STASIS OF ^{111}In-DTPA IN THE POSTERIOR FOSSA IN PATIENTS WITH CEREBELLAR DEGENERATION

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Prolonged stasis of ^{111}In-DTPA was noted in the posterior fossa in two patients during cisternography. Both patients had clinical signs of cerebellar dysfunction and pneumoencephalographic evidence of marked cerebellar degeneration. Cisternography may be a useful adjunct in evaluating patients with suspected cerebellar atrophy.

Many radioactive agents in both colloidal and soluble forms have been used for the cisternographic investigation of the central nervous system (*1–3*). Extensive information has been gained on the patterns of cerebrospinal fluid flow in both normal and pathologic states. The great bulk of this information concerns the syndrome of "low-pressure hydrocephalus" and various forms of dementia associated with diffuse and focal cortical degeneration. Cisternography has also been much used to evaluate the patency of various types of cerebral shunts. Much less is known regarding the use of cisternography in those conditions localized primarily in the posterior fossa. Recently the opportunity to study two patients with advanced cerebellar degeneration presented itself; in both patients cisternography was performed.

CASE REPORTS

Case 1. A 52-year-old black man presented with a 10-year history of progressive difficulty with gait, coordination, and speech. He had been a heavy alcohol user from early youth. Ten years earlier, difficulty with his balance first developed, resulting in a broad-based gait. This progressed until he was confined to a wheelchair. About 1 year before the present admission, he noticed difficulty with speech, which progressed to a point where he had difficulty making himself understood. At the same time, he became aware of difficulty in coordinating the extremities and trunk.

On examination he was confined to a wheelchair. His speech was slurred and nasal. There was titubation of the head, even at rest. He had truncal ataxia and marked finger–nose dystaxia. His deep tendon reflexes were symmetric, and strength was good. Sensory examination showed evidence of peripheral neuropathy with decreased vibration and position sense in the lower extremities. Neurologic evaluation included a normal electroencephalogram and a lumbar puncture that revealed clear cerebrospinal fluid at normal pressure, with a protein of 72 and glucose of 66 mg/dl. A pneumoencephalogram (Fig. 1) showed an atrophied cerebellum with an enlarged

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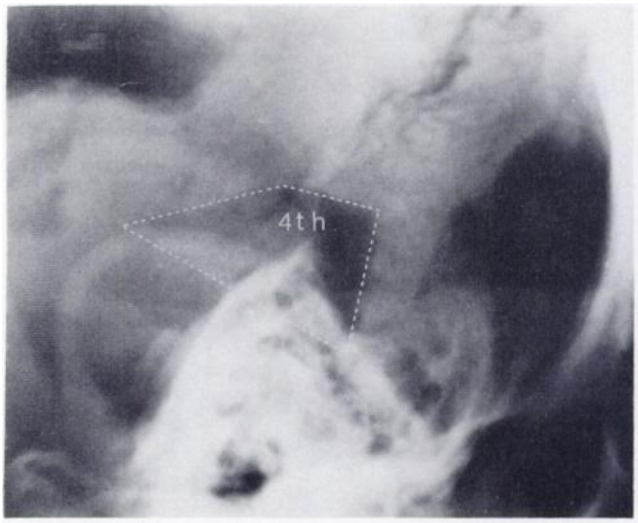

FIG. 1. Pneumoencephalogram in Case 1 shows marked dilatation of fourth ventricle with enlargement of cisterna magna. Appearance is compatible with advanced cerebellar degeneration.

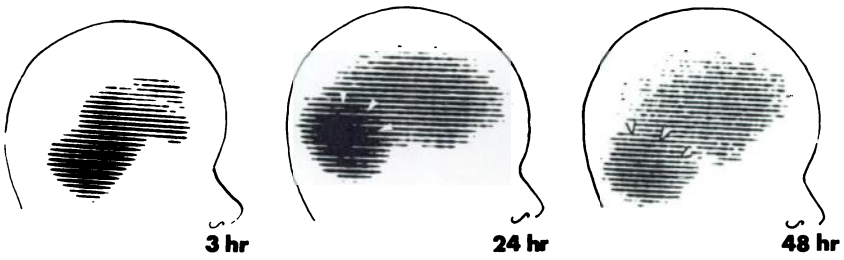

FIG. 2. Lateral ^{111}In-DTPA cisternogram in Case 1 shows rapid ascent and accumulation of radionuclide in posterior fossa at 3 hr. At 24 hr there is still marked pooling in posterior fossa with no ascent over cerebral hemispheres. Ventricular reflux is also noted. At 48 hr pattern of posterior fossa stasis is still observed.

