UNILATERAL VENTRICULAR REFLUX: CASE REPORT

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Reflux of ^{131}I-human serum albumin with a dilated ventricle from the contralateral ventricle was observed in a patient with an abnormal cisternogram.

Complete ventricular reflux is a well-known phenomenon in some cases of communicating hydrocephalus (*1*). Following suboccipital or lumbar introduction of an albumin-bound tracer (e.g., ^{131}I-IHSA) there may be a complete reflux of the tracer into the lateral ventricles, whereas normally it is distributed through the basal cisterns into the subarachnoid spaces around the hemispheres. In such cases following intraventricular introduction, the tracer may not be transported to the basal cisterns and to the hemispheres as in the normal subject, and there can be ventricular stasis. It is generally accepted that reflux and stasis are attributable to a blockade of the normal pathways of the cerebrospinal fluid at the base or around the convexities of the cerebral hemispheres due to the sequelae of a subarachnoid hemorrhage or meningitis. In communicating hydrocephalus with ventricular reflux, the cerebrospinal fluid presumably is transported through the stretched ependyma of the ventricles and reabsorbed into the circulation through the brain–blood barrier. Some observations in man (*2*) and in animals (*3–7*) support this view. The following case report needs further interpretation.

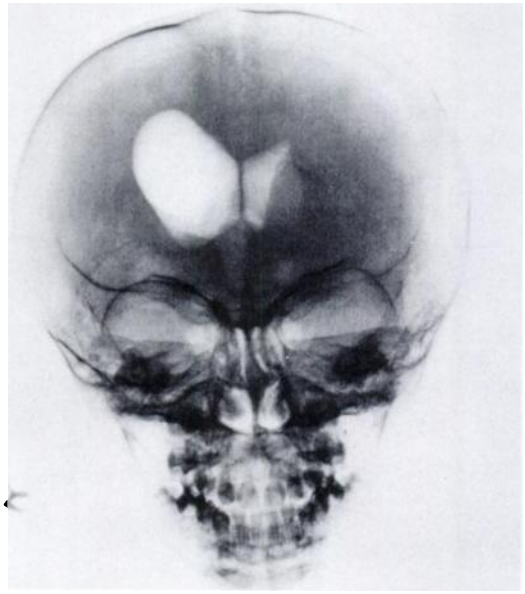

FIG. 1. Air-ventriculogram reveals dilated right lateral ventricle.

CASE REPORT

Unilateral ventricular reflux in a 2-year-old girl is described. The mother's pregnancy and delivery were without complications. The child was born with a left-sided spastic hemiparesis but did not develop any further neurologic deficit. She was mentally retarded.

Radiologic examination revealed the presence of a dilated right lateral ventricle seen in the air-ventriculogram (Fig. 1). The most interesting finding, however, was a distribution of ^{131}I-IHSA, administered by suboccipital injection, solely into the right lateral ventricle (Fig. 2). Similarly, injection of the tracer in the left lateral ventricle showed presence of the tracer in the right dilated lateral ventricle within 20 min (Fig. 3).

DISCUSSION

To our knowledge, a ventricular reflux as described here has not yet been reported and interpretation seems to be difficult. We can, however, offer two explanations of this phenomenon:

1. Aside from a basal lesion which caused the ventricular reflux, a second lesion may exist in the left hemisphere that does not permit passage of the tracer through the ependyma of the ventricle. A left-sided hemisphere lesion is, however, not compatible with the

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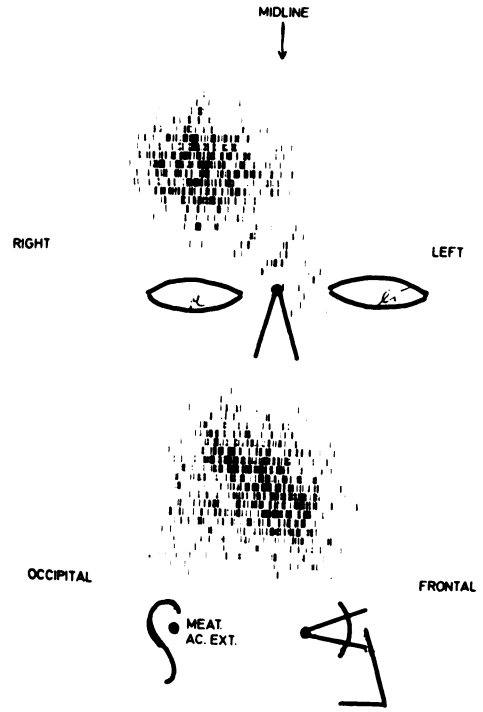

