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Reversible Hyperintensity of the Anterior Pituitary Gland on T1-Weighted MR Images in a Patient Receiving Temporary Parenteral Nutrition

Kouichirou Okamoto, Jusuke Ito, Tetsuya Furusawa, Kunio Sakai, and Susumu Tokiguchi

Summary: We report a patient with severe anorexia nervosa, treated with temporary total parenteral nutrition (TPN), in whom reversible hyperintensity of the anterior pituitary gland was seen on T1-weighted MR images. The anterior pituitary was isointense with white matter before TPN therapy and became markedly hyperintense after 3 months of treatment. The intensity normalized after TPN therapy was discontinued. The transient hyperintensity was also seen in the basal ganglia and dorsal brain stem. We believe the hyperintensity of the anterior pituitary may be attributed to the TPN therapy.

It is well known that the basal ganglia may have a hyperintense signal on T1-weighted MR images in patients receiving total parenteral nutrition (TPN) and in those with chronic hepatic failure (1–4). Alterations in signal intensity of the basal ganglia in patients receiving TPN is reversible, and the cause of this change may be related to parenteral administration of manganese (2). In patients with chronic hepatic failure, hyperintensity on T1-weighted images is also observed in the mesencephalon surrounding the red nuclei and in the pituitary gland (4). Hyperintense signal of the anterior pituitary gland has not been described in patients receiving TPN. We report a patient with severe anorexia nervosa, treated with temporary TPN, in whom reversible hyperintense signal was seen in the anterior pituitary as well as the basal ganglia.

Case Report

An 18-year-old woman with a 3-year history of anorexia nervosa was admitted to our hospital because of frequent hypoglycemic attacks and severe emaciation. On admission, she was drowsy and was markedly dehydrated. Blood pressure was 78/44 mm Hg, pulse rate was 60 beats per minute, body temperature was 36.4°C, and blood sugar level was 40 mg/dL. Her weight was 21 kg for a height of 150 cm (at 15 years of age she had weighed 48 kg).

The patient could not take any nutrition orally, and continuous infusion of high-dose glucose was necessary to maintain the blood sugar level within the normal range. T1-weighted sagittal (600/15/2 [TR/TE/excitations]) MR images of the brain, obtained on a 1.5-T unit, showed complete loss of fat within the scalp and bone marrow of the skull. The signal intensity of the anterior pituitary and the basal ganglia was normal (Figs 1A and B).

TPN was started with the patient's consent, and consisted of 300 g dextrose per day, 40 g of mixed amino acids per day, 80 g of fat per week, and 2 mL of trace-element solution (which included iron, manganese, zinc, copper, and iodine) per day (Elemenmic, Hoechst Marion Roussel, Tokyo). After 3 months of TPN therapy, the patient's weight increased to 31 kg. She was alert and her mental status was stable, there were no neurologic deficits, her pituitary function was normal, and she could take food orally. TPN therapy was discontinued. Her weight increased gradually up to 38 kg. A repeat MR examination (Figs 1C and D) showed restoration of fat in the scalp and bone marrow. There was increased signal intensity from the dorsal brain stem in contrast to the belly of the pons, which was not seen on the initial MR examination. Marked hyperintensity was also seen in the anterior pituitary and basal ganglia on T1-weighted images obtained with identical parameters to the initial study. Although the increased signal intensity of the scalp and bone marrow was completely depressed on T1-weighted images obtained with the use of radio-frequency selective presaturation of the lipid peak, the signal intensity of the anterior pituitary remained markedly hyperintense.

Four months after cessation of TPN therapy, another MR examination showed that the signal intensity of the anterior pituitary and that of the basal ganglia, as well as that of the dorsal brain stem, had normalized (Fig 1E).

Discussion

The globus pallidus is isointense or hypointense relative to white matter on T1-weighted MR images in healthy subjects (1, 2). Hyperintense signal is seen in the basal ganglia bilaterally and symmetrically in patients receiving long-term TPN therapy or in persons with chronic hepatic failure (1–4). This increased signal intensity is homogeneous and is most pronounced in the medial segment of the globus pallidus (1). No corresponding alteration in signal inten-

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From the Department of Radiology, School of Medicine (K.O., T.F., K.S.), and the Department of Radiology, School of Dentistry (J.I., S.T.), Niigata University, Japan.

