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MR Imaging of the Ectopic Bright Signal of Posterior Pituitary Regeneration

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High-field-strength MR studies of 13 patients with sellar and/or parasellar tumors revealed an aberrant location of the posterior pituitary bright signal in the hypothalamus in seven cases and in relation to the pituitary infundibulum in six cases. Five of the MR studies were obtained in patients who had not had surgery and had pituitary adenomas producing compression and/or destruction of the posterior lobe. In the other eight cases, the aberrant bright signal occurred after hypophysectomy in seven patients and after removal of a craniopharyngioma in the eighth.

Accumulation of neurosecretory material and regeneration of pituitary tissue in these ectopic locations have been documented previously in animal experiments and in a few reports in humans after hypophysectomy. MR imaging now provides further corroborative evidence in vivo of this process in which a "miniature posterior lobe" is formed.

Formation of an ectopic hypothalamic or infundibular miniature posterior pituitary lobe was first described in animal experiments [1-3] and later confirmed in humans after hypophysectomy with autopsy examination [4-6]. With the advent of MR imaging, direct in vivo visualization of this physiological response to posterior pituitary tissue loss is now possible. We report our experience in 13 patients.

Materials and Methods

The 13 patients included four men and nine women, ranging in age from 23 to 66 years old. They were divided into two groups of five and eight patients, respectively. The five patients in group I had not been operated on at the time of MR imaging, and all had presumed pituitary adenomas, four with varying degrees of suprasellar extension (see Table 1). In the fifth patient, with intrasellar adenoma, MR demonstrated hemorrhage within the tumor at the time of clinical presentation of pituitary apoplexy, and a repeat MR was performed as a follow-up study 1 yr later. The follow-up MR findings provided the basis for including this patient in our report. One other patient in this group was also operated on after MR imaging, with surgical confirmation of a prolactinoma.

The eight patients in group II all had MR performed at intervals ranging from 9 months to 20 years postsurgery (see Table 2). Preoperative MR was also available in one of these cases, a patient with a very large craniopharyngioma. There were three cases of large chromophobe adenoma and four cases of prolactinoma, including one large and three small tumors.

The MR examinations were obtained on a GE 1.5-T superconducting magnet with multislice spin-echo pulse sequences. Sagittal and coronal T1-weighted images were acquired with 800/20/4 (TR/TE/excitations), 3-mm slice thickness with 0.6-mm interslice gap, 256 × 256 acquisition matrix, and a 24-cm field of view for the sagittal images and a 20-cm field of view for the coronal images. Axial and coronal cardiac-gated intermediate and T2-weighted images were acquired with 2000/20/70/1 5-mm slice thickness with 2.5-mm interslice gap, 256 × 256 acquisition matrix, and a 24-cm field of view for the axial images and a 20-cm field of view for the coronal images.

The imaging features evaluated on the MR scans in each group included the location and size of an ectopic bright signal found in relation to either the median eminence of the

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TABLE 1: Nonoperative Cases (*n* = 5)

Case No.	Age	Gender	Tumor Type	Bright Signal		
				Location	Size	Intensity
1	56	F	Large, presumed chromophobe adenoma	Median eminence	5 × 2 mm	+++
2	23	F	Large, presumed chromophobe adenoma	Median eminence	3 × 2 mm	+++
3	66	M	Hemorrhage into intrasellar adenoma (pituitary apoplexy); empty sella 1 yr later	Median eminence	4 × 7 mm	+++
4	26	F	Large prolactinoma, later confirmed surgically	Lower infundibulum	6 × 5 mm	+++
5	34	F	Sarcoidosis; CT-enhancing mass; hyperprolactinemia; bromocriptine treatment 2 yrs prior to MR	Lower end of herniated infundibulum in empty sella	4 × 2 mm	+++

+++ = Same intensity as a normally located posterior pituitary bright signal.

