Wernicke encephalopathy after bariatric surgery: Losing more than just weight

D. Foster, DO; M. Falah, MD; N. Kadom, MD; and R. Mandler, MD

Bariatric surgery is a frequent treatment for obesity. Neurologic complications after surgery include encephalopathy, behavioral abnormalities, seizures, cranial nerve palsies, ataxia, plexopathy, myelopathy, polyneuropathy, mononeