Ectopia of the Posterior Pituitary Gland or Lipoma?

I read, with great interest, the article [1] by Benshoff and Katz, "Ectopia of the Posterior Pituitary Gland as a Normal Variant: Assessment with MR Imaging," that appeared in the July/August 1990 issue of the AJNR. I am writing because of my concern about the confusion that this article may create. It is my opinion that the ectopic posterior pituitary gland does not occur as a normal variant as described by Benshoff and Katz.

In a series of 16 ectopic neurohypophyses and in a review of the world literature, I have not encountered an ectopic neurohypophysis occurring as a normal variant. However, I have seen several lipomas in the region of the tuber cinereum that look just like ectopic neurohypophyses. Such a lipoma is shown in Figure 6 in the article "Pathogenesis of Intracranial Lipoma: An MR Study in 42 Patients" [2] by Truwit and Barkovich. These lipomas are differentiated from ectopic neurohypophyses by the presence of a normal infundibulum, chemical-shift artifact, normal pituitary function, slightly more posterior location, and often more elongated shape. I think that the cases presented by Benshoff and Katz [1] are lipomas rather than ectopic neurohypophyses. In both cases, the patient had a normal infundibulum and normal pituitary function. A chemical-shift artifact was present in one case, and the lesion was located posterior to the position of the normal infundibulum in the second case. It is not significant that Benshoff and Katz did not see the normal posterior pituitary bright spot, as this is also difficult to detect in many normal subjects.

In summary, I think that the cases presented by Benshoff and Katz are lipomas rather than ectopic neurohypophyses. I do not think that ectopic neurohypophysis should be considered a normal variant.

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Reply to Dr. Abrahams

It is unfortunate that Dr. Abrahams is confused by our article [1], but we think that several of his basic assumptions about our cases are inaccurate.

Several points support our contention that our cases represent ectopic neurohypophyses rather than lipomas. First, several authors [2, 3] have reported visualizing hyperintense signal in the posterior pituitary gland in 90-100% of patients with normal pituitary glands. This has been our experience also. In fact, the absence of the bright signal has been the basis for the majority of the papers that describe ectopic pituitary tissue in diseased states. We think that absence of the hyperintense posterior pituitary signal is unusual and, in most cases, significant.

Second, the posterior pituitary signal is brighter than fat signal in the dorsum and clivus on proton-density and T2-weighted images [2, 3]. This is shown clearly in our two cases. Pure lipomas would have considerably lower signal on these sequences. The ectopic posterior...
pituitary tissue contained signal characteristics identical to what
would be expected normally within the sella.

Third, ectopic neurohypophyses occur in a distinctly different an-
atomic location than suprasellar lipomas do. Our cases show non-
descended posterior pituitary tissue along the hypothalamoneurohy-
pophyseal tract within the median eminence. We agree with Dr.
Abrahams that Figure 6 in the article by Truwit and Barkovich [4]
shows a lipoma. All of the suprasellar lipomas they reported occurred
posterior to the median eminence within the tuber cinereum and
mamillary body region. The anterior lesions in our cases 1 and 2 are
both anterior to the usual location of lipomas and are ectopic neuro-
hypophyses. The lack of hypogenesis of adjacent structures, often
seen with lipomas [4], was absent in our cases, also supporting the
presence of ectopic neurohypophyses. We do concede, however, on
the basis of the article by Truwit and Barkovich, that the posterior
lesion in our case 1 is probably a lipoma, which explains the chemical-
shift artifact present.

Finally, Dr. Abrahams’s review of the world literature had an
oversight. Brooks et al. [5] reported a case identical to ours in their
Figure 6: "Normal variant of posterior pituitary bright signal in median
eminence of hypothalamus." We agree with Brooks et al. that this
entity does occur.

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Commentary

I am pleased to have the opportunity to comment on the letters to
the editor by Drs. Abrahams and Berns, who disagree with the
findings of the paper [1] "Ectopia of the Posterior Pituitary Gland as
a Normal Variant: Assessment with MR Imaging," by Benshoff and
Katz. Dr. Berns also takes issue with the labeling of Figure 6 in an
article by Truwit and Barkovich [2], which he thinks is labeled incor-
rectly as a lipoma. I also have reviewed the reply of Drs. Benshoff
and Katz to the original letter of Dr. Abrahams. The essence of the
disagreement is whether the two cases described in the paper by
Benshoff and Katz [1] and Figure 6 in the paper by Truwit and Barkovich [2] are ectopic posterior pituitary glands or lipomas.