Address reprint requests to K. Okamoto, MD, Department of Radiology, Niigata University School of Medicine, 1–757 Asahimachi-dori, Niigata, 951-8510 Japan.

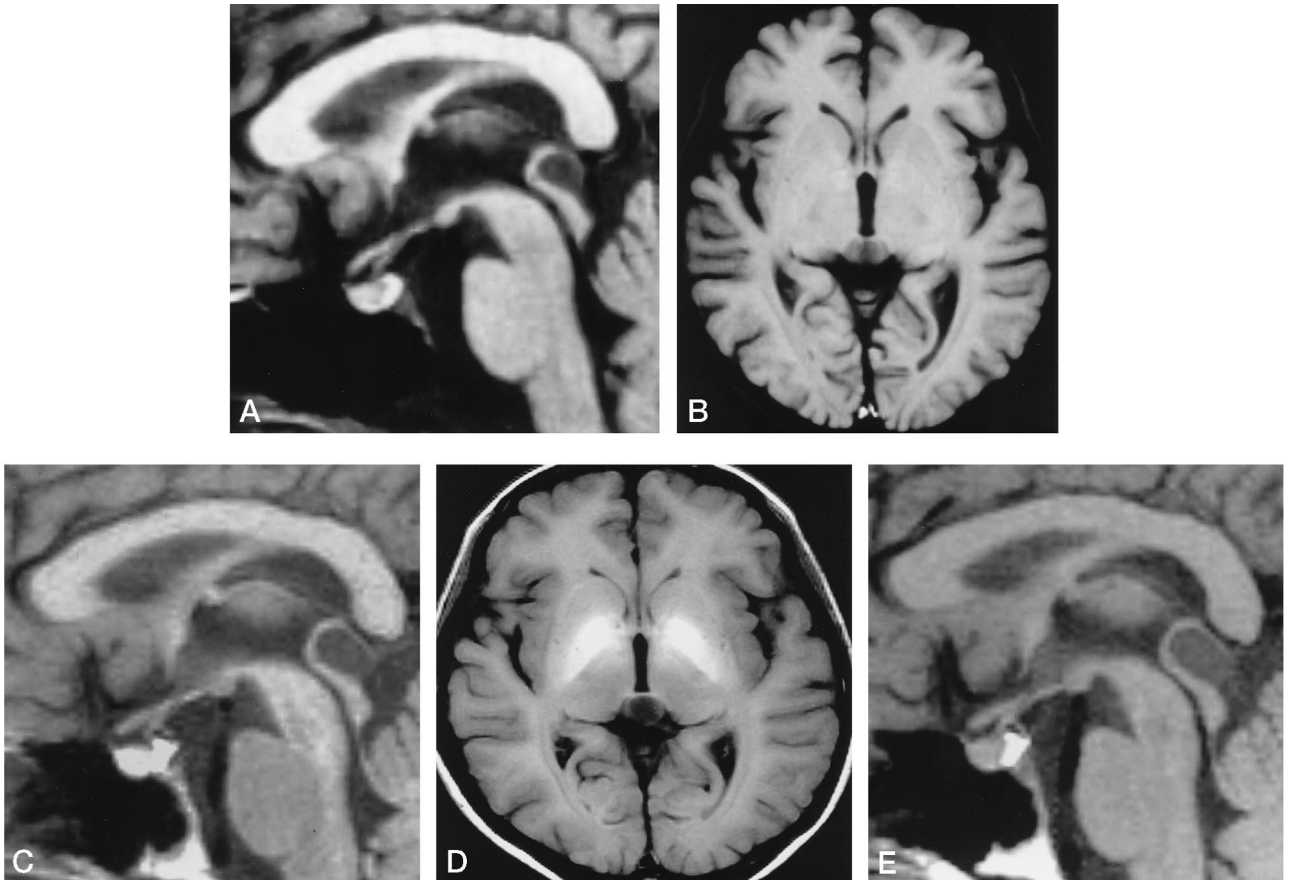

FIG 1. 18-year-old woman with frequent hypoglycemic attacks and severe emaciation consequent to 3-year history of anorexia nervosa.

A and *B*, T1-weighted MR images (600/15/2) acquired before the start of TPN therapy. Magnified midsagittal image (*A*) shows normal signal of the anterior pituitary and normal hyperintense signal of the posterior pituitary. Signal intensity of the brain stem is normal. A small pineal cyst is seen. Axial image shows the globus pallidus is isointense with white matter (*B*). Normal hyperintensity of fat is not seen in the scalp.