fourth ventricle; the lateral ventricles were not enlarged. The cerebral hemispheres were normal and air was able to extend over them. A cisternogram was performed using 500 μCi of ^{111}In-DTPA, injected at L4–5. Serial views, obtained at 3, 24, and 48 hr, showed an abnormal and persistent stasis of the radionuclide in the posterior fossa (Fig. 2). There was also evidence of ventricular reflux. The diagnosis was primary cerebellar degeneration, secondary to alcoholism.

Case 2. The patient was a 38-year-old white man who, from the age of 17 to 25, had competed in motorcycle racing. However, 12 years prior to admission he noted difficulty with his balance and the appearance of nasal speech. The dystaxia progressed to a point where his gait was broad-based and he required the assistance of a walker. His mother and an aunt had had similar difficulties.

On examination, he was alert, with normal mentality. His gait was severely ataxic. He had difficulty with upward gaze. There was no nystagmus. He had distinctly nasal speech, titubation of the head, and truncal ataxia. A mild intention tremor and marked

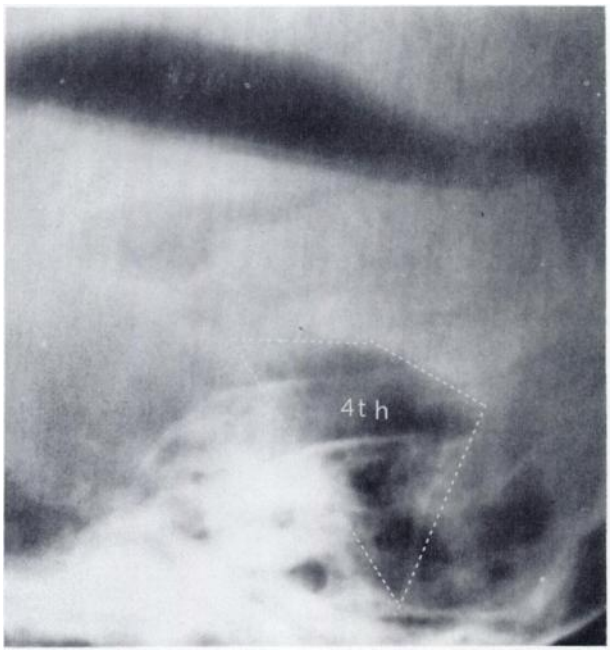

FIG. 3. Pneumoencephalogram in Case 2 shows advanced dilatation of fourth ventricle and enlargement of cisterna magna. Appearance suggests advanced cerebellar degeneration.

