Manganese in Total Parenteral Nutrition

58

Daniel Thomas Ginat

58.1 Uses

Manganese is a trace element included in total parenteral nutrition therapy preparations in order to prevent development of deficiency symptoms, such as nausea, vomiting, weight loss, dermatitis, and changes in growth and color of hair. Manganese intoxication can also result from intravenous methcathinone (ephedrone) abuse.

58.2 Mechanism

When manganese is administered parenterally, the normal regulatory mechanisms that prevent excess absorption are bypassed, leading to its deposition in specific locations within the brain. Manganese is a paramagnetic transition metal with T1 shortening effects on MRI.

58.3 Imaging Findings

Hyperintense signal on T1-weighted MRI sequences is frequently observed in the basal ganglia bilaterally and symmetrically in patients receiving long-term total parenteral nutrition therapy. This increased signal intensity is homogeneous and is most pronounced in the globus pallidus (Fig. 58.1). In addition, intrinsic high signal on T1-weighted sequences can also be found in the cerebral peduncles, dorsal brainstem, and anterior pituitary. The hyperintense signal normalizes after cessation of TPN therapy. Transcranial ultrasound can be TCS sensitive in detecting the trace metal accumulation in the lenticular nuclei, which appears as abnormal hyperechogenicity.

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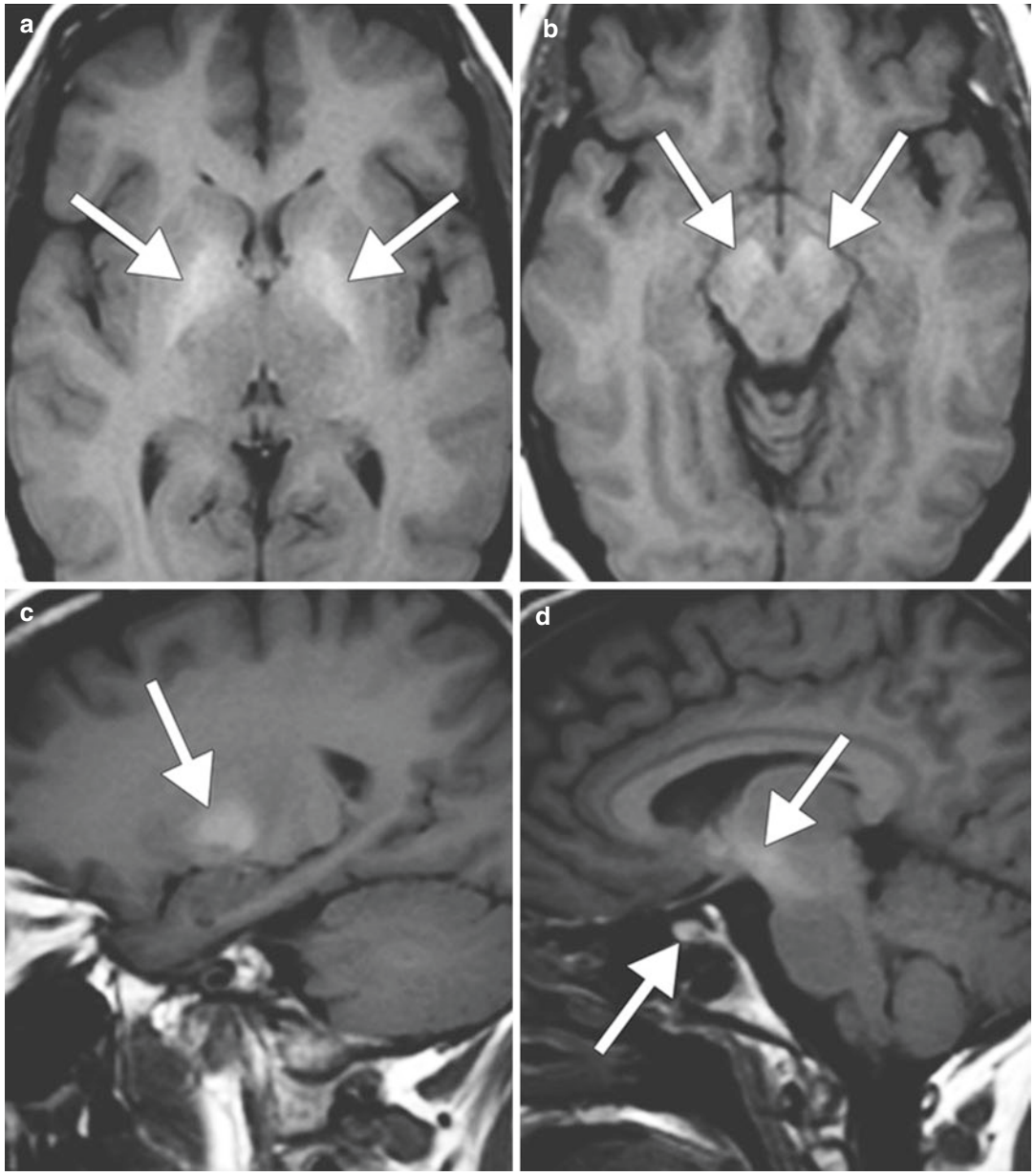

