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The posterior lobe of the pituitary gland was studied by MR imaging in 30 children without pituitary gland disease and compared with studies from a group of 13 children with central diabetes insipidus, including eight cases of primary diabetes insipidus and five cases of diabetes insipidus secondary to suprasellar tumors (four proved germinomas, one still unknown tumor). Two components in the sella turcica were identified in all 30 children without pituitary gland disease, and the posterior lobe was identified as a high-intensity structure on T1-weighted images. In all 13 patients with diabetes insipidus, the normal hyperintense signal of the posterior hypophysis was absent on T1-weighted images. Three patients with suprasellar tumors presented with a progressively enlarging pituitary stalk on follow-up.

Our findings show that absence of the normal hyperintense signal of the posterior lobe is closely related to a loss of function of the neurohypophysis. Size or signal modification of the pituitary stalk should suggest the development of a suprasellar tumor.

Central diabetes insipidus results from low blood levels of antidiuretic hormone (ADH). ADH is secreted by the neurohypophysis, whose development, blood supply, and function differ from the anterior lobe of the pituitary gland. This condition can be primary (idiopathic, sporadic, or familial) or secondary to tumor, histiocytosis, infection, cerebral malformation, or trauma.

Several authors have shown that MR imaging is able to identify the normal posterior lobe as a region of characteristically high signal intensity on the posterior pituitary fossa on T1-weighted images [1, 2]. Since we lack references about the normal and pathologic MR appearance of the posterior hypophysis in childhood, a control group was studied and compared with a group of prospectively studied patients with known central diabetes insipidus to evaluate the usefulness of MR imaging in childhood diabetes insipidus.

Materials and Methods

Thirty children 3 months to 18 years old (mean, 8 years) with no signs or symptoms of pituitary gland or sella disease were examined with MR imaging between January 1985 and February 1988 for a variety of reasons. Those who had had radiotherapy, craniopharyngioma, or previous surgery in the pituitary region were excluded. The patients were retrospectively studied, and the morphology and intensity of the signal of the posterior lobe were assessed. (All infants were examined by CT simultaneously or in the previous 1–7 months.)

In comparison, 13 children—10 girls and three boys, 2–19 years old (mean, 10 years)—with diabetes insipidus were examined prospectively. The biochemical evaluation included concomitant blood and urine osmolality after controlled water deprivation and response to exogenous arginine vasopressin. Laboratory tests for tumor markers (HCG, α-fetoprotein) were performed on serum and CSF. Given the frequency of associated anterior hypophysis insufficiency in diabetes insipidus, growth hormone levels were obtained in selected cases. Polyuria and dilute urine of psychogenic or renal origin were excluded.

All patients were examined in the supine position on a 0.5-T superconducting MR unit.* Images were acquired with a head coil, a 256 × 256 matrix, and a 256-mm field of view.

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Earlier scans were obtained by using 6–7-mm-thick sections. Because of improved techniques, contiguous, thinner 4–5-mm-thick sections were used for the later scans. T1-weighted sagittal sections were acquired by using gradient refocusing (TR = 300–400, TE = 14) or spin-echo (TR = 300–500, TE = 21) sequences. Coronal sections and T2-weighted sequences (TR = 1500–1800, TE = 60–120) were added in selected cases.

The interval between onset of diabetes insipidus and the first MR examination varied from 6 months to 14 years. The MR features studied included presence of a hyperintense signal in the region of the posterior hypophysis on T1-weighted images; relative intensity of the signal compared with the anterior hypophysis, white matter, corpus callosum, and optic chiasm; size and signal of the pituitary stalk; and appearance of the structures on T2-weighted images, when available. Using ROI analysis, measurements of the signal intensity were made when the signal of the posterior hypophysis was not clearly brighter than that of the white matter and corpus callosum.

Results

Control Group

A distinct hyperintense area in the posterior region of the sella turcica was seen in all 30 of the control subjects (Fig. 1). More precisely, the signal of the posterior hypophysis was higher than the signal of the anterior hypophysis in all cases. The relative intensity of the signal compared with other structures was variable: the signal of the posterior hypophysis was hyperintense relative to the white matter in 20 of 30 cases, to the corpus callosum in 20 of 30 cases, and to the optic chiasm in 27 of 30 cases.