TABLE 2: Postoperative Cases (*n* = 8)

Case No.	Age	Gender	Tumor Type	Preoperative Tumor Size	Treatment	Surgery-MR Interval	Bright Signal		
							Location	Size	Intensity
1	23	F	Prolactinoma	Small	Transsphenoidal hypophysectomy	3 yr	Lower stalk	2.5 × 3 mm	++++
2	26	M	Prolactinoma	Large	Transsphenoidal hypophysectomy, two operations	1 yr	Median eminence and upper stalk	5 × 5 mm	++
3	24	F	Prolactinoma	Small	Transsphenoidal hypophysectomy, radiotherapy	3 yr	Median eminence	4 × 4 mm	+++
4	30	F	Prolactinoma	Small	Transsphenoidal hypophysectomy	10 yr	Upper infundibulum	2 × 3 mm	+++
5	46	M	Chromophobe adenoma	Large	Frontal craniotomy, transsphenoidal hypophysectomy, radiotherapy	20 yr	Infundibular stalk	2 × 3 mm	++
6	63	F	Chromophobe adenoma	Large	Frontal craniotomy	12 yr	Median eminence	5 × 2.5 mm	++++
7	45	F	Chromophobe adenoma (acromegaly)	Large	Frontal craniotomy	17 yr	Lower stalk	2 × 3 mm	++
8	21	M	Craniopharyngioma	Large	Frontal craniotomy, radiotherapy	9 mo	Median eminence	8 × 6 mm	++++

++ = Less intensity than a normally located posterior pituitary bright signal.

+++ = Same intensity as a normally located posterior pituitary bright signal.

++++ = Greater intensity than a normally located posterior pituitary bright signal.

hypothalamus or the pituitary infundibulum. The intensity of the aberrant bright signal was noted relative to the bright signal intensity usually seen in the neurohypophysis in its normal location on T1-weighted images. The signal change characteristics on the long TR axial and coronal dual-echo sequences were also evaluated. The appearance of the sella, the size and extent of pituitary tumor if present, and other related features such as an empty sella with distortion of normal anatomy in patients who had had surgery were noted as well.

Results

The MR studies of individuals in group I (the nonoperated patients) showed an ectopic bright signal in the median eminence of the hypothalamus in two patients who both had large pituitary tumors with suprasellar extension presumed to

be chromophobe adenomas (Fig. 1). No bright signal was evident in the usual location of the posterior pituitary gland. The hypothalamic bright signal appeared equal in intensity to that of the posterior pituitary bright signal on T1-weighted images that can be seen in normal patients. A third case with median eminence bright signal had an empty sella 1 yr after a clinical episode of pituitary apoplexy that had also been documented by an MR study at that time showing hemorrhage within an intrasellar pituitary adenoma (Fig. 2). In the fourth case, an ectopic bright signal was evident in relation to the lower infundibulum (Fig. 3) in a patient with a large prolactinoma that was later verified by surgery; again, as with the other cases, no bright signal could be seen in the normal location of the posterior pituitary gland. The final patient in group I had clinical and laboratory diagnoses of both sarcoidosis and hyperprolactinemia. A cranial CT scan 2 yr previously

Fig. 1.—Group I, case 1: Presumed chromophobe adenoma.

A and B, Sagittal (A) and coronal (B) MR images show large pituitary tumor compressing and presumably destroying posterior pituitary lobe. Note bright signal of the ectopic posterior lobe in the median eminence (arrow).