Fig. 58.1 Manganese toxicity in total parenteral nutrition. Axial (**a**, **b**) and sagittal (**c**, **d**) T1-weighted MRI images show patchy hyperintensity within the bilateral basal ganglia, cerebral peduncles, and anterior pituitary (*arrows*)

58.4 Differential Diagnosis

Besides excess total parenteral nutrition, a variety of conditions can produce T1 shortening in the basal ganglia, including hepatic encephalopathy (refer to Chap. 2), hypoxic ischemic injury (refer to Chaps. 3, 35, and 61), hypertensive hemorrhage, mineralizing angiopathy (refer to Chap. 19), hypoparathyroidism, pseudohypoparathyroidism, Fahr disease (refer to Chap. 19), Cockayne syndrome (Fig. 58.2), neurofibromatosis type 1

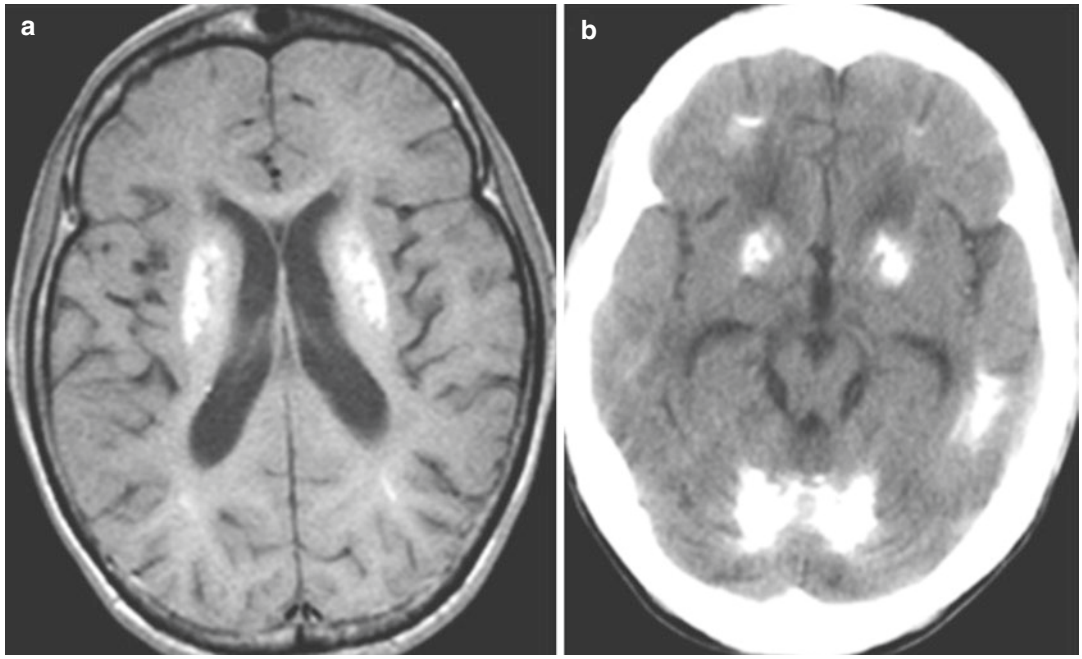

Fig. 58.2 Cockayne syndrome. Axial T1-weighted MRI (**a**) shows hyperintensity within the bilateral basal ganglia. Axial CT image (**b**) shows extensive cerebral calcifications

