittal proton density–weighted image (2200/20).

B, Sagittal T2-weighted image (2200/80). Hyperintense lesion at the T-6 to T-8 level (*arrows*).

C, Axial gradient-echo images (400/18, 15° flip angle). The lesion is in the central part of the cord with involvement of the gray and white matter. Both dorsal and lateral columns are involved.

D, Sagittal T1-weighted image (500/15) after gadopentetate dimeglumine infusion. Enhancement seemed to be more intense along the posterior border of the spinal cord (*arrows*).

E and *F,* Sagittal proton density–weighted and T2-weighted images (2200/20−80) 12 months after the acute episode. There is regression of the lesion and no definite hyperintensity at the T-6 to T-8 level.

G, Axial gradient-echo image (400/18, 15° flip angle). At the T-6 to T-8 level, cord atrophy is now present.

The causes of ATM vary greatly. Spinal cord ischemic lesions may occur in vasculitic and atherosclerotic diseases. The high and medium thoracic spinal cord regions are the main anatomic locations at higher risk of vascular insufficiency, because at these levels there are few major arterial suppliers and the anterior spinal axis is very thin.

Parainfectious events are frequently associated with ATM. Jeffery (24) found in a retrospective study of 33 cases of acute and subacute noncompressive myelopathy that 45% of these patients had parainfectious transverse myelitis. These latter patients had spinal cord swelling. We observed similar MR findings, but in a smaller percentage of patients. Three pa-

Fig 5. Patient 13: myelopathy of unknown cause.

A, Thoracic sagittal T2-weighted image (2000/90). Abnormal hyperintense signal at the onset of ATM.

B, Thoracic axial T2-weighted image (2000/90). Central involvement of both gray and white matter (mainly dorsal columns).

C, Cervical sagittal T2-weighted image (2400/90). Seven months after the beginning of the ATM, abnormal signal was present only from C-2 to C-5.

D, Thoracic sagittal T2-weighted image (2400/90). Seven months after the beginning of the ATM, no signal abnormalities are seen.

E, Cervical sagittal T2-weighted images (2600/90). After 36 months, despite worsening of tetraparesis, only a slightly hyperintense signal persisted at C-2 to C-5 levels.

tients, of whom one (case 10) had cord swelling, had final diagnoses of viral myelopathy. Viral myelopathy however may be related to reactive immune-mediated postviral inflammation, which should be included in the wide spectrum of acute disseminated encephalomyelitis (25).

From a clinical standpoint, it is very important to determine what specific radiologic findings can differentiate patients who will have MS from patients who will not. This would help prognosis and early inclusion in clinical trials. It appears that location at the C-2/C-3 level is much more common in MS than in other diagnostic categories. Our results are in accordance with those of Miller (26), who examined 40 MS patients and found lesions of the cord in the middle or upper cervical regions. Another MR marker for lesions in myelitis caused by MS seems to be an oval shape. Oval lesions extending longitudinally along the cord might be caused by the breakdown of the blood-brain barrier (23), with involvement also of vertical venous anastomoses (in the intrinsic venous system) (27) following the white matter tracts. This might lead to diffusion of the antimyelin antibodies and plaques could progress in elongated shapes (23, 28). As in the patients of other studies (24), none of our MS patients with spinal cord lesions had cord swelling. Our percentage of patients in whom MS developed (26.6%) is larger than that quoted in the literature (5, 29), and all except one patient presented with an incomplete spinal syndrome. The reported risk of getting MS, when ATM is complete, is low (ranging between 3% and 8%) but much higher (80%) when partial or asymmetric forms are considered (9).

TABLE 3: MR and CSF pattern at the onset of the myelopathy and final diagnoses

Final Diagnoses (no. of patients)	Spinal MR		Brain MR		OB	
	+	−	+	−	+	−
Multiple sclerosis (8)	4	4	8	0	6	2
Encephalomyelitis (1)	1	0	1	0	0	1
Viral myelitis (3)	2	1	1	2	0	3
Myelopathy of unknown cause (18)	7	11	1	17	1	17
Total	**14**	**16**	**11**	**19**	**7**	**23**

Note.—OB indicates oligoclonal bands; +, abnormal; and −, normal.

In a previous study (30), lesions within the brain in 36.3% of the patients with acute, clinically isolated spinal cord syndromes were demonstrated on MR. In our study we found a similar positive percentage. Previous studies have demonstrated the presence of oligoclonal bands in CSF of 90% to 100% of patients with clinically

TABLE 4: Myelitis caused by MS (8 patients): percentage of abnormalities

Spinal MR	50%
Oligoclonal bands	75%
Cranial MR	100%

definite MS (31). Oligoclonal bands were found in the CSF of 53.8% of the patients with clinically probable MS and in 7.1% of the patients with suspected MS (32). Therefore, the sensitivity of oligoclonal bands in the CSF for MS is high when the clinical diagnosis is definite. The specificity of oligoclonal bands for MS is not as high, because they can be detected in the CSF in many other neurologic diseases (eg, neurosyphilis, meningitis, meningoencephalomyelitis, and Lyme disease) (33). Ormerod et al (30) did not find oligoclonal bands in the CSF of any of nine patients with acute clinically isolated spinal cord syndromes, whereas we found CSF oligoclonal bands in a larger percentage of patients, especially MS patients. In conclusion, in ATM caused by MS one may see (*a*) brain lesions; (*b*) detectable oval spinal lesions, mainly cervical in location and without cord swelling; or (*c*) no detectable spinal lesions (Table 4).

Multiple factors can influence the positivity of MR scans in myelitis. Normal spinal MR findings can not completely rule out the presence of demyelinating lesions. In fact, clinical evolution of MS was also observed in some patients with no detectable lesions. Spinal cord lesions may be too small in some cases to be detectable by current MR systems. Also a breakdown of the blood-brain barrier and not demyelination may be the only abnormality in the early phase of a bout. Lipton and Teasdall (29) found cervical MR lesions in five of seven patients with acute isolated cervical spinal cord syndromes. MR did not demonstrate lesions within the cord in any of their patients with clinically localized lesions of the thoracic cord. These latter results seem to confirm partially our relatively low positive percentage (36.8%) of spinal MR scans in patients with clinically isolated thoracic cord syndrome as compared with the higher percentage (71.4%) of positive spinal scans for the group of patients with cervical cord syndromes. Furthermore, in some cases of ATM caused by systemic lupus erythematosus, spinal MR findings may be normal (34). Therefore it is important to use more sensitive and more rapid methods for demonstrating spinal cord lesions. The application of fast spin-echo sequences with improved spatial resolution, using a 512×512 matrix and thinner sections, will probably increase the detection of small cord lesions. Long-repetition-time images, especially of the cord, are often time consuming and marred by motion artifacts. When fast spin-echo sequences are combined with phased-array coils, a long-repetition-time scan of the entire cord can be performed rapidly. The advantages of the phased-array coils are a large field of view and increased signal-to-noise ratios. The increase in the field of view coupled with an increase in the matrix size to 512×512 improves resolution. All these technical factors will probably contribute to increasing the percentage of positive spinal MR scans in ATM.

Gadopentetate dimeglumine administration is now widely used, and it will probably improve the percentage of positive cases. However, in our study spinal MR findings after gadolinium administration remained normal for patients with normal baseline scans. Subtle blood-brain-barrier leakage may be missed on immediate postcontrast MR images but becomes more evident on delayed images in MS myelitis (35). All patients with enhancing lesions had good outcomes. The rapidity of resolution of the enhancement, observed on follow-up MR scan, could be a helpful prognostic indicator (36), and because of this the use of gadolinium is recommended for diagnosis and monitoring of treatment in patients with relapsing-remitting or secondary progressive MS. It increases the yield of active inflammatory lesions and improves their clarity (37).