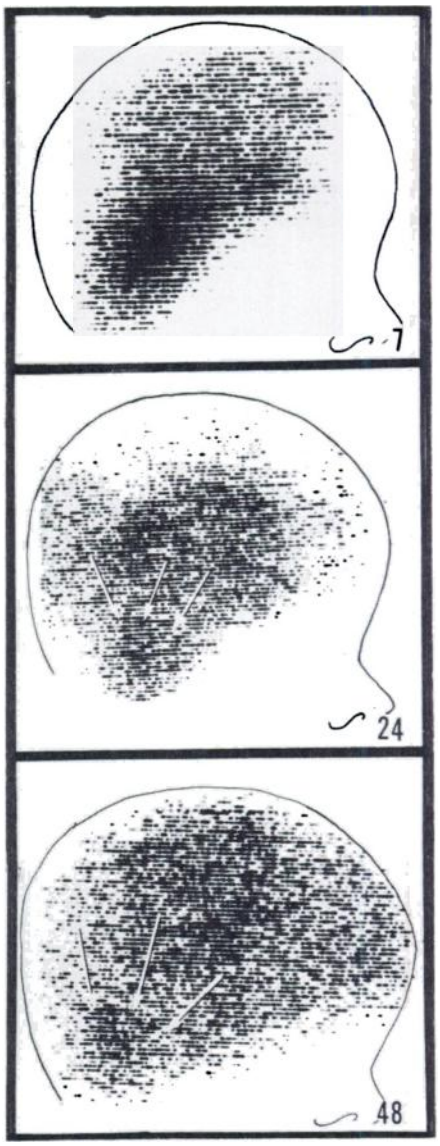

FIG. 4. Lateral ^{111}In-DTPA cisternograms in Case 2. Study at 7 hr shows concentration of radionuclide in posterior fossa with early ascent over cerebral hemispheres. At 24 hr tracer persists in posterior fossa and also in lateral ventricles. At 48 hr radionuclide has ascended over cerebral hemispheres. However, distinct pooling is still noted in posterior fossa (white arrows).

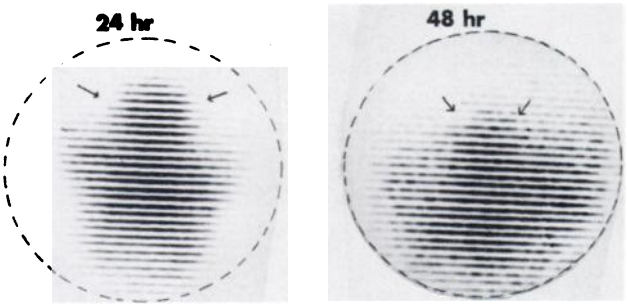

FIG. 5. Brow-down anterior cisternograms in Case 2 show stasis of radionuclide in posterior fossa at 24 and 48 hr. Reflux into lateral ventricles is seen at 24 hr, with some ventricular reflux persisting at 48 hr.

finger–nose dystaxia were present. His electroencephalogram showed bilateral cerebral dysrhythmia. A pneumoencephalogram (Fig. 3) showed dilatation of the fourth ventricle with atrophy of the cerebellum and moderate enlargement of the lateral ventricles. A cisternogram was performed using 500 μCi of ^{111}In-DTPA injected at L4–5. Views, obtained at 7, 24, and 48 hr, showed a persistent significant stasis of the radionuclide in the posterior fossa (Figs. 4 and 5). He was believed to have a form of hereditary degeneration.

DISCUSSION

Cerebellar degeneration is a relatively rare clinical condition. It occurs both as a focal condition and associated with brain stem, cerebral, and spinal involvement (*4*); it may be sporadic or familial. Cerebellar degeneration also has been described secondary to hypoxia, alcoholism, and diphenylhydantoin therapy (*5*). Suspected cerebellar degeneration is usually documented by the use of pneumoencephalography. The classic findings are enlargement of the cisterna magna and fourth ventricle and shrinkage of the cerebellar folia.

Cisternography has not been widely applied in the diagnosis of cerebellar degeneration, and the pattern of stasis noted in these two patients has not been previously reported. Examples of posterior fossa stasis on cisternography have been reported in cases of resection of cerebellar tumors, in postsurgical decompression of Arnold–Chiari malformations, and in extradural introduction of radionuclide through cisternal radiotracer injections (*6*). In all these conditions, increased activity in the posterior fossa is related to increased cerebrospinal fluid volume.

Prolonged transit time is observed if inflow to this region is normal or decreased: $T = V/F$, where T is the mean transit time, V is the volume, and F is the flow rate. Above the tentorium, abnormal pooling occurs with subarachnoid cysts, porencephalic cysts, focal atrophy, and surgical defects (*7*). These abnormal pools fill early and empty late. The pooling observed in these two cases of cerebellar degeneration is probably due to localized enlargement of the posterior fossa cisternae and increased cerebrospinal fluid volume, even though the occurrence of ventricular reflux does suggest some alteration of flow. Pneumoencephalography gave no evidence of blockage or air over the cerebral hemispheres. The ventricular reflux may just be due to the prolonged presence of the radionuclide in the posterior fossa in the area where spinal fluid outflows. Similar reflux, though more transient, has been observed in cisternograms during the early phase while the tracer is in the posterior fossa in patients otherwise completely normal (*8*).

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