ROI measurements were obtained in 10 control patients in whom the signal of the posterior hypophysis was not brighter than that of the corpus callosum. The intensity numbers of the posterior hypophysis were higher in nine cases and equal in one case. A slight obliquity of the plane of section was noted in all 10 of these patients.

Diabetes Insipidus Group

In all 13 patients with diabetes insipidus, regardless of its cause, the normal hyperintensity of the posterior pituitary lobe was absent on sagittal T1-weighted images. Eight patients had primary diabetes insipidus, and the anatomy of the hypothalamic and pituitary region was found to be normal (Fig. 2). In five patients, a suprasellar mass invading the pituitary stalk and the floor of the third ventricle was demonstrated (four proved germinomas, one still unknown tumor). Germinomas were diagnosed on the basis of CSF cytology. Tumoral markers were positive in three patients. Two patients presented with associated low levels of growth hormone.

Three patients with germinomas and diabetes insipidus demonstrated a thick pituitary stalk on the first MR scan without mass effect in the suprasellar region. A hyperintense signal of the stalk on T2-weighted images was seen in two of them. Follow-up MR scans of the three patients confirmed the progressive enlargement of the stalk and the presence of a growing suprasellar tumor (Fig. 3).

Discussion

The embryology, histology, blood supply, and function of the two pituitary lobes are quite different. Recent embryologic studies have demonstrated the common origin of facial structures and brain, and have described the dorsoventral rotation of the prosencephalic neural plate containing the antehypophyseal primordium during the second week of gestation. Thus, part of the antehypophysis could be of neural origin [3]. The posterior hypophysis develops as a downward projection of the hypothalamus.

ADH, a polypeptide hormone secreted from the neurohypophyseal system, acts on the collecting tubules of the kidney to conserve water and concentrate urine. The hormone is formed in the cells of the supraoptic and paraventricular nuclei of the hypothalamus, bound to a carrier protein called neurophysin, and stored in neurosecretory granules. The axons of the cells lying in the supraoptic and paraventricular nuclei extend down to the pituitary stalk and terminate in the posterior lobe of the pituitary gland. The neurosecretory granules are transported in the axons of the neurosecretory cells to the posterior lobe, where ADH can be released in the bloodstream. Therefore, central diabetes insipidus is related to any
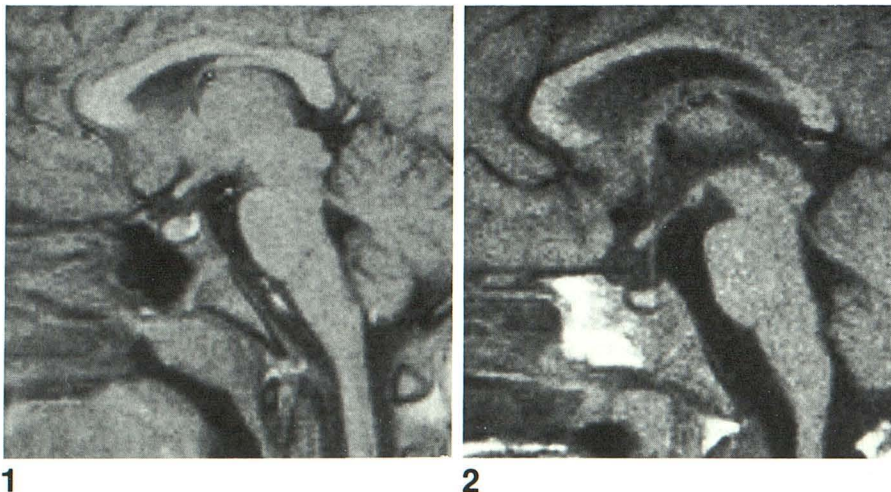

1   **2**

Fig. 1.—Normal study, 7-year-old girl. T1-weighted MR image, 360/14, midsagittal section, shows two components in the sella turcica: posterior lobe of pituitary gland displays a bright signal as compared with anterior part.