In summary, we have found that spinal MR contributed to establishing the cause of ATM in 40% of our cases. In addition, this study demonstrated two different spinal lesion patterns. Another important finding is that the majority of brain lesions present at the onset of myelopathy correspond to demyelinating lesions. The percentage of negative spinal MR findings in ATM is still high (53.3%), particularly in MS patients (50%).

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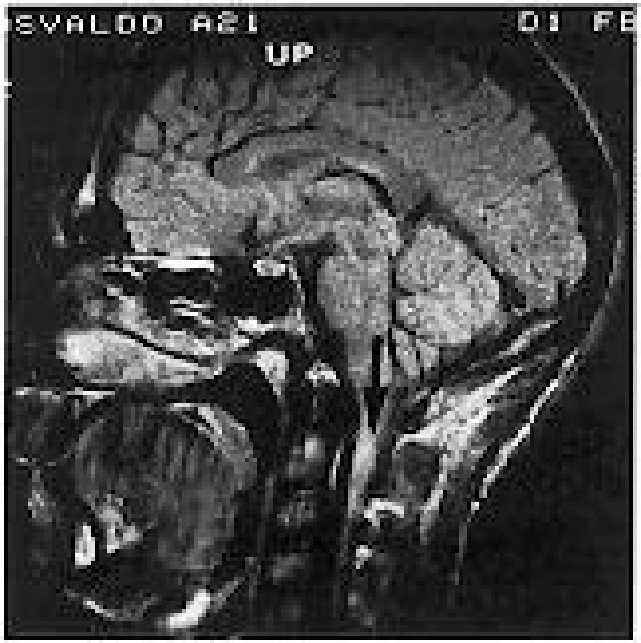

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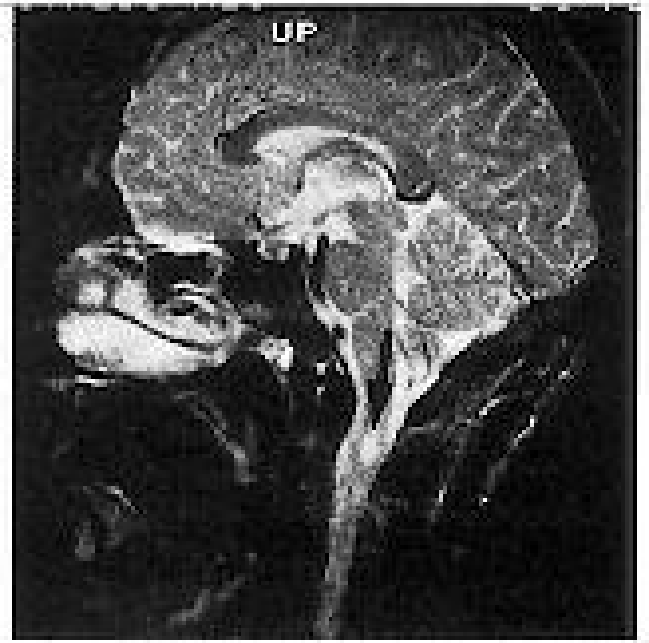

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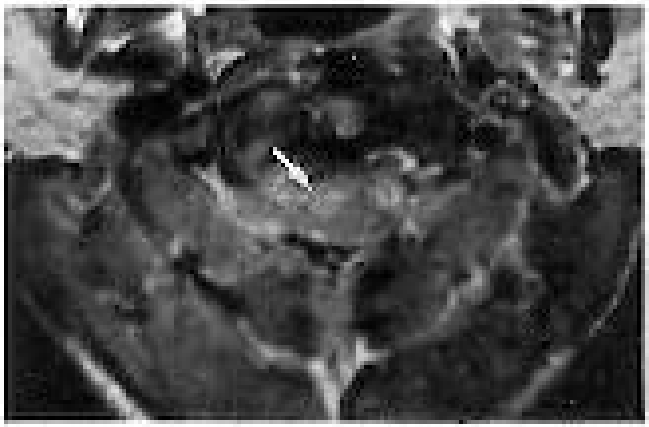