C and *D*, T1-weighted MR images (600/15/2) obtained after 3 months of TPN therapy. Magnified midsagittal image (*C*) shows the anterior pituitary is hyperintense. The signal intensity of the anterior pituitary is similar to that of the posterior pituitary. Increased signal intensity of the dorsal brain stem in contrast to the belly of the pons is also seen. Axial image at level of the globus pallidus (*D*) shows bilateral and symmetrical hyperintensity without mass effect in the globi pallidus. The fat in the scalp is of normal hyperintensity.

E, Magnified T1-weighted midsagittal MR image (600/15/2) obtained 4 months after cessation of TPN therapy shows the anterior and posterior pituitary lobes are of normal signal intensity. The signal intensity of the dorsal brain stem is also normal.

sity is seen on T2-weighted images (1–4). Although the cause of the increased signal intensity on T1-weighted images remains undetermined in patients with chronic hepatic failure (3, 4), deposition of an as-yet unidentified paramagnetic substance that bypasses the detoxification mechanisms of the liver, owing to portacaval shunting or hepatocyte dysfunction or to altered intracellular water relaxation associated with the proliferation of astrocytic cytoplasmic organelles, is postulated as the likely mechanism for this MR manifestation (4).

In one reported study of a patient receiving long-term TPN therapy, the marked hyperintensity of the globus pallidus on T1-weighted images regressed after cessation of parenteral manganese administration; the changes in signal intensity in the basal ganglia were therefore attributed to deposition of manganese and/or to reactive changes stemming from the presence of manganese (2). Manganese is one of the trace elements administered to patients undergoing TPN therapy and is a paramagnetic transition metal leading to shortening of T1 relaxation resulting in increased signal intensity on T1-weighted images (1, 2, 5). When manganese is ingested orally, highly effective regulatory mechanisms prevent excessive absorption or retention, and there is no accumulation within the brain under normal conditions (1, 5–7). When manganese is administered parenterally, however, these regulatory mechanisms are bypassed and its deposition occurs in the brain in a specific pattern (1, 5). The site of the earliest and most pronounced deposition of manganese is the globus pallidus, especially its medial segment (1, 5, 8). Other structures that have appeared hyperintense on T1-weighted images after parenteral manganese administration in monkeys include the caudate nucleus, putamen, substantia nigra, subthalamic nucleus, and ventromedial hypothalamus (5). In patients receiving long-term TPN therapy, hyperintensity has been observed in the subthalamic nucleus, variable portions of the lateral

thalamic nuclei, and internal capsules on T1-weighted images (1).

In addition to the globus pallidus, the pituitary gland also has a particular affinity for manganese in monkeys (5). Although a hyperintense anterior pituitary lobe has been observed in patients with chronic hepatic failure, it has not been described in patients receiving TPN therapy (4). In our patient, the anterior pituitary lobe and basal ganglia, as well as the dorsal brain stem, showed normal signal intensity on the initial MR examination. These regions became hyperintense on a repeat MR study obtained 3 months after the start of TPN therapy. Although fat was restored in the scalp and bone marrow, the hyperintense signal of the anterior pituitary did not result from fat deposition, because this signal intensity remained on fat-suppressed T1-weighted images. The hyperintense signal in the anterior pituitary, globus pallidus, and dorsal brain stem normalized after cessation of TPN therapy. This observation suggests that the cause of the hyperintensity in these regions was related to TPN therapy, and most likely to deposition of manganese.

Clinical manifestations and neuropathologic changes of acute or chronic intoxication from inhaled manganese have been described (1, 7–11). Parenteral administration is not an important source of regional accumulation of manganese (5). No neurologic symptoms related to manganese intoxication have been reported in patients receiving long-term TPN therapy (1, 2). Although protein malnutrition has been shown to promote the neurotoxic effects of manganese (12), our patient had no signs of manganese intoxication, and her pituitary function was normal.

Conclusion

Our observation supports the fact that the pituitary gland and the globus pallidus have a selective affinity for manganese and that the concentration of manganese required to produce visible T1 shortening on MR images is lower than the threshold necessary to result in clinical symptoms (1). Hyperintensity of the anterior pituitary lobe in patients receiving TPN therapy is reversible, like that of the globus pallidus, and should not be confused with other pathologic conditions, such as Rathke cleft cyst, dermoid, or pituitary hemorrhage.

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