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MR of Omental Myelosynangiosis

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PURPOSE: To describe MR findings in patients who have undergone omental transposition (omental myelosynangiosis) for spinal cord revascularization. **METHODS:** Spin-echo MR images, without and with intravenous gadolinium, were obtained before and after surgery in three patients using a quadrature spine coil. Three-dimensional time-of-flight spinal MR angiography was also performed. **RESULTS:** On routine MR, the transposed omentum is an irregular, lobulated fat-equivalent mass, containing serpiginous areas of flow void, which extends through the laminectomy site to lie directly adjacent to the cord surface. MR angiography demonstrated small omental vessels, some coursing to the omentum-cord interface; however, no definite extension into the cord was detected. In all patients, there was alteration in cord size and contour after transposition, but no change in cord signal. Clinical improvement was observed in one of the three patients. The signal characteristics of the transposed omentum changed, showing less homogeneity and a gradual loss of the signal over a period of 4 months. **CONCLUSIONS:** MR delineates transposed omentum and associated postoperative changes in omental myelosynangiosis. MR angiography is useful as an adjunct to demonstrate the small vessels near the omentum-cord interface, but lacks sufficient resolution to demonstrate neoangiogenesis within the cord.

Index terms: Spinal cord, magnetic resonance; Spinal cord, surgery

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Omental myelosynangiosis is a surgical technique in which a pedicled omental graft is laid on the surface of the spinal cord for the purpose of augmenting vascularization of cord tissue (1). This procedure takes advantage of the extraordinary neovascularization capability of the omentum in order to revascularize the cord by direct extension. It has added a new possibility for the surgical approach to chronic spinal cord ischemia. There have been a few reports in the neurosurgical literature describing this technique (1–3). The magnetic resonance (MR) findings in three patients who underwent omental myelosynangiosis are presented in this paper.

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Subjects and Materials

Three men, ages 22, 39 and 46 years, with suspected chronic ischemic cord insult underwent omental myelosynangiosis 1 to 3 years after the onset of myelopathy (Table). Institutional permission for this surgery was secured on a case-by-case basis.

Before and after surgery, clinical evaluation along with MR imaging on a 1.5-T scanner equipped with a quadrature, receive-only spine coil was done. T1-weighted sagittal and axial spin-echo images (600/20/2 [repetition time/echo time/excitations]) were acquired with section thickness of 3.5 to 5.0 mm, no intersection gap, and field of view 18 to 25 cm. Axial gradient-echo (1000/18/2, 20° flip angle) and sagittal gradient-echo or dual spin-echo (2000/20, 80/2) scans were also acquired using similar section thickness and field of view as for the T1-weighted images. The T1-weighted images were obtained before and after intravenous injection of 0.1 mmol/kg of gadopentetate dimeglumine.

Preoperative and postoperative three-dimensional time-of-flight MR angiography was performed before and after intravenous gadolinium administration. The sequence parameters were 50/10, 20° flip angle, 36 to 46 sections, each 0.7 to 0.8 mm thick, 18 to 20 cm field of view, image matrix 256 × 256, with coronal or sagittal slab orientation. Selective presaturation was used to limit artifacts caused by respiratory motion.

Omental myelosynangiosis (OMSA): preoperative clinical and MR findings

Patient (age, y/sex)	Cause	Time to OMSA	Level of Surgery	Preoperative Cord Appearance	Time from OMSA to Follow-up MR	Postoperative Cord Appearance	Follow-up Neurologic Status
1 (39/M)	Ischemic myelopathy	2 years	C-2 to C-7	Atrophy	6 months	No change in signal	Improvement in grip strength
				Isointense (T1), linear hyperintensity (T2); posterior position of cord at C-1/C-2	10 months	Omentum apposed to cord with indistinct myeloomental interfaces	
2 (46/M)	Progressive paraparesis 2° to a combination of cystic/ noncystic myelopathy, ischemia, and cord tethering	6 years	T-6 to T-11	Cord enlarged; hypointense (T1), hyperintense (T2)	2 months	Decrease in cord size; omentum apposed to cord with distinct myeloomental interface; concave dorsal cord surface	No further progression of neurologic deficits
3 (22/M)	Postoperative ischemic anterior cord syndrome	3 years	T-9 to T-11	Normal	3 months	Omentum apposed to cord with distinct myeloomental interface	No further progression of neurologic deficits