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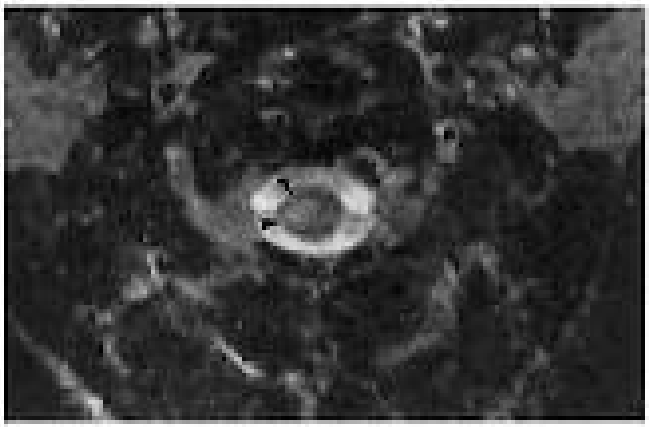

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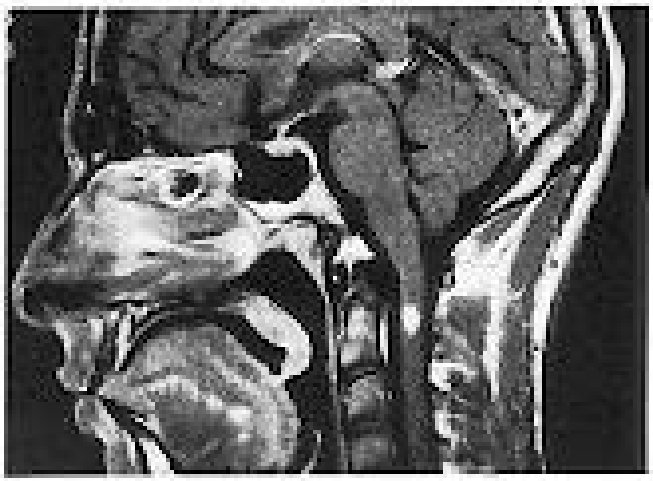