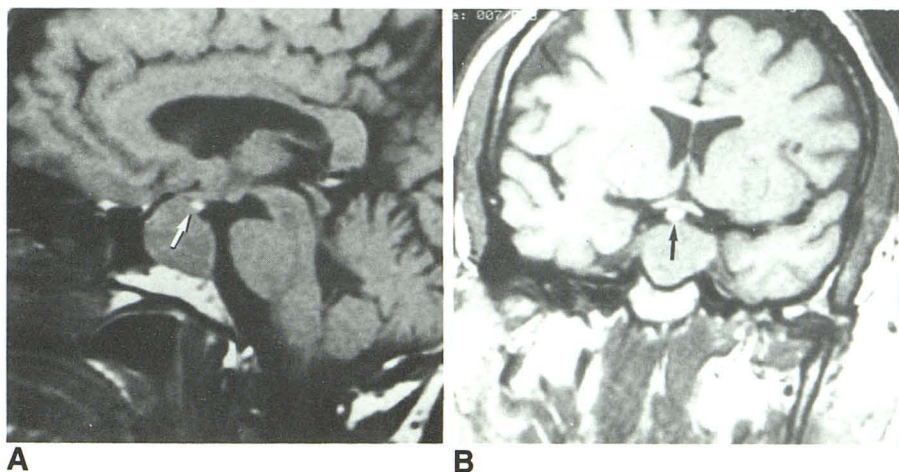
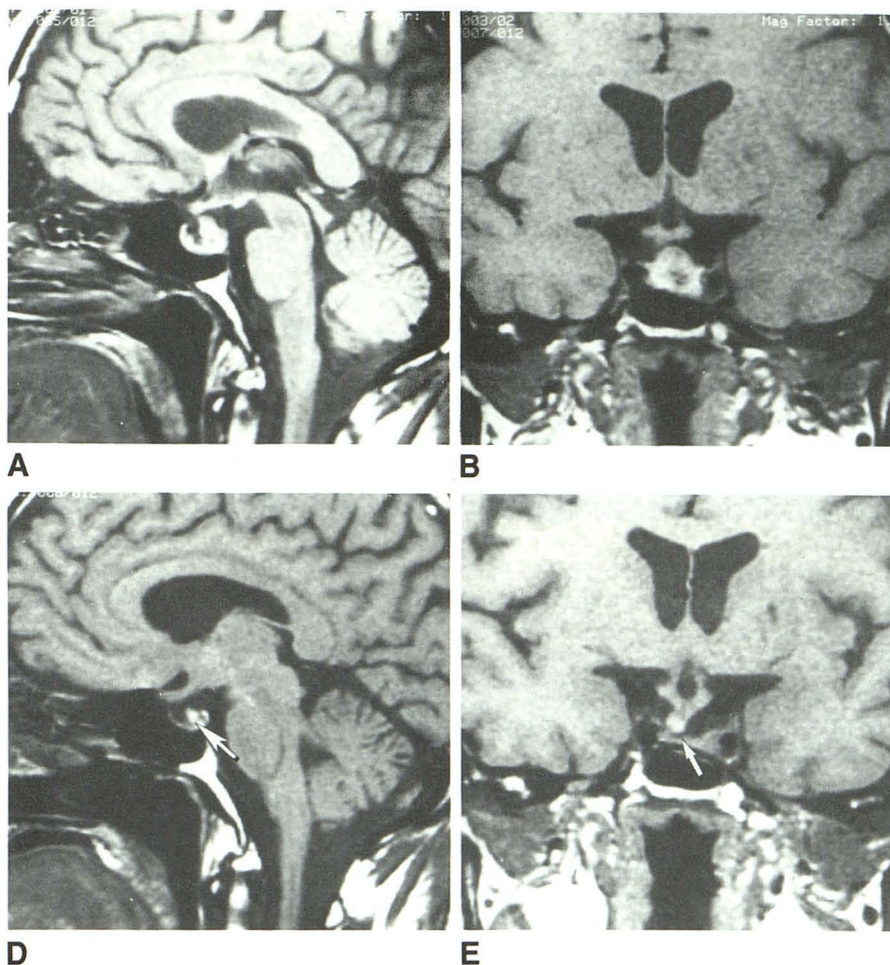


Fig. 2.—Group I, case 3: Pituitary apoplexy.

A and B, MR images show high signal of hemorrhage in a pituitary adenoma in sagittal (A) and coronal (B) sections.

C-E, 1-year follow-up study shows empty sella (C) and bright signal of displaced median eminence (arrow) in paramedian sagittal (D) and coronal (E) sections.



had demonstrated an enhancing suprasellar mass. The patient received bromocriptine therapy over the following 2-yr interval with resolution of both the suprasellar mass and the hyperprolactinemia. The MR study obtained then revealed an

ectopic bright signal at the lower end of the pituitary infundibulum herniated inside an empty sella.