The surgery involved extensive laminectomy, opening of the dura, and release of adhesions, which were present between the cord and duraarachnoid. The omental pedicle graft was funneled through the skin of abdomen, chest, and neck (for cervical omental myelosynangiosis) and brought around to be transposed onto the exposed posterior surface of the spinal cord. The graft was then secured to the margins of the dural incision.

Patients underwent interval follow-up neurologic assessment and MR imaging of the area operated on, as described above.

Results

Clinical information and MR findings before and after surgery are summarized in the Table. Two of three patients were suspected of having chronic ischemia of the cord based on the clinical evaluation. In patient 1, the cause of ischemia was considered degenerative canal stenosis, secondary to previous surgery at the craniocervical junction. The cervical segment of the anterior spinal artery was not seen angiographically. In patient 3, ischemia was attributed to aortic clamping, done at the time of repair of a traumatic aortic dissection. Chronic myelitis was diagnosed in patient 2, and concern regarding possible secondary ischemia was the impetus for undertaking the surgical procedure. The myelopathy was progressive in the first two patients but had stabilized by the time of surgery in patient 3. Two of the three patients had abnormal focal signal in the cord on preoperative images (Fig 1B).

Technically, the surgical procedure in each patient was successful and the postoperative period uneventful. Subsequent neurologic examinations revealed improvement from the preoperative state in patient 1 and a halt to the progressive neurologic deterioration in patient 2. In patient 3 there was no change in the neurologic status. MR imaging demonstrated laminectomy defects and postoperative changes (Table and Figs 1 and 2). An irregularly marginated, lobulated mass, having signal characteristics of fat, and containing serpiginous areas of signal void, was identified within the surgical bed. This mass, representing omental tissue, was apposed to the posterior cord surface. In patient 1, the cord was retracted posteriorly into the laminectomy defect and had lost the normal

