NEUROGENIC BLADDER AS A COMPLICATION OF ISOTOPE CISTERNOGRAPHY

Saran Jonas and Philip Braunstein

New York University School of Medicine, New York, New York

There have been several reports of aseptic type of meningeal reaction following the intrathecal injection of ^{131}I-IHSA (*1–4*). The reported reactions have been of the same general type, with high fever, stiff neck, increase of cell count and of protein in the CSF. Sugar usually (*1*), although not invariably (*3*), remains normal; cultures have always been negative. Complete recovery within a few days has been the rule. No objective neurological signs have been reported.

We have lately seen a patient who apparently developed a neurogenic bladder following attempt at radioisotope cisternography.

CASE REPORT

On the morning of October 11, 1971, a 77-year-old male had an attempt at cisternography by the lumbar route for evaluation of a clinically suspected "normal pressure hydrocephalus" syndrome with difficulty in walking, confusion, and urinary incontinence. An injection of 0.28 cc of a commercially produced high-specific-activity ^{131}I-IHSA (Albumotope) was made. CSF withdrawn at the time of this puncture was not remarkable. His temperature rose to 103.4° that afternoon. During the next 2 days his temperature fluctuated up to 101°; thereafter he was afebrile. There was no nuchal rigidity.

Scan of the lower spine 6 hr after the lumbar injection showed the characteristic appearance of spinal subdural (extra-arachnoid) distribution of a significant proportion of the injected radiopharmaceutical. Serial scanning of the head area on October 11 and 12, 1971, showed evidence of only minimal ascension of radioisotope into the subarachnoid space of the head. The study was thus a technical failure.

On October 19, 1971, a PEG was performed. At this time CSF showed 6,000 RBC and 50 WBC (70% polymorphs, 30% lymphocytes). Protein was 490 and glucose 78. Culture was negative. The PEG showed enlarged ventricles; no air passed over the convexities. He had no fever following the procedure.

On October 20, 1971, nine days after the original radioisotopic procedure, he was noted to have a urinary bladder residual volume of 750 cc. Foley drainage was instituted.

On October 26, 1971, IHSA cisternography was successfully initiated by the lumbar route. CSF was xanthrochromic, with 262 RBC, 22 WBC, protein of 120, and glucose of 65. The rapid passage of radioisotope into the ventricles, its notable persistence there, as well as its failure to concentrate over the convexity of the brain in the next 72 hr was compatible with a diagnosis of "normal pressure hydrocephalus." There was no fever following this procedure.

On November 4, 1971, his bladder was studied by cystometrography. This revealed pressure of 8 cm of water at 400 cc, 20 cm at 800 cc, and 35 cm at 1,000 cc. The patient had no sensation of fullness. The findings were interpreted as indicative of an atonic neurogenic bladder. No other objective signs of neurological damage were elicited.

On November 23, 1971, a ventricular-jugular shunt was inserted. He subsequently showed some clinical improvement with reference to his presenting symptoms.

Repeat cystometrography was performed on January 8, 1972; pressure-volume curve and volume-sensation correlations were interpreted as normal by the urologist. The patient subsequently was able to void voluntarily.

DISCUSSION

Fever, high CSF white cell and protein values, with normal sugar, negative cultures, and the subsequent regression of these phenomena without antibiotic therapy all favor the viewpoint that the pa-

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For reprints contact: Saran Jonas, Dept. of Neurology, New York University School of Medicine, 566 First Ave., New York, N.Y. 10016.

tient had suffered a form of chemical meningitis after the first attempt at IHSA cisternography on October 11, 1971.

The cystometric findings of November 4, 1971, indicated that denervation of the bladder had occurred, with recovery indicated by the study of January 8, 1972.

It is of interest that following insertion of the radiopharmaceutical on October 11, 1971, most had remained in the lower spinal area where it lay extra-arachnoidally and to a large extent in the subdural space. It can be postulated that the CSF findings of inflammation were consequent to an irritative process beginning, at least in part, on the outer surface and passage through to the inner (CSF-facing) surface of the arachnoid membrane. It seems reasonable to infer that the same irritative process transited the arachnoid coatings of the nerve roots as they passed through the subdural space of the lower spinal canal and caused temporary injury to the sacral nerve roots supplying the bladder. The sacral nerve root involvement may well have been intensified by the fact that the inserted radiopharmaceutical remained in the subdural space in high local concentration about these roots. It was not diluted or carried away as it would have been had it been properly inserted into the subarachnoid space. Thus a critical "duration of exposure-concentration" threshold for nerve root damage was possibly exceeded.

In the previous reported cases, including those of Barnes, et al (*1*) adequate subarachnoid insertion of at least a major proportion of the radiopharmaceutical is implied by the appearance of adequate radioactivity in the head area. It is reasonable to infer that the attendant dilution and dispersion by the CSF of the radiopharmaceutical kept the localized "duration of exposure-concentration" factor below the critical threshold level. Even in the presence of diffuse meningeal reaction local neural injury may thus be avoided.

The precise factors responsible for reactions in IHSA cisternography are not established at this time (*1,3,5*). It has been suggested that pyrogens in doses too low to be detected in the routine rabbit testing may at times be responsible (*1*). However, our experience as described above leads us to believe that in our case a chemical or physical irritant, and not a pyrogen, was responsible.

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