Intrathecal phenol and glycerin in metrizamide for treatment of intractable spasms in paraplegia

Case report

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Intractable lower extremity spasms after spinal cord injury is a significant source of morbidity. A case of refractory spasticity in paraplegia was successfully converted to flaccid paraplegia by intrathecal injection of phenol and glycerin in metrizamide. This chemical rhizotomy is simple and effective, and the presence of metrizamide allows both fluoroscopic guidance for accurate intrathecal phenol placement and good miscibility with cerebrospinal fluid. A brief comparative review of alternative therapeutic modalities is presented.

KEY WORDS: spasticity, phenol, metrizamide, chemical rhizotomy, glycerin

SPASTICITY resulting from corticospinal tract lesions in brain and spinal cord is seen in many neurological conditions. Infrequently, spontaneous spasms and contractures associated with upper motor neuron disease are incapacitating and add to the patient's handicap. Various treatments, including surgical rhizotomy and radiofrequency percutaneous rhizotomy, percutaneous embolization of the artery of Adamkiewicz to produce spinal cord infarction, continuous percutaneous epidural neurostimulation, and intrathecal injection of sclerosing agents, such as alcohol or phenol, have been advocated to treat this problem. For radiographic control of intrathecal injections, solutions with Pantopaque (iodophenylate) have traditionally been used. Problems inherent in this method will be noted below.

A case of posttraumatic paraplegia with severe hip flexor and adductor spasms is presented that was successfully treated with intrathecal instillation of phenol and glycerin in metrizamide (Amipaque, 3.75 gm). The advantages of using metrizamide for radiographic control of sclerosing agents are discussed.

Case Report

This 20-year-old Caucasian man was involved in a motor-vehicle accident 2 years before admission. He suffered a thoracic spine fracture that resulted in spastic paraplegia with a T-8 sensory level. The hip flexor and thigh adductor spasms in both legs deteriorated gradually. Spasms were frequently spontaneous and followed any tactile stimulation of the lower extremities. He was unable to sit in a chair or lie prone due to spasms and subsequently developed decubitus ulcers over the sacrum and both trochanters. The decubiti became infected and required surgical debridement and antibiotic therapy before definitive treatment of the spasticity.

Various treatments of refractory spasticity were reviewed, and intrathecal administration of phenol dissolved in glycerin appeared to be the most promising approach because it did not require an open surgical procedure in the presence of contaminated wounds, was effective, and could easily be repeated if necessary.

Injection Procedure. The injection procedure was performed as follows. The patient was placed in the left lateral decubitus position on the fluoroscopy table. Phenol crystals had been dissolved in glycerin to achieve a concentration of 10% by weight. Metrizamide was reconstituted with 4 ml of diluent, to achieve a concentration of approximately 310 mg iodine/ml; then 6 ml of phenol in glycerin solution was added to the bottle of reconstituted metrizamide and the mixture was shaken thoroughly. Final concentrations were 6% phenol and approximately 150 mg iodine/ml of metrizamide. Lumbar puncture was performed at the L2-3...
level. Four milliliters of the phenol-glycerin-metrizamide solution was slowly injected into the subarachnoid space with intermittent fluoroscopic monitoring. The table was positioned so that dye was visible surrounding the L-1 to L-4 nerve roots on the left side (Fig. 1), and was kept in this position for 20 minutes. The patient was then rolled into the right lateral decubitus position, an additional 3 ml of the solution was slowly injected with fluoroscopic monitoring, and the lumbar puncture needle was removed. Metrizamide was visible outlining the right T-12 to L-5 nerve roots (Fig. 2). The patient was kept in this position for 20 minutes and then he was transferred to a gurney in the sitting position. He maintained an upright posture for the next 4 hours and subsequently lay down. There were no complications.

Postinjection Course. Immediately following the procedure the lower extremities were flaccid, without reflexes, clonus, or spasms. Within 48 hours after the procedure, he experienced recurrence of hip flexor spasms, worse on the right side. One week after the first procedure he was brought back to the fluoroscopy suite and placed in the left lateral decubitus position while an L1–2 lumbar puncture was performed. Again, 4 ml of the phenol-glycerin-metrizamide solution was instilled into the subarachnoid space and the table was positioned so that the solution covered the left T-11 to L-5 nerve roots (Fig. 3). After the patient had maintained this position for 15 minutes, 3 ml more was injected and he was placed in the right lateral decubitus position. While in the Trendelenburg position to manipulate the solution in the subarachnoid space, he complained of right-sided thoracic paresthesias. The top of the phenol-glycerin-metrizamide column was fluoroscopically observed at the T-6 level, above the spinal cord lesion, and in contact with functioning nerve roots. His position was changed so that the T-11 to L-5 nerve roots on the right side were bathed with the solution for 15 minutes. The legs were flaccid following the procedure without recurrence of spasms at reevaluation 7 months after treatment.