Overall, in the group I cases, no significant difference in size of the ectopic bright signal could be noted with respect

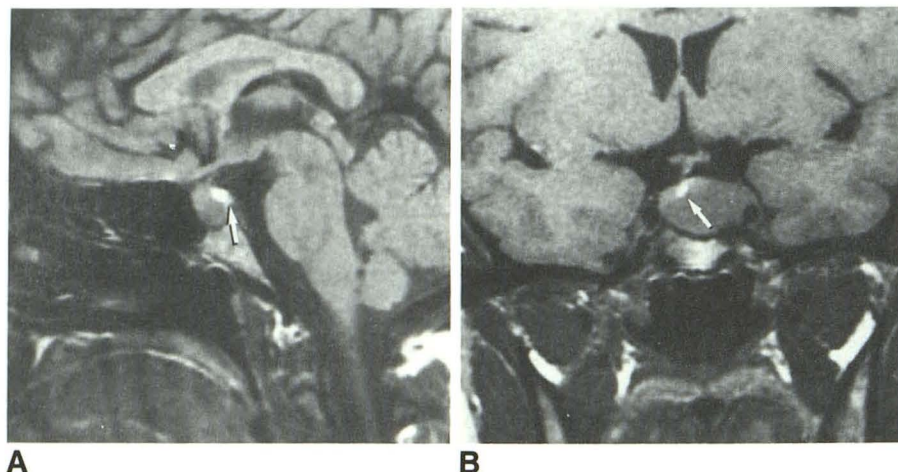


Fig. 3.—Group I, case 4: Prolactinoma. A and B, Large intrasellar tumor and bright signal in relation to displaced infundibulum (arrow) are seen in sagittal (A) and coronal (B) sections. Note tumor invasion of the left cavernous sinus (B).

to location in the hypothalamic median eminence or in relation to the infundibulum. In every case, the intensity of the ectopic bright signal appeared equal to the bright signal intensity that can be observed in the neurohypophysis in its normal location on T1-weighted images, and in no case was a normally located posterior pituitary bright signal evident. Proton-density images showed the bright signal equivalent to that seen with normal posterior pituitary tissue, and on the axial and coronal T2-weighted images, the ectopic bright signal could no longer be discriminated separately from the high signal of the surrounding CSF.

Seven of the eight cases in group II had been operated on for a pituitary adenoma, including four prolactinomas and three chromophobe adenomas. Three of these patients had residual tumor present, and the remainder all showed evidence of empty sella on the postsurgical MR studies (Figs. 4 and 5). The final patient in group II had a very large cranio-pharyngioma preoperatively with no evidence of residual tumor and very little visible pituitary tissue on the follow-up MR (Fig. 6).

In group II, the ectopic bright signal was located in the hypothalamic median eminence in half the patients and in relation to the pituitary stalk in the other half. The ectopic bright signal was of small size (2×3 mm) when located in the infundibulum and was larger in every case when seen in the median eminence (Table 2). Again, a normally located posterior pituitary bright signal was not visible in any patient in group II. Signal intensity was equal to or brighter than that of a normal posterior pituitary bright signal in five of eight cases on T1-weighted images and appeared slightly less hyperintense than a normal posterior pituitary bright signal in one patient, in whom it was located in the median eminence, and in two patients in whom the ectopic location was in relation to the infundibulum. The change in signal intensity for the long TR dual-echo sequences were the same as for the group I patients.

Discussion

The intact hypothalamic hypophyseal tract affords passage of ADH and oxytocin elaborated in the supraoptic and para-

ventricular nuclei along the infundibular axons of these nuclei to the neurohypophysis for storage and later release into the circulation. Laboratory animal experiments as early as 1951 demonstrated that loss of the structural integrity of the hypothalamic hypophyseal tract resulted in formation of a "miniature posterior lobe" in the median eminence or proximal infundibular stem that resulted in functional preservation of hormonal production and release [1–3]. Later, reports in humans who were treated with hypophysectomy for metastatic disease confirmed the accumulation of neurosecretory material in the hypothalamic median eminence and proximal pituitary stalk at autopsy [4]. These findings were then reported again in other human studies [5, 6]. More recently, MR studies of pituitary dwarfs focused on the structural changes in the infundibulum in these patients with resultant loss of the normal MR posterior pituitary "bright spot" and its visualization in an ectopic location [7, 8].

We now report the MR observation of posterior pituitary ectopia in a group of patients with infundibular and/or posterior pituitary tissue compression or destruction as a consequence of intrasellar and/or parasellar tumor, surgery, and, in one case, pituitary hemorrhage. On the basis of previous work and the cases reported here, it is apparent that MR can afford in vivo visualization of the regeneration of the posterior pituitary in a wide variety of clinical circumstances in which there has been disruption of the integrity of the hypothalamic hypophyseal tract including traumatic, vascular, tumoral, inflammatory, and hemorrhagic causes.