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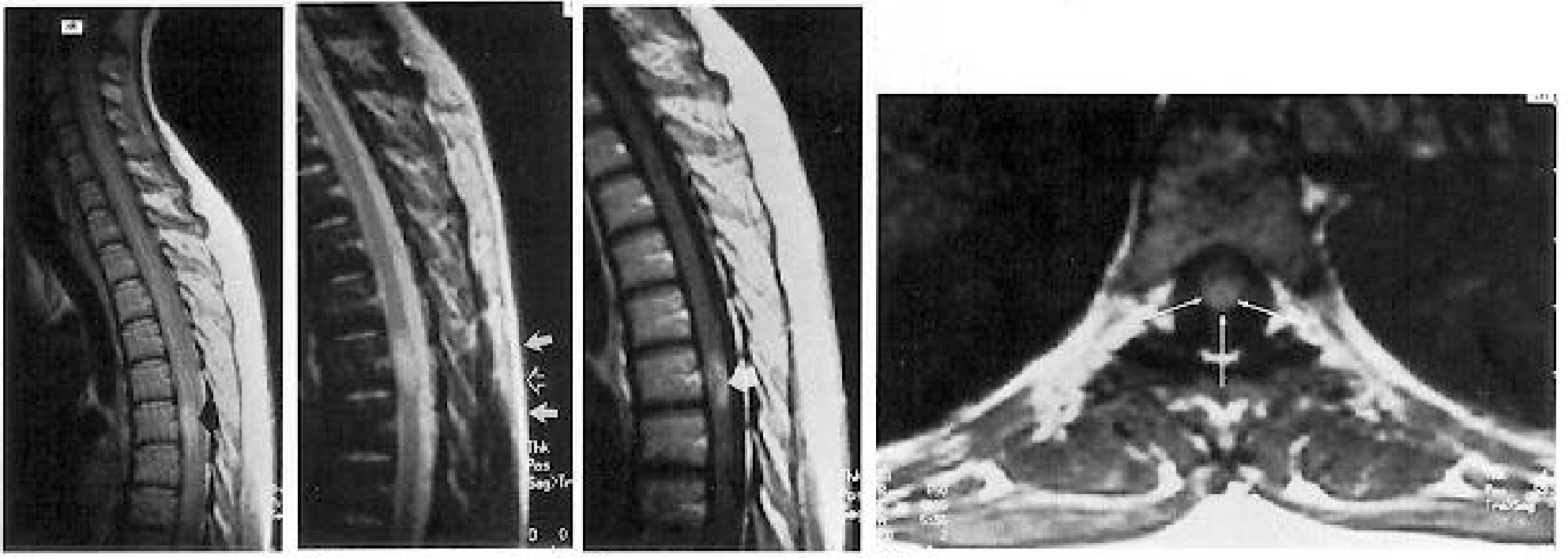
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