Benshoff and Katz contend that high-intensity nodules present in
the suprasellar and interpeduncular cisterns in two endocrinologically
normal persons are ectopic posterior lobes. They base their argument
on anatomic location and signal characteristics. They describe the
locations of the nodules as the superior portion of the infundibulum
in one case and in the median eminence in the other. They describe the
MR characteristics as bright on T1-weighted images and dimin-
ished, but remaining brighter than fat, on proton-density and T2-
weighted images. One case is described as showing chemical-shift
artifact. Specific comment is not made on the presence or absence
of chemical shift in the other case. The usual intrasellar high-intensity
signal of the posterior lobe was not visible in either case.

Although the actual chemical source of the signal in the posterior
lobe remains controversial, it is my opinion that relatively widespread
agreement exists on the following points:

1. Visualization of the high-signal intensity in the posterior lobe
indicates a hormonally intact hypothalamoneurohypophyseal sys-
tem. Nonvisualization of the high signal on MR occurs in 10% to
20% of control subjects. I think that nonvisualization probably is
due to the small size of the lobe or an eccentric location. Therefore,
I believe undue diagnostic significance should not be placed on
nonvisualization; more often than not, patients who do not have
this signal do not have abnormalities.

2. The high signal is due to a neurosecretory product stored in
the posterior lobe, whether that product be vasopressin, neurophysin,
the phospholipid vesicular membrane, or some combination
thereof. The high signal is always absent in patients who have
central diabetes insipidus.

3. The signal does not originate from lipid protons and therefore
does not have a chemical shift associated with it [3]. We have
shown that phospholipid vesicles have a similar signal to that observed
in the posterior lobe but that phospholipid signal origi-
nates from water protons interacting with the phospholipid mem-
brane, not from the lipid moiety itself [4]. Therefore, if a phospho-
lipid does play a role in the generation of the signal, a chemical
shift would not be expected [4].

4. Injury to the hypothalamoneurohypophyseal tract can cause an
accumulation of the neurosecretory product in a suprasellar loca-
tion. However, the "ectopic" location is always along the anatomic
path of that tract; that is, it must be in the pituitary stalk or the
median eminence.

On the basis of these anatomic and MR criteria, I cannot accept
the cases presented by Benshoff and Katz as ectopic posterior
lobes. In both cases, the sagittal images clearly show that the nodules
are posterior to the stalk and median eminence. I think that this also
is the case in Figure 6 in the Truwit and Barkovich paper [2] alluded
to by Dr. Berns. The case shown in Figure 6 is identified properly as
a lipoma and also falls into the 10–20% range of normal posterior
pituitary nodules in which the posterior pituitary bright spot is not
visualized on MR.

The coronal image in case 1 in the paper by Truwit and Barkovich
is misleading. Because of the obliquity of the stalk, the nodule appears
in the same coronal plane as the inferior portion of the stalk; the
anterior sloping of the stalk must be recognized to avoid this error.
In case 2, the lesion is in the floor of the third ventricle posterior to
the median eminence. As far as signal is concerned, chemical shift is
readily apparent in the first case, indicating the presence of signal
from fat protons and therefore further disqualifying this nodule from
consideration as an ectopic posterior lobe. It is difficult to be sure of
chemical shift in the second case. In this location, it is difficult to
detect chemical shift on T1-weighted images because fat becomes
superimposed on dark CSF; the summation of signal is poorly
demonstrated. I suspect that chemical shift is present on the proton-
density image. The fat signal is shifted anteriorly, becoming superim-
posed on suprasellar CSF, and it has been misinterpreted as high
signal from the lesion itself.

In summary, it is my opinion that Dr. Abrahams’s criticisms of the
article by Benshoff and Katz [1] are valid. Dr. Berns is correct in his
criticism of the cases presented by Katz and Benshoff but not in his
criticism of the paper by Truwit and Barkovich [2]. I do not think that
an ectopic neurohypophysis can be considered a normal variant. I
believe that the presence of an ectopic neurohypophysis always indicates an injury to the hypothalamoneurohypophyseal tract, although that injury may be temporally remote.

The two cases illustrated in the paper by Benshoff and Katz have imaging characteristics much more in keeping with lipomas than with ectopic neurohypophyses.

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Editor’s note.—See related article by Mark et al. on pages 529–532 in this issue.