FIG. 2. Scintigram 2 hr after suboccipital injection of 10 μCi ^{131}I-IHSA. Complete reflux of tracer into right lateral ventricle through basal cisterns is seen. In both instances tracers were completely absorbed after 48 hr (posterior and left lateral view).

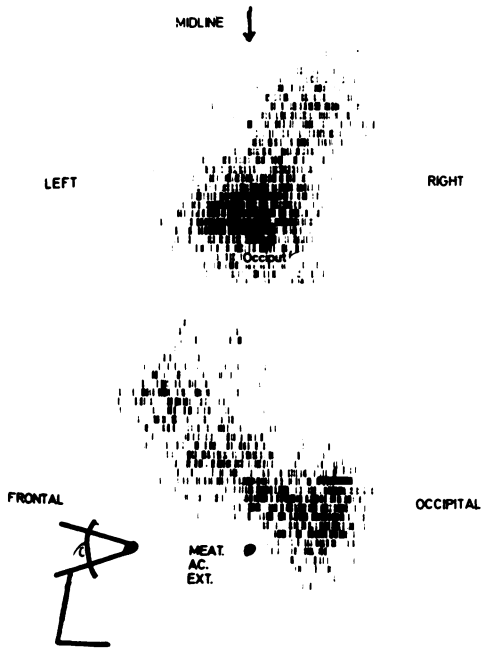

FIG. 3. Scintigram 4½ hr after injection of 10 μCi ^{131}I-IHSA into left lateral ventricle. Passage of tracer into right lateral ventricle through third ventricle was completed within 20 min after injection (anterior and right lateral view).

clinical finding of a left spastic hemiparesis and with the assumed mechanisms of transependymal distribution of the tracer previously mentioned.

2. A lesion may exist in the right hemisphere only. This lesion may alter the ependyma and/or the subependymal tissue of the right lateral ventricle in a way which causes easy penetration of cerebrospinal fluid through this ependyma and subsequent absorption. Therefore, the normal or other abnormal pathways of cerebrospinal fluid are not used and are functionally inactive.

These explanations are of course tentative and further observations of similar cases would be of considerable interest.

REFERENCES

1. OBERSON R, MARTINI T: Scintigraphie des espaces sousarachnoidiens pericérébraux. In *Colloque sur la Scintigraphie Medicale au Moyen de Radioisotopes.* Salzburg, Austria, 6.–15.8. 1968

2. MILHORAT TH, HAMMOCK MK: Isotope ventriculography. Interpretation of ventricular size and configuration in hydrocephalus. *Arch Neurol* 25: 1–8, 1971

3. SAHAR A, HOCHWALD GM, RANSOHOFF J: Alternate pathway for cerebrospinal fluid absorption in animals with experimental obstructive hydrocephalus. *Exp Neurol* 25: 200–206, 1969

4. WELLER RO, WISNIEWSKI H: Histological and ultrastructural changes with experimental hydrocephalus in adult rabbits. *Brain* 92: 819–828, 1969

5. MILHORAT TH, CLARK RG: Some observations on the circulation of phenolsulfonphtalein in cerebrospinal fluid: normal flow and flow in hydrocephalus. *J Neurosurg* 32: 522–528, 1970.

6. MILHORAT TH, CLARK RG, HAMMOCK MK, et al: Structural, ultrastructural, and permeability changes in the ependyma and surrounding brain favoring equilibration in progressive hydrocephalus. *Arch Neurol* 22: 397–407, 1970

7. CLARK RG, MILHORAT TH: Experimental hydrocephalus: Part 3. Light microscopic findings in acute and subacute obstructive hydrocephalus in the monkey. *J Neurosurg* 32: 400–413, 1970