

Transient Expressive (Nonfluent) Dysphasia after Metrizamide Myelography

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Four (3.4%) of 117 patients undergoing metrizamide myelography experienced transient expressive dysphasia 7–8 hr after myelography and lasting up to 36 hr. All four patients had lumbar myelograms obtained with 15 ml of 190 mg I/ml (2850 mg I). Metrizamide was injected via lumbar puncture with a 20 gauge spinal needle under fluoroscopic control. Neurologic complications after metrizamide have been reported, but so far have appeared to be transient. It is likely that the transient expressive dysphasia experienced by the four patients reported here was a neurotoxic reaction, rather than a seizure phenomenon.

Although Dimer-X marked the advent of nonionic, water-soluble myelographic contrast media, complications from it forced its removal from clinical use. Shortly thereafter, metrizamide was introduced and has remained the only such medium available for routine clinical use in this country to date. All the advantages of a water-soluble contrast medium with relatively few transient side effects were attributed to it. However, as with all medications put to widespread use, its advantages and disadvantages soon became known. This article retrospectively reports transient expressive (nonfluent) dysphasia in four (3.4%) of 117 patients who underwent lumbar (102), thoracic (four), and cervical (11) metrizamide myelography performed by the author since November 1979.

Materials and Methods

All myelograms (lumbar, thoracic, or cervical) were obtained with metrizamide administered in appropriate concentrations. All patients were given clear liquid breakfasts, followed by pushed fluids by mouth and an intravenous infusion of dextrose 5% in one-half normal saline at 75–125 ml/hr. All patients either were already taking Valium or were given Valium 5 or 10 mg three times in the 24 hr before myelography. No patient was taking phenothiazines.

A 20 gauge spinal needle was used for lumbar puncture. Contrast material was injected under fluoroscopic control to T9–T10 with the patient in the prone position. For lumbar myelography the maximum dose was 15 ml of 190 mg I/ml (2850 mg I). For thoracic and cervical myelography the only dose was 10 ml of 250 mg I/ml (2500 mg I).

All myelograms were obtained via lumbar puncture, except one lumbar myelogram in a patient with a complete block at the L3 level. Because there was question as to the subarachnoid location of the contrast material, a C1–C2 puncture was done, confirming the block and subarachnoid location of the contrast material.

After myelography, all patients were in bed with the head elevated 30° for 8 hr before it was turned down flat. The intravenous infusion was continued until at least 8 A.M. of the first day after myelography, longer if the patient was unable to consume fluids. The patient was kept at bedrest for 8 hr and then allowed bathroom privileges only. No phenothiazine derivatives were administered for nausea or vomiting. No treatment was administered to patients with dysphasia.

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Case Reports

Case 1

A 56-year-old right-handed woman had undergone iophendylate myelography and cervical laminectomy 2 years before for cervical spondylosis. She now underwent lumbar metrizamide myelography for low back and bilateral leg pain with a presumptive diagnosis of lumbar stenosis, which proved to be present at L4 and L5. Neurologic examination was normal except for L5 and S1 root findings.

In the evening, 8 hr later, she developed expressive (nonfluent) dysphasia of moderate degree that cleared within 24 hr. In addition, she was aware of hallucinations that lasted 24 hr. The upper level of contrast material seen during myelography was at L1–L2.

Case 2

A 61-year-old right-handed man had had a lumbar iophendylate myelogram for a herniated lumbar disk followed by surgery 3 years before. Because of recurrent lumbar symptoms, he underwent metrizamide lumbar myelography, which showed bilateral stenosis at L4–L5, with the upper level of contrast material at L1–L2. Eight hr after myelography, the patient developed moderate expressive dysphasia, and he was aware of having hallucinations. Both cleared totally within 24 hr. His neurologic examination before myelography showed only right L5 root findings.

Case 3

A 36-year-old right-handed woman had low back and right leg pain for 1½ years after a motor vehicle accident. She had clinical evidence of an L4–L5 disk herniation on the right side only and underwent lumbar metrizamide myelography, which was normal. The upper level of contrast material was at T11–T12.

Eight hr after myelography she developed very marked expressive dysphasia that lasted almost 36 hr before clearing completely.

Case 4

A 40-year-old right-handed woman underwent lumbar metrizamide myelography for low back and right leg pain. Before myelography, her only indications were L5 and S1 root findings. About 7 hr after myelography she had a mild expressive dysphasia that she told the physician about only 18 hr later, at which time it had cleared fully. Myelography showed an L3–S1 stenosis with midline protrusions at L3–L4 and L4–L5. The upper level of contrast material was at L2–L3.

Discussion

Metrizamide is a water-soluble, nonionic, myelographic contrast medium. Its advantages include water solubility, which allows it to opacify the subarachnoid space well, affording more accurate diagnosis. It need not be removed because it is excreted over 24–48 hr. No permanent complications, such as arachnoiditis, have been reported [1].