Hyperintense Signal on MR Images of the Pituitary Gland

The source of the hyperintense signal in the posterior lobe of the pituitary gland on T1-weighted MR images is controversial. In 1987, my colleagues and I [1] hypothesized that the signal reflects the functional integrity of the hypothalameurohypophyseal system and that the probable source of the signal is neurosecretory granules. Subsequently, Kucharczyk’s group [2] proposed the hypothesis that lipid droplets within the pituicytes are the source of the hyperintense signal. In 1988, they reported an experimental study [3] and concluded that the lipid droplet theory or the neurosecretory granule theory was correct. In the paper “The Effect of Phospholipid Vesicles on the NMR Relaxation of Water: An Explanation for the MR Appearance of the Neurohypophysis” [4] in the July/August 1990 issue of the AJNR, they proposed a newer hypothesis: the phospholipid theory. The phospholipid theory states that the high concentration of the total phospholipid in the posterior lobe, existing mainly in the lipid droplets within the pituicytes and in the membranes of the axons and the neurosecretory granules, is the source of the hyperintense signal. I read their paper with great interest and found some problems.

In their discussion (p. 697 in [4]), they describe what they had done and mention the saline overload experiment in their 1988 paper [3]. In fact they did not do this experiment. In 1989, my colleagues and I [5] reported an experimental study that showed that the hyperintense signal in the posterior lobe disappeared after 2 weeks of administration of hypertonic saline solution, which stimulated the release of antidiuretic hormone from the posterior lobe. In their 1988 experiment, Kucharczyk et al. observed that the hyperintense signal increased in volume under the stimulation of release of antidiuretic hormone. The results of the two experiments were quite opposite. Which result is correct is the key to solving the controversy.

Previously, my colleagues and I [6] indicated several serious problems in the 1988 experiment. Here, I point out an additional one, which contradicts the phenomenon Kucharczyk et al. observed in the posterior lobe. An understanding of the mechanism of hormone release at the axon terminal is necessary for evaluation of their 1988 and 1990 experiments and our 1989 experiments.

Gial cells in the posterior lobe are called pituicytes. Historically, it was known that lipid droplets exist within the pituicytes, especially in the rat. At first, researchers thought that the pituicytes were glandular cells and that the lipid droplets were secretory granules. Those ideas were disproved by two new findings. One was the neurosecretory theory. The other was that two of the posterior lobe hormones, antidiuretic hormone and oxytocin, were found to be oligopeptides and not lipids. Some researchers [8, 9] concluded that the pituicytes were not related to the function of the posterior lobe. Still others [10, 11] proposed that excessive membranes of the neurosecretory granules at exocytosis are the source of the lipid droplets in the pituicytes (Fig. 1). They observed that the number of droplets increased when the function of the posterior lobe was stimulated. The number of neurosecretory granules decreased under such conditions. In their 1988 paper, Kucharczyk et al. [3] reported that they had observed a significant increase of both lipid droplets and neurosecretory granules in dehydration-stimulated animals. The neurosecretory granules should decrease under such conditions. Concerning the fate of the excessive membranes of the granules, another hypothesis is that the granules migrate in the axon up to the hypothalamus for reuse [12] (Fig. 1). Synthesis and release of antidiuretic hormone is thought to increase the excessive membranes at the axon terminals and consequently to increase the total amount of phospholipid in the posterior lobe because the membranes contain phospholipid. Thus, both the lipid droplet and the phospholipid theories do not explain the absence of the hyperintense signal observed in the posterior lobe in the hypertonic saline overload experiment.

In their results, Kucharczyk et al. [4] concluded that the pattern of signal intensities of liposome solutions was similar to that of the human posterior lobe. However, I believe that the liposome solutions had signal intensities markedly higher than those of the posterior lobes of volunteer subjects on proton-density and T2-weighted MR images (Figs. 3B and 3C in [4]). I think that it is incorrect to equate the liposome solutions and the human posterior lobe model. The significance of the 1990 experiment [4] is that the liposome, the size of which is similar to that of the neurosecretory granule in the posterior lobe, induced a remarkable shortening of relaxation times. I think that the mechanism observed in the 1990 experiment may explain the neurosecretory granule theory.

In 1987, my colleagues and I [13] first reported the ectopic posterior lobe in patients with pituitary dwarfism and hypothesized that the ectopic lobe is caused by stalk transection at birth because of the high correlation with abnormal delivery. In 1988, Kucharczyk’s