Discussion

Lower extremity spasticity after spinal cord injury can be incapacitating, may retard rehabilitation, and may be associated with intractable decubitus ulcers.\(^{3,8}\) Chemical rhizolysis using phenol or ethanol destroys neurons, and thereby interrupts reflex arcs mediated by the treated nerve roots. Ethanol is hypobaric relative to cerebrospinal fluid (CSF); it is radiolucent and causes rapid, permanent destruction of nerve fibers,\(^{16}\) hence, accurate restriction of treatment to a selected group of nerve roots is difficult. Phenol has a slower and potentially reversible effect on nerve roots\(^{3-6,9}\) and retains its effectiveness when mixed with Pantopaque to form a hyperbaric, immiscible solution relative to CSF. The high viscosity of Pantopaque and its tendency to form non-coalescing globules make it impossible to completely fill a nerve root sleeve with a solution containing this agent. Phenol must diffuse from the oil phase of Pantopaque into the aqueous phase of CSF surrounding the distal nerve root and dorsal root ganglion to be

![Fig. 1. Cross-table radiograph with the patient in the left lateral decubitus position after an initial instillation of phenol-glycerin-metrizamide solution. The left L1-4 nerve roots are clearly outlined by metrizamide. Lumbar puncture was performed at L2-3.](image1)

![Fig. 2. Cross-table radiograph with the patient in the right lateral decubitus position after an additional 3 ml of phenol-glycerin-metrizamide solution was instilled into the subarachnoid space. The right T-12 to L-5 nerve roots are easily demonstrated surrounded by metrizamide, although the contrast material has been diluted by rolling the patient.](image2)

![Fig. 3. Cross-table radiograph with the patient in the left lateral decubitus position after the initial instillation of phenol-glycerin-metrizamide solution. The left T-12 to L-5 nerve roots are clearly outlined. Lumbar puncture was performed at L1–2.](image3)
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maximally effective. Phenol in glycerin is hyperbaric and miscible in CSF but is also radiolucent. It is more effective than phenol in Pantopaque; its miscibility in CSF and its lower viscosity enable the mixture to bathe a treated nerve root more uniformly and as far distally as the subarachnoid space extends.

In the case presented, the patient required relief of flexor and adductor spasms. An open percutaneous radiofrequency rhizotomy appeared unwise in the presence of infected decubitus ulcers so near the operative field. Major spinal artery embolization did not appear to have the reliability and simplicity of intrathecal chemical rhizotomies using phenol. We considered that phenol in glycerin was superior to phenol in Pantopaque, but were concerned about lack of fluoroscopic imaging. By adding metrizamide to the phenol-glycerin mixture, we preserved the good qualities of phenol in glycerin, namely the hyperbaric nature relative to CSF and miscibility in CSF, while gaining radiopacity. Diluting metrizamide with both phenol and glycerin also ensured that only a small amount of metrizamide would be injected and the risk of its toxic side effects were lowered accordingly.

Results of this treatment were excellent. The solution was easily visible on fluoroscopy and plain films. The phenol-glycerin-metrizamide solution flowed with gravity control and engulfed the nerve roots in the same manner as metrizamide alone during a myelogram. Clinically, the patient's spastic paraplegia was converted to flaccid paraplegia at completion of the first treatment. However, over the ensuing 48 hours, hip flexor spasms returned in a milder form. This was most likely due to inadequate treatment of the nerve roots supplying the iliopsoas muscles. Hence, the second procedure was performed using a higher lumbar puncture site and allowing the solution to flow more cephalad, involving the T-11 nerve roots and below (Fig. 3). Following the second treatment no further hip flexor or adductor spasms occurred and the patient was able to lie prone.

No significant alteration in the patient's spastic neurogenic bladder function occurred following the procedures. The amount and position of the phenol-glycerin-metrizamide solution was controlled carefully so that the most caudal nerve roots affected were at L-4 or L-5, sparing the S2-4 nerve roots involved in urinary bladder function. When he assumed the sitting position following the procedure, phenol was diluted sufficiently to obviate nerve root injury.

Short-term reversibility of phenol in phenol-glycerin-metrizamide solution was demonstrated when the solution ran to the T-6 level while the patient was being positioned. As this was above the spinal cord lesion, he began to have burning paresthesias in the anterolateral aspect of the chest at the level corresponding to the rostral extent of the column on fluoroscopy. These paresthesias were promptly relieved without sequelae by repositioning the patient so that the rostral extent of the solution was below the level of the spinal cord lesion.

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References


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