Fig. 2.—Primary diabetes insipidus, 2-year-old boy. T1-weighted midline sagittal section, 400/14, shows no space-occupying lesion. Note absence of normally high signal of posterior hypophysis.

condition impairing the function of the neurohypophyseal system.

The spatial resolution of recent MR imagers allows us to clearly delineate the clinoids, the clivus, and the two components of the pituitary gland. The posterior lobe has been described as a high-intensity structure in the posteroinferior margin of the pituitary gland on T1-weighted sagittal images [1, 2, 4]. Nishimura et al. [1] identified the high-intensity signal of the posterior hypophysis in every case of 60 normal volunteers. Colombo et al. [4] reported that in 13% of healthy subjects, the high signal intensity of the posterior hypophysis could not be identified. In our series, the hypersignal was demonstrated in 30 of 30 control subjects. However, strong variations in signal intensity are induced by an obliquity of the plane of section caused by partial volume effect of nearby structures of low intensity (carotid arteries, cisterns). We think that slice thickness and strict sagittal sections to avoid averaging artifacts are the most important factors for a successful delineation of the posterior hypophysis (Fig. 4). In our control group, the lowest measured values of the signal of the posterior hypophysis were related to a slight obliquity of the plane of section.

Several authors have expressed controversial views on the potential causes for the hyperintense signal of the posterior hypophysis on T1-weighted images. The presence of fat, subacute hemorrhage, and slowly flowing blood has been advocated [5, 6]. Other investigators concluded that the posterior lobe itself was hyperintense on T1-weighted images acquired by using chemical shift or STIR studies (Brunelle F, unpublished data); these authors described the short T1 value of the posterior hypophysis with ADH-containing neurosecretory granules or lipid material in the pituicytes.

There are few published observations about the MR appearance of the pituitary gland in infants and children. Wolpert et al. [6] reported no visible intensity differences across the sella in eight normal infants under 8 weeks of age. In an adult study that included three pediatric cases, Fujisawa et al. [7] speculated that the high signal of the posterior hypophysis is related to its functional integrity.

In our study, we were able to identify two components of the pituitary fossa in all control group patients, the youngest of whom was 3 months old. We observed the loss of the hyperintense signal of the posterior hypophysis in all 13 patients with diabetes insipidus. Therefore, we suggest that