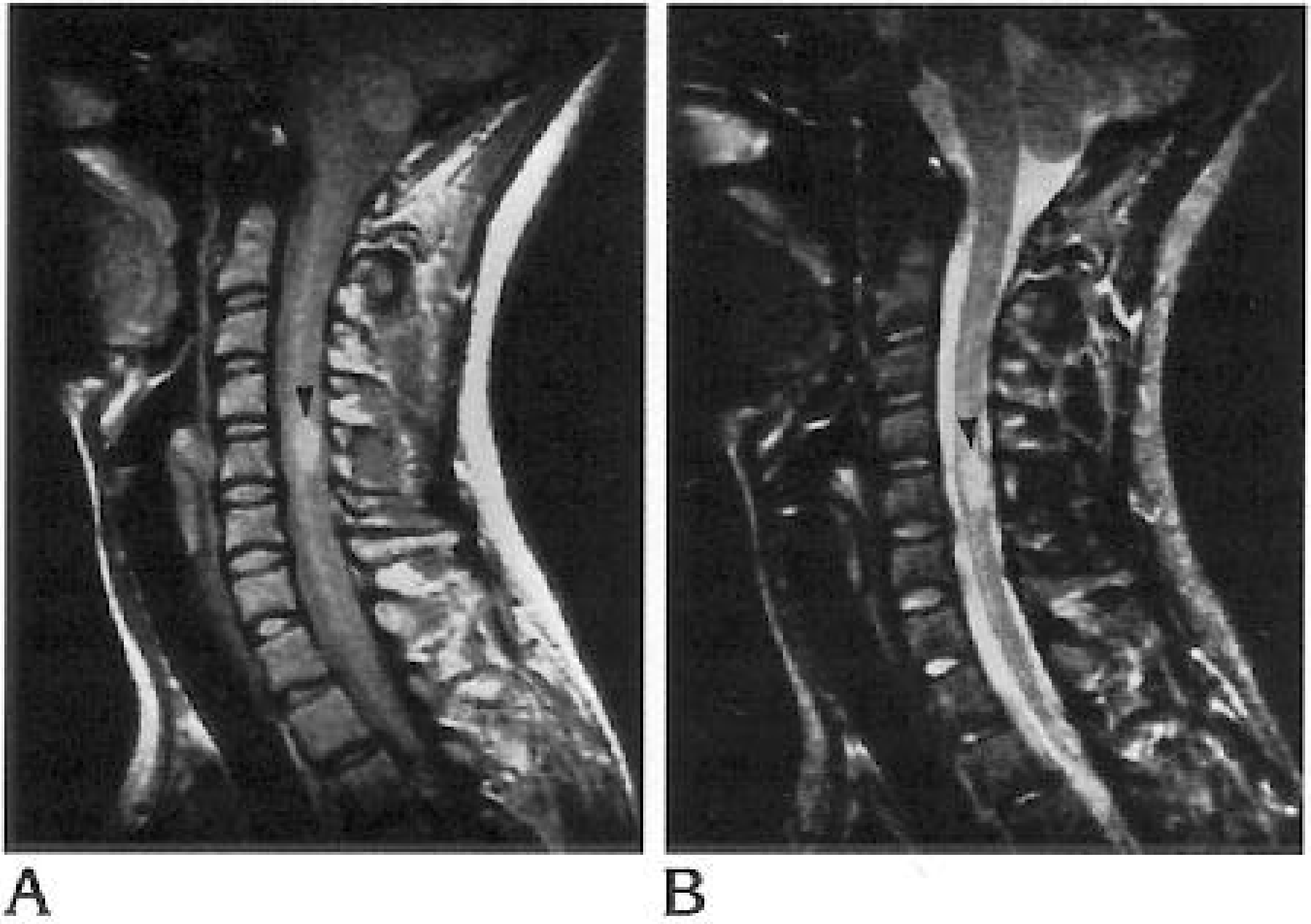
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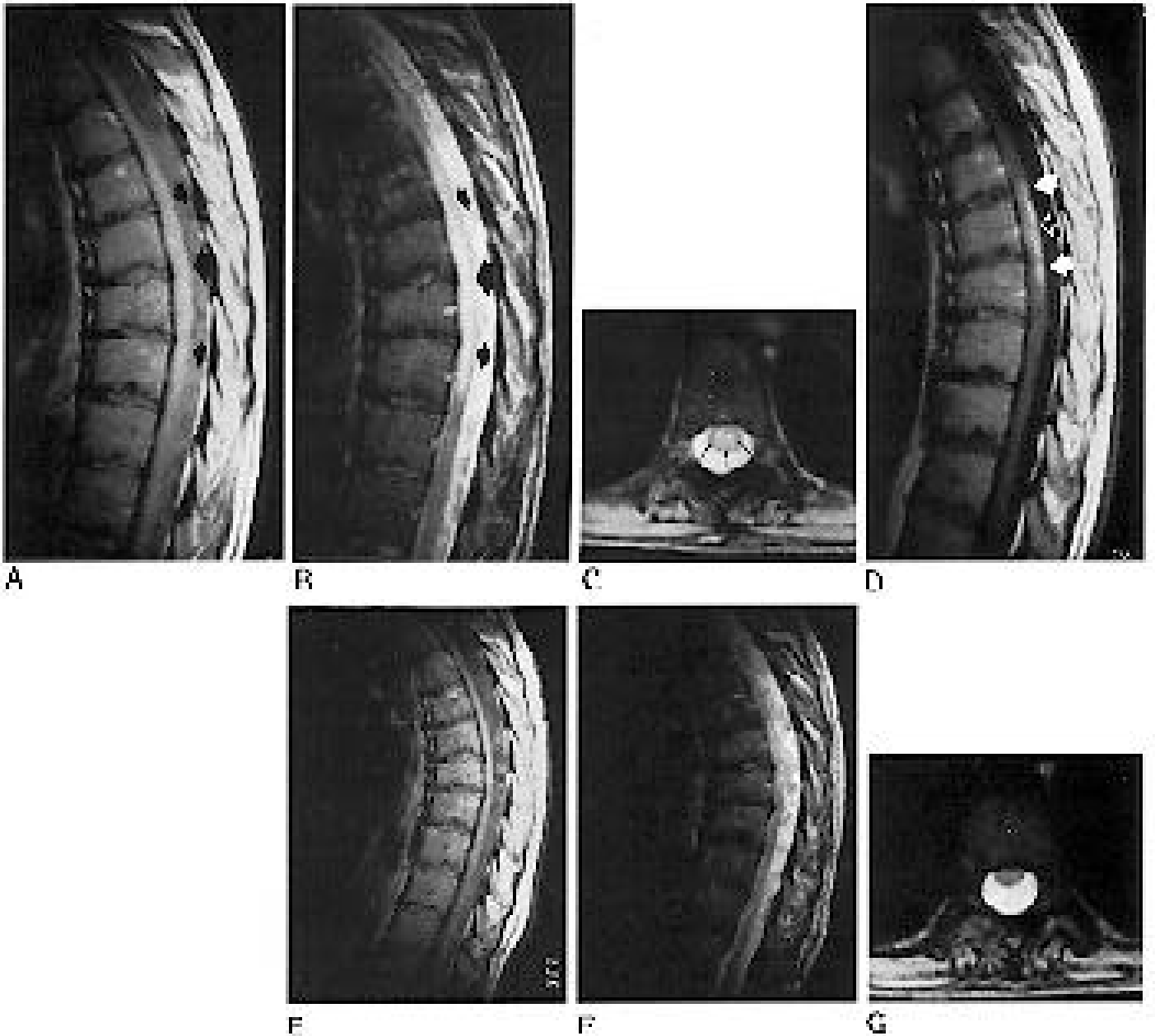