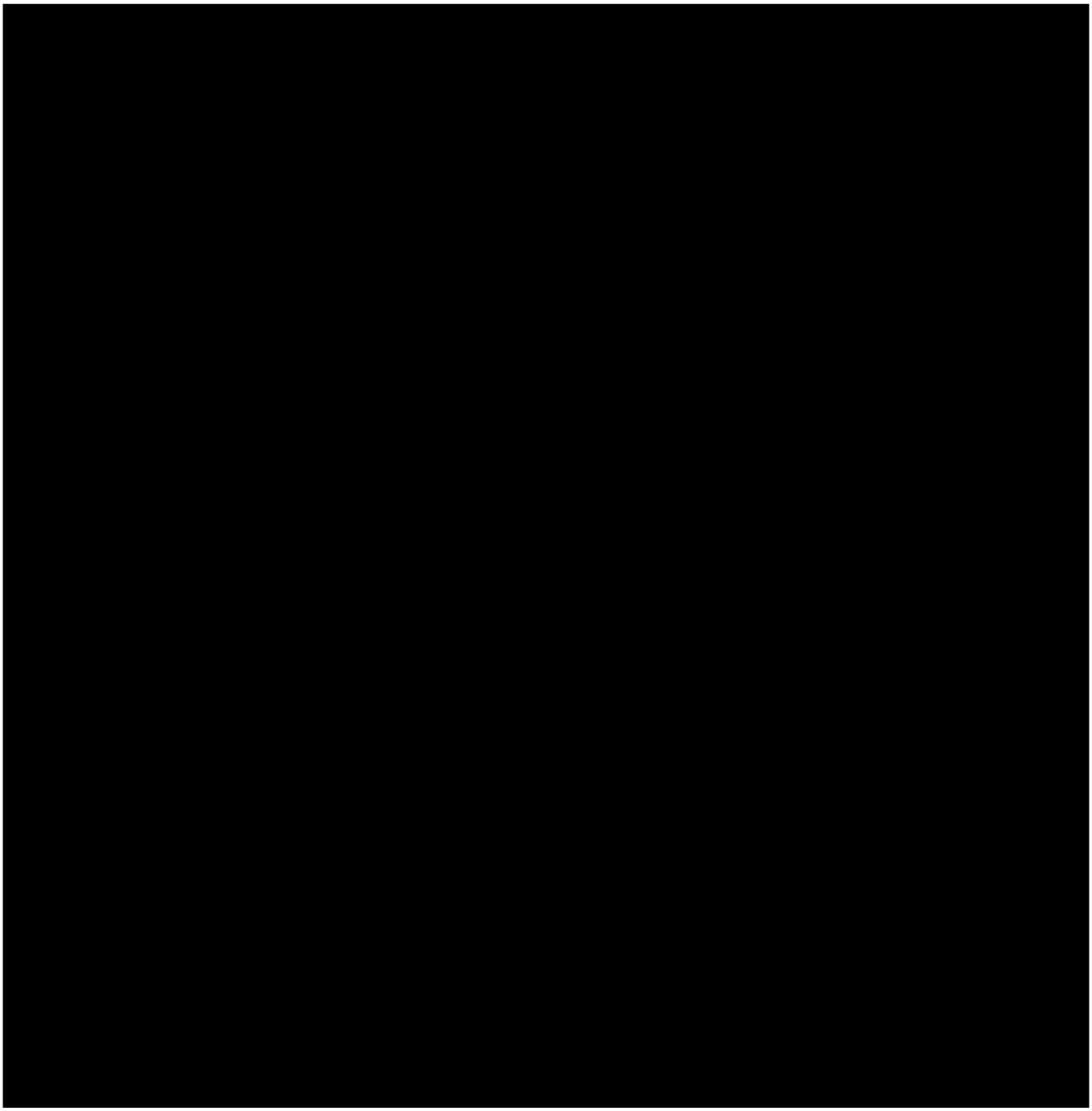

Fig 1. Omental transposition to the cervical cord (patient 1). Preoperative (*A* and *B*) and postoperative (*C–F*) images.

A and *B*, The sagittal T1-weighted image (*A*) shows posterior displacement of the cervical cord at C-1/C-2. The posterior arch of C-1 and part of the occipital bone were previously resected (*double arrowheads*). The T2-weighted image (*B*) shows focal, hyperintense signal in the postcentral region of the cord at C-3/C-4 (*arrow*). The spinal canal from C-3/C-4 to C-5/C-6 is stenotic.

C–E, On the sagittal T1-weighted image (*C*) and corresponding gradient-echo image (*D*) the cervical cord is displaced posteriorly from C-3 to C-7 compared with its position in *A* and *B*. The posterior margin of the cord is contiguous with the omental graft (between *large arrows*), which has signal characteristics of subcutaneous fat, yet is more heterogeneous. Serpiginous areas of decreased signal (flow void) in *C* and increased or decreased signal in *D* represent omental vessels (*small arrows*). Persistent hyperintense signal (*arrow*) within the cord is shown in *D* (compare with *B*). The T1-weighted image (*E*) obtained 4 months after *C* demonstrates loss of fat signal in several regions (*arrows*) of the omental graft. Adhesions (*single arrowheads*) between the cord and anterior dura are best demonstrated in *C* and *E*.

F, Axial T1-weighted image at C-4/C-5 shows the posterior displacement and change in contour of the cord after omental myelosynangiosis. Omental tissue is contiguous with the posterior surface of the cord, and the graft pedicle (*arrows*) extends from the patient's right side.

Fig 2. Omental transposition to thoracic cord (patient 2). Preoperative (*A*) and postoperative (*B* and *C*) sagittal images, and postoperative (*D*) 3-D time of flight MR angiography image.

A, On the T1-weighted image, there is evidence of previous laminectomies and surgery for shunt placement from T-6 to T-11, with abnormal signal within the cord (*superior to arrow*).

T1-weighted (*B*) and gradient-echo (*C*) images, corresponding to A, demonstrate the omental graft (*between large arrows*) with signal characteristics of fat and vascular structures (*single arrowhead*). A small vessel (*double arrowheads*) at the level of T-7 courses toward the omentum-cord interface.