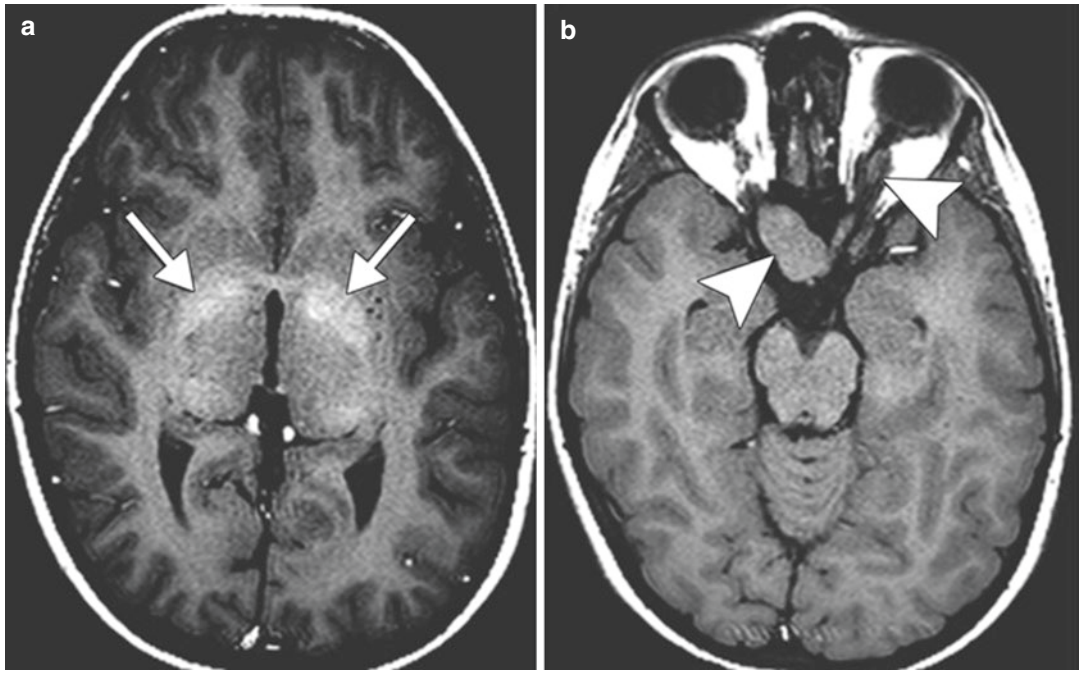

Fig. 58.3 Neurofibromatosis type 1. Axial T1-weighted MR image (**a**) shows irregular areas of high signal in the bilateral basal ganglia (*arrows*), bridging across the anterior commissure. Axial T1-weighted MR image (**b**) shows bilateral optic gliomas (*arrowheads*)

(Fig. 58.3), and neurodegenerative Langerhans cell histiocytosis (Fig. 58.4), among other conditions. In addition to clinical history, other imaging findings may help differentiate these conditions from the effects of manganese deposition. Ultimately, the history of hyperalimentation in manganese toxicity versus the presence of finding beyond the basal ganglia in the other conditions is key for elucidating the diagnosis.

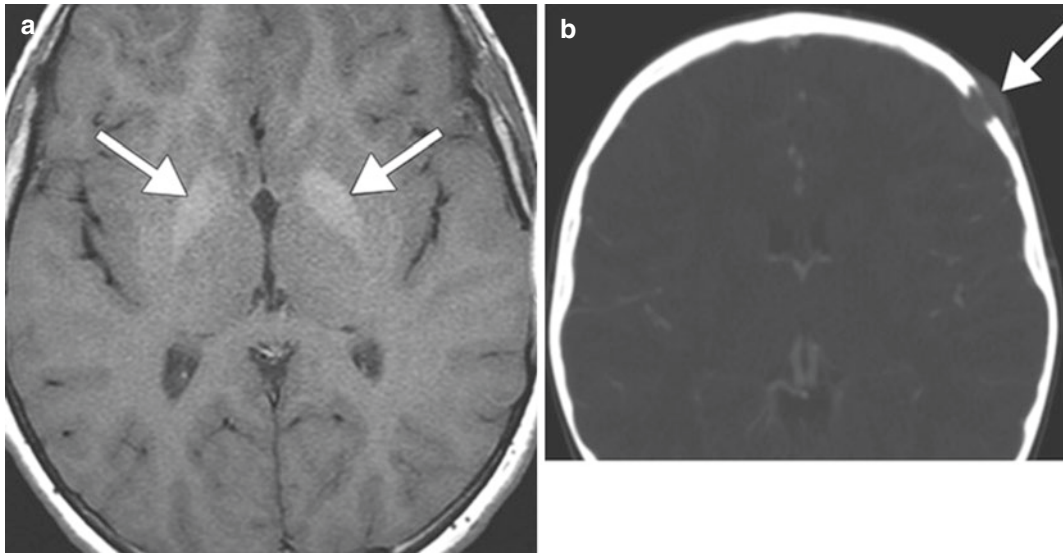

Fig. 58.4 Neurodegenerative Langerhans cell histiocytosis. Axial T1-weighted MRI (**a**) shows bilateral symmetric T1 hyperintensity within the globi pallidi (*arrows*). Axial CT image (**b**) shows an expansile lytic lesion within the left frontal bone (*arrow*)

Suggested Reading

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