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Commentary

The Posterior Sella Bright Spot: A Perspective

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The high-intensity signal seen on MR images of the posterior pituitary has been the subject of numerous reports, including an important one in this issue of *AJNR* [1]. In 1984, we studied anatomic sections of the sella. A curvilinear pad of fat in the posterolateral recesses of the sella corresponded in location to the high-intensity signal on MR images [2]. Subsequent observations on the failure of the signal to diminish when fat-suppression techniques were used were inconsistent with the theory that lipid was the source of this signal (Mark L, Haughton V, Hendrix L, et al., unpublished data; Blatter D, Jobsz F, Morris J, et al. and Kim B, Kido D, Simon J, et al., personal communications). With better resolution MR imaging and thinner sections, it appeared that the high-intensity signal most likely was related to the posterior pituitary gland rather than to tissue outside the gland. A number of investigators deserve credit for improving our understanding of the high-intensity signal in the posterior lobe.

Dr. Fujisawa's group [3–5] was one of the first to suggest that the signal intensity might indicate the functional status of the hypothalamoneurohypophyseal axis and that the source of the signal was neurosecretory granules. These are intracellular vesicles that are transported from the hypothalamic nuclei to the posterior lobe of the pituitary gland. They contain the polypeptides vasopressin or oxytocin, or their precursors, preprohormone or prohormone. The polypeptides are linked to one of several carrier molecules called neurophysins. Whether the high-intensity signal is related to one of the six different neurophysins or to heavy- or light-molecular-weight pools of posterior lobe neurosecretory granules or to the hormones, prohormones, or preprohormones or to the membranes was not revealed. Why the high-intensity signal is not present in the hypothalamus or the infundibulum where neurosecretory granules are also present and the mechanism by which neurosecretory granules shortened T1 or increased signal in a short-TR image were not explained.

Another group [6] reported that the high-intensity signal in the posterior sella might be related to lipid in posterior lobe pituicytes. Twenty percent of the posterior lobe is made up of pituicytes, which are supportive cells analogous to glial cells. Using various techniques, including electron microscopy, investigators have found lipoid bodies within the cytoplasm of these pituicytes. Some investigators also have referred to these lipoid bodies as liposomes. The number of lipoid bodies and the functional state of the posterior pituitary gland were found to be directly related [6]. Although the presence of lipoid bodies within pituicytes clearly has been shown, the relationship of these lipoid bodies to the bright signal has not. The lack of chemical shift [7] and of suppression when fat-suppression techniques are used (unpublished data and personal communications [see previous citations]) associated with the high-intensity signal are not explained by the theory that lipid is the source of the high-signal intensity in the posterior pituitary gland. Although lipoid bodies, which may contain phospholipid, may play a minor role in the signal

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intensity of the posterior pituitary gland, recent work now suggests that phospholipids in membranes of axons or neurosecretory granules are the major factor.

Kucharczyk et al. [1] used experimentally produced liposomes to study the effect of phospholipids on surrounding water and were able to reproduce many of the MR features of the high-intensity signal of the posterior sella. They suggest that phospholipids in the membranes of the neurosecretory granules or axons produce the high-signal intensity of the posterior pituitary lobe. Their paper contributes significantly to the unraveling of sellar imaging and to MR imaging of phospholipid structures. It is hoped that further research will explain how phospholipids enhance the relaxation of water. Is it the phospholipid itself or polar glycoprotein complexes of the extracellular matrix that are associated with the membranes? What are the factors that make the phospholipid of the pituitary posterior lobe different from that seen elsewhere in cell membranes or myelin sheaths where no relaxation enhancement effect has been noted?

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