D, Targeted, sagittal reprojection (maximum intensity projection) angiogram shows the continuation of the vessel (*double arrowheads*) identified in B to the omentum-cord interface at T-7/T-8.

convex contour of the anterior cord surface (Figs 1C and F). In patient 2, the posterior surface of the cord also had lost the normal convex contour. No other significant abnormality was detected. Enhancement of the transplanted omentum was not identified; however, the vessels within the transplant were seen better on MR angiography after gadolinium than before gadolinium (Fig 2D). MR angiography showed small omental vessels, which coursed to the omentum-cord interface, but the extension of these vessels into the cord substance could not be demonstrated.

Discussion

In animal experiments, Goldsmith (2) demonstrated that transposition of an intact, pedicled omentum to the brain surface resulted in neoangiogenesis at the omentum-cerebrum interface. Vessel growth was attributed to presence of lipid angiogenic factors and neurotransmitters in the omentum. These experiments led to the hypothesis that increased blood flow to ischemic neural tissue might have beneficial effects for residual viable tissue with decreased neuroelectric activity. Reports of the use of omental transposition to treat chronic ischemia of the cord after trauma (1, 2, 5) or to improve blood flow to ischemic brain tissue (2, 4, 5) subsequently appeared in the literature.

At present, rehabilitation is the commonly used method to achieve maximum neuromuscular function after spinal cord injury. Neuroradiologic studies of patients with chronic paraplegia after trauma have been few. In a study of 125 patients, Perovitch et al (6) found that the main causes of the histopathologic state of the posttraumatic spinal cord are intramedullary and leptomeningeal vascular alterations. In view of these results, omental myelosynangiosis may benefit patients with reversible ischemia by improving vascularization.

The technique in general involves elongating the omentum and developing a subcutaneous tunnel up to the spinal area of interest. The duraarachnoid is opened, and the omentum is placed directly on the spinal cord. Goldsmith

(2) demonstrated that omental placement on a normal cord led to development of new vessels at the omentum-cord interface within 72 hours; neovascularization of an injured cord occurred even more rapidly. In addition to angiogenic capability, the omentum has great capacity to absorb vasogenic edema. This is believed to be the basis for preserved somatosensory evoked potentials and motor function in animals after spinal cord injury, as long as the omentum is applied to the cord within 3 hours of injury.

Because omental myelosynangiosis is a relatively new technique, the criteria for selection of patients who will most likely benefit from this procedure have not yet been established. There are reports of clinical improvement after omental myelosynangiosis in patients with traumatic paraplegia complicated by recurrent arachnoiditis and syringomyelia (1, 5), and in a patient with healed tuberculous arachnoiditis and syringomyelia (7). Goldsmith (2) reported encouraging results in five quadriplegic and two paraplegic patients who underwent omental myelosynangiosis 2 to 15 years after cord injury. Recovery of function after surgery was variable and related to the extent of preoperative cord damage and postoperative axonal regeneration (5).

The imaging findings in cases of omental myelosynangiosis are characteristic. The omentum is easily recognized because of its fat signal and the interspersed foci of flow void caused by omental vasculature. Areas within the surgical bed having characteristics of edema, hemorrhage or fibrosis could be observed depending on the timing of the postoperative MR study (Figs 1C–E). Where the omentum abuts the cord, there is loss of the normally convex contour of the cord. Omental vessels extending to the cord interface are demonstrated on spin-echo and gradient-echo images, especially postgadolinium 3-D time of flight MR angiography source images. However, neoangiogenesis, which predominantly represents capillary ingrowth (2), could not be demonstrated. Improving the resolution of MR angiography images (eg, with a 512 $\times$ 512 matrix) or using intraarterial digital subtraction angiography may allow demonstration of larger vessels associated with neoangiogenesis.

As omental myelosynangiosis becomes an established procedure, further neuroimaging studies will be needed to identify those findings that correlate with the clinical diagnosis of reversible cord ischemia and to elucidate any abnormalities in regional spinal vascular anatomy before surgery. Recognition of neoangiogenesis after surgery may require correlation of special MR techniques, such as fat suppression imaging and MR angiography, with conventional spinal angiography.