Fig. 3.—Germinoma, 14-year-old girl.
A, Initial MR study. T1-weighted sagittal section. A large pituitary stalk and a hypointense posterior lobe are seen.
B, Follow-up MR study, 6 months later. A large tumor invading the hypothalamus is seen. Posterior lobe is hypointense.
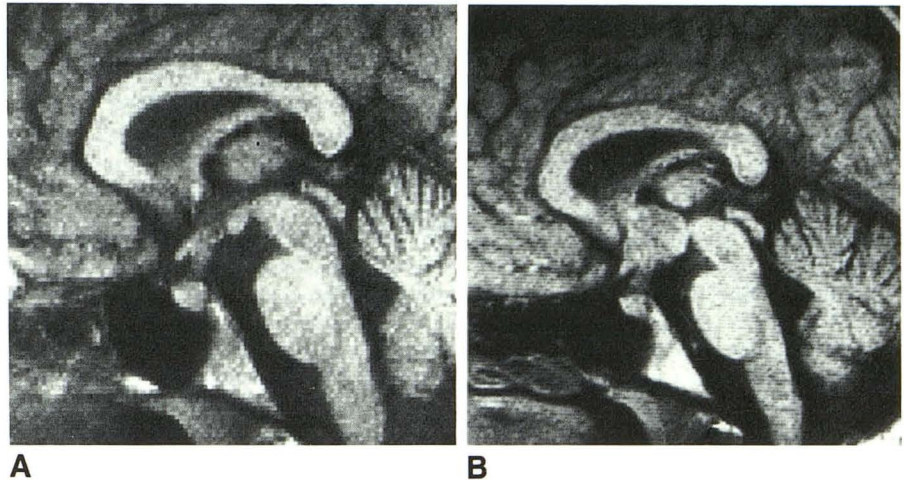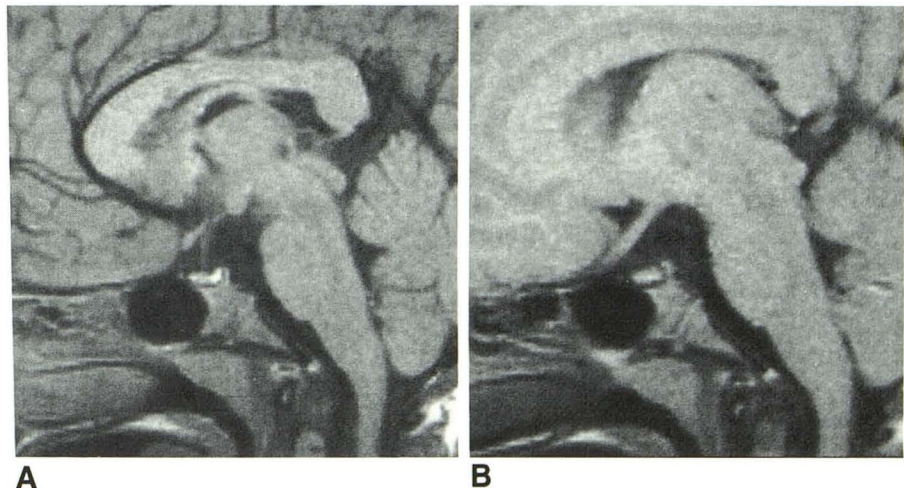

A

B

Fig. 4.—Normal study, 6-year-old girl.
A, Midsagittal T1-weighted section. Normal posterior lobe is hyperintense.
B, Midline section off 4.8 mm to left. Although most of the midline structures are seen (IV ventricle, chiasm), the slight midline shift induces an apparent loss of posterior hyperintensity caused by volume averaging.

A

B

this MR feature could be the most specific sign in the diagnostic imaging of diabetes insipidus in children. Although some authors [8] reported that the posterior lobe can be differentiated from the anterior hypophysis on CT, anomalies of the posterior lobe have not been reported yet. Thus, MR alone can provide information about the morphologic and functional aspects of the posterior hypophysis.

Two categories of diabetes insipidus have been described: primary and secondary [9]. The proportion of primary form (idiopathic, isolated, or familial) can vary from about 9–45%. The proportion in our series is even higher, 62%, but this could be due to selection bias, probably because several cases were children with known nontumoral diabetes insipidus prospectively included in the study. In our institution, children with no tumor development on a 4-year follow-up after onset of diabetes insipidus are considered to have a primary form of the disease. These patients showed no hypothalamohypophyseal morphologic anomalies.

The major causes of secondary diabetes insipidus are intracranial tumors, including germinomas, optic and hypothalamic gliomas, astrocytomas, lymphomas, histiocytosis X, cerebral malformations, infection, and trauma. Among the many causes of diabetes insipidus, we found a high proportion of germinomas in our series (four of our 13 cases), even though this cause is described as less frequent than histiocytosis X [9, 10]. Most of these germinomas showed the same MR features: suprasellar tumors often associated with a progressively enlarging pituitary stalk. Their diagnosis was made by CSF cytology and tumoral markers, when present.

Comparing MR with CT, all masses demonstrated by CT were also demonstrated by MR imaging. In the future, the use of Gd-DOTA* could perhaps improve the sensitivity and specificity of tumoral detection by MR imaging. Because of its ability to produce multiplanar sections, MR imaging allows a better tumoral staging than CT does, especially in the region of the floor of the third ventricle.

Therefore, we think that MR imaging should be performed in the diagnostic workup of central diabetes insipidus in childhood to search the hypothalamohypophyseal region for a thick pituitary stalk or a suprasellar tumor.

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