The four patients reported here experienced mild to moderately severe expressive (nonfluent) dysphasia with metrizamide. All reactions occurred about 7–8 hr after myelography and lasted up to 36 hr. All the patients who had this complication had lumbar myelograms with a maximum of 2850 mg I being injected into the lumbar subarachnoid space. Eleven

patients had cervical myelograms and four had thoracic myelograms, all by lumbar puncture, without this complication. One of the cervical myelogram patients had a nonfocal grand mal seizure 8 hr after injection of contrast material while undergoing CT of the cervical spine. Only one patient had a lateral C1–C2 puncture for a total block at L3, which was in doubt after a lumbar puncture had been used for myelography.

A recent review of 100 consecutive myelograms looking specifically for neurologic complications of metrizamide demonstrated a 1% complication rate [2]. Another article reported two patients with arteriovenous malformations of the spinal cord who developed transient expressive dysphasia. The authors attributed this to a damaged brain in prolonged contact with metrizamide [3]. A third article reported a patient with confusion, dysphasia, and asterixis [4].

In this article, case 3 had a normal lumbar myelogram. Case 2 had a previous lumbar iophendylate myelogram obtained by the author for a lumbar disk and had undergone lumbar laminectomy. Cases 1, 2, and 4 had positive myelograms for lumbar stenosis. Case 1 had a previous iophendylate cervical myelogram for cervical spondylosis followed by a cervical laminectomy. Both patients with previous iophendylate myelograms had no evidence of arachnoiditis. Spinal fluid proteins were normal in all patients.

Three patients were female; one was male. All were right-handed. Only expressive dysphasia was found on neurologic examination, other than premyelographic radicular findings for which the myelograms were obtained. On examination, all four patients were unable to speak the appropriate words. When this episode cleared, all stated that they knew what they had wanted to say, but could not say it.

All episodes occurred in the evening 7–8 hr after myelography (about 9 P.M.). The time is probably because of hospital scheduling and availability of fluoroscopy. The 7–8 hr period appears to be constant in this series, and applied also to the one patient who had a nonfocal grand mal seizure 8 hr after injection of contrast material.

In one series of 100 patients, side effects usually became apparent in 4–6 hr, peaked at 24 hr, and disappeared by 48 hr. Lumbar myelographic patients had a higher incidence than cervical patients, and women were more affected than men [2]. In a report of two patients, motor aphasia began 16 hr after lumbar injection of contrast material in one and during the night in a second [3]. In a series of 67 patients with pluridirectional myelography and cisternography, eight patients who were placed in the left lateral decubitus position developed dysphasia 1–10 hr after injection of contrast material. Six patients developed dysphasia in 1–5 hr, one in 6 hr, and one in 6–10 hr. All symptoms disappeared by 48 hr and appeared to be prevented by using the right lateral decubitus position. Ten authors with 20 patients reported latency at 0–24 hr with symptoms lasting 1–3 days [5].

There was no clinical or familial evidence of focal sensory seizures in the four patients described here. After the dysphasia cleared, all patients noted that they knew what they wanted to say but could not say it. No electroencephalograms (EEGs) were obtained in any of these patients, although there

are reports of patients who had EEGs before and after metrizamide [4]. Although EEG abnormalities have been reported in 8%–16%, a series by Kaada [6] reported 10% pre- and 15% postmetrizamide abnormalities. Kaada's interpretation was that metrizamide aggravated the preexisting abnormality [6].

Certainly an abnormal EEG is not diagnostic of a seizure disorder. Clinically, none of the patients reported here had evidence of a seizure disorder. Seizure after metrizamide is thought to be a toxic reaction. In a study of dogs using autoradiography, metrizamide concentrated along a gradient in the superficial layers of the cerebral cortex [7]. Thus, it would appear that the dysphasia is a focal neurotoxic reaction, rather than a seizure phenomenon. In a recent publication metrizamide was found to be both anticholinergic and an anticholinesterase in *in vitro* studies. This could well explain its toxicity [8].

Thus, while metrizamide is an excellent agent for opacification, it does have worrisome complications, which fortunately have been transient to date.

REFERENCES

1. Heinz ER. Techniques in imaging of the spine. Part 3: myelography. In: Rosenberg RN, ed. *The clinical neurosciences*, vol 4. New York: Churchill Livingstone, **1984**:795–797
2. Hauge O, Falkenberg H. Neuropsychologic reactions and other side effects after metrizamide myelography. *AJNR* **1982**;3:357–360, *AJR* **1982**;139:357–360
3. Boker DK, Sartor K, Winkler D. Motor aphasia after cervical myelography with metrizamide. *ROFO* **1980**;133:204–207
4. Smith MS, Laguna JF. Confusion, dysphasia, and asterixis following metrizamide myelography. *Can J Neurol Sci* **1980**;7:309–311
5. Butler MJ, Cornell SH, Damasio AR. Aphasia following pluridirectional tomography with metrizamide. The effect of patient position. *Arch Neurol* **1985**;42:39–45
6. Kaada B. Transient EEG abnormalities following lumbar myelography with metrizamide. *Acta Radiol [Suppl]* (Stockh) **1977**;355:380–386
7. Drayer BP, Rosenbaum AE. Metrizamide brain penetration. *Acta Radiol [Suppl]* (Stockh) **1977**;355:280–293
8. Marder E, O'Neil M, Grossman RI, Davis KR, Taveras JM. Cholinergic actions of metrizamide. *AJNR* **1983**;4:61–65