In summary, the signal characteristics of transposed omentum and its relationship to the spinal cord are well demonstrated on routine spin-echo and gradient-echo MR images. The radiologist should be aware of these findings to recognize the MR appearance of omental myelosynangiosis.

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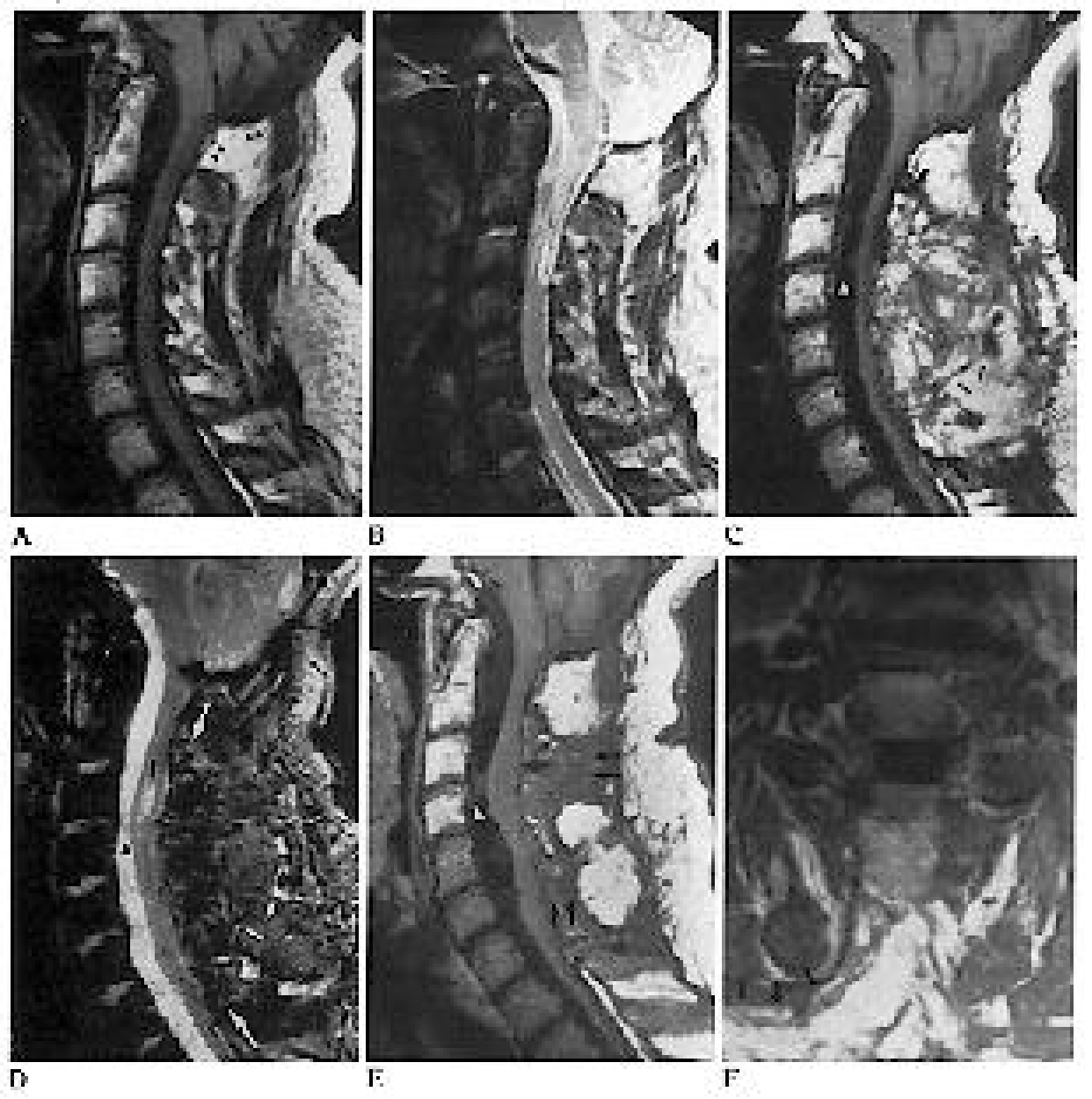
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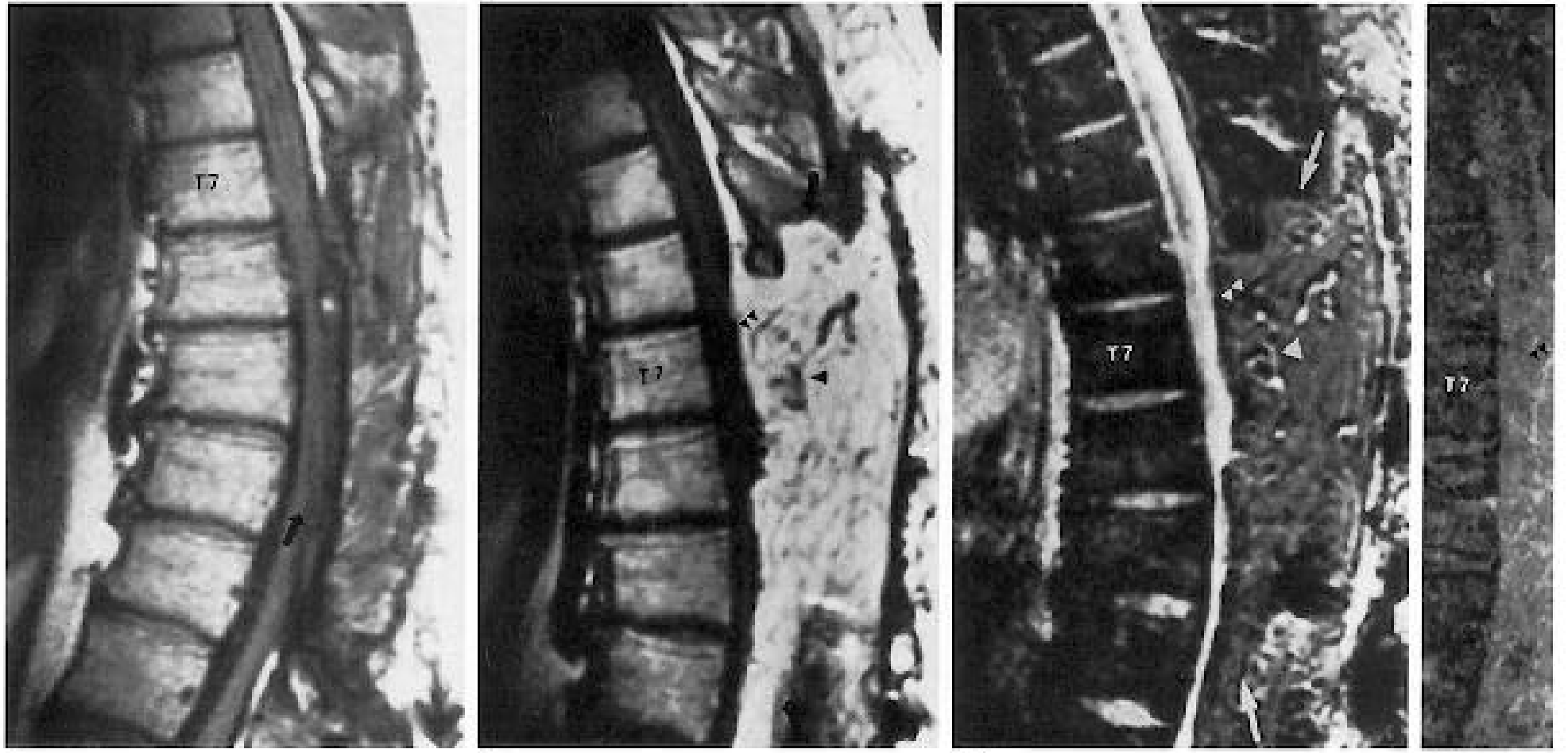
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