The cause of the posterior pituitary bright signal, whether in its normal or an ectopic location, remains a subject of current controversy. The concept has been advanced that the polypeptide ADH-carrier protein neurophysin complex may be responsible for the T1 shortening that results in the posterior pituitary "bright spot" [9, 10]. The possible role of pituitary proliferation and intracellular lipid accumulation in response to stimulation of ADH/neurophysin release has more recently been discussed [8, 11, 12]. As early as 1962, convincing evidence in both normal, and hypophysectomized animal models demonstrated that significant depletion of neurosecretory material occurs in the posterior pituitary both in its usual location in the normal animal and in an ectopic

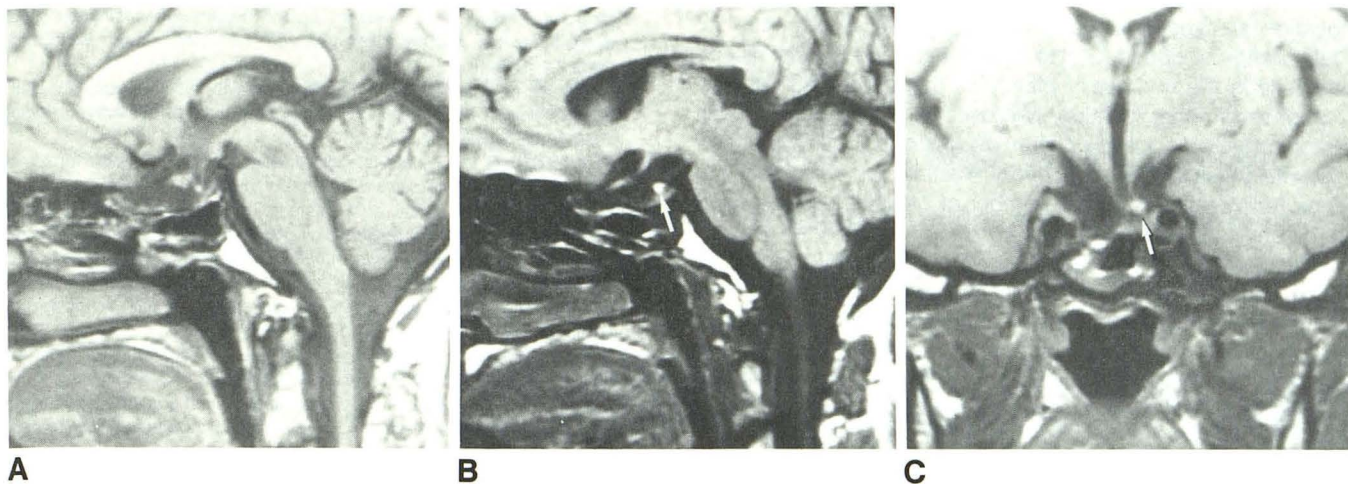


Fig. 4.—Group II, case 3: Postoperative empty sella after removal of small prolactinoma.
A–C, Sagittal section (A) shows empty sella and inferior displacement of third ventricle inside the sella. Note ectopic bright signal in displaced median eminence (arrow) in paramedian sagittal (B) and coronal (C) views.