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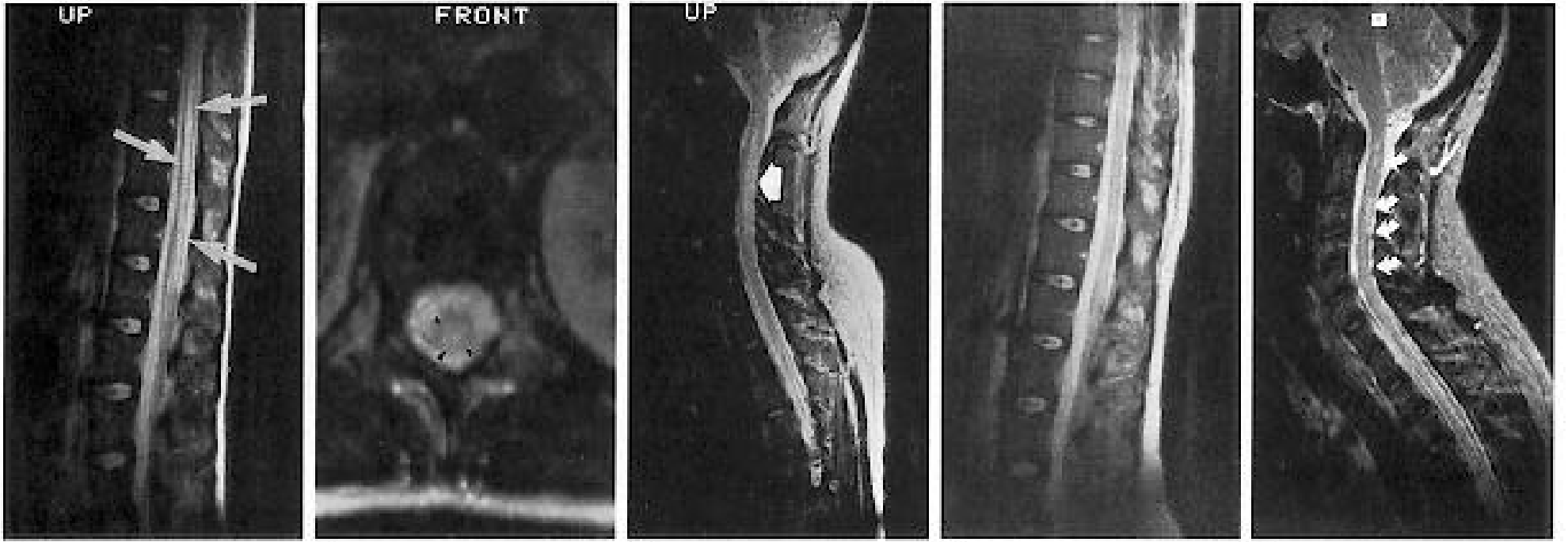
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