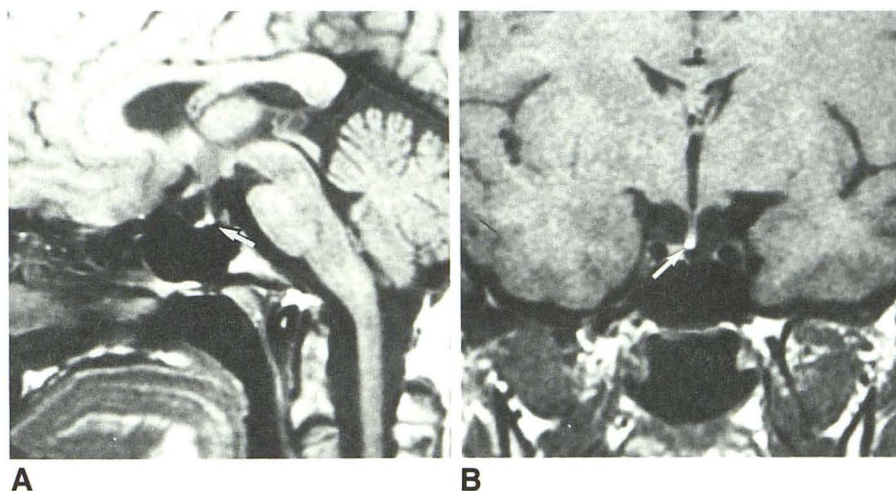


Fig. 5.—Group II, case 1: Postoperative empty sella.
A and B, Note bright signals in displaced infundibulum within the empty sella (arrow) in sagittal (A) and coronal (B) sections.

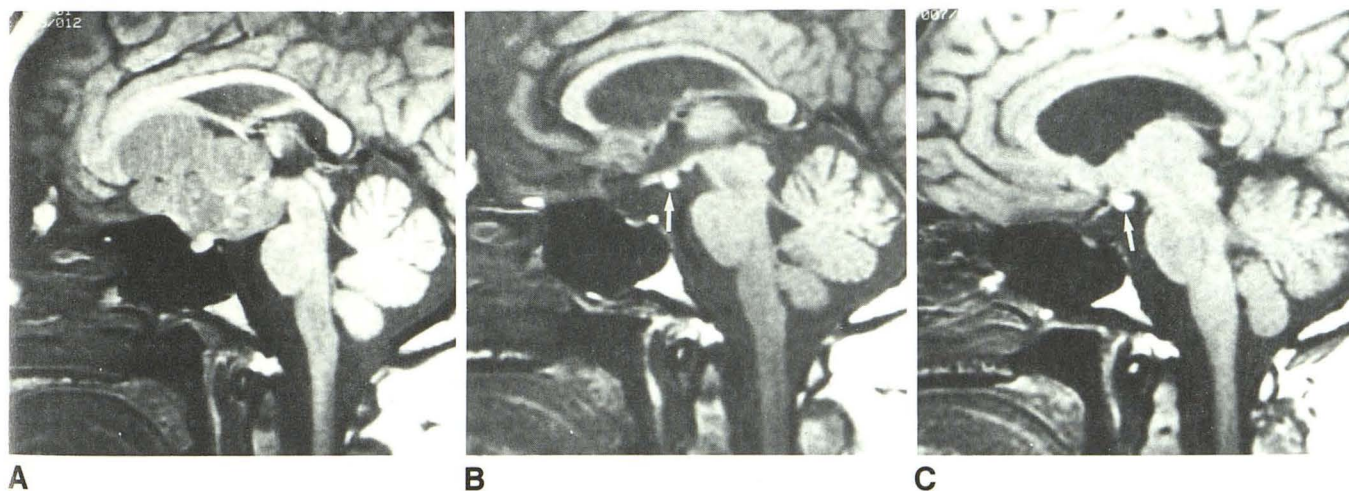


Fig. 6.—Group II, case 8: Craniopharyngioma.
A, Preoperative MR image of large suprasellar craniopharyngioma.
B and C, 9-month postoperative study with complete tumor removal. There is minimal residual pituitary tissue within a small sella. Note bright signal of ectopic posterior lobe in median eminence (arrow in B). A paramedian sagittal section shows exact size of large ectopic posterior lobe (arrow in C).

location in hypophysectomized animals as a response to dehydration or stress [3]. However, there was no significant increase in the number of pituicytes present as part of this response in either location. Microscopic examination of the ectopic miniature posterior lobes of animals showed abundant accumulations of neurosecretory material in relation to the axon terminals, but a small number of pituicytes were noted to be unusually large [3].

These data appear to favor the hypothesis of Fujisawa et al. [9, 10] that it is the ADH/neurophysin complex itself that may be predominately responsible for the bright signal. In this circumstance, dehydration, stress, and possibly other factors not yet elucidated, will affect, through the mechanism of stimulation of storage or release of ADH/neurophysin, the size and intensity of the ectopic or normal posterior lobe bright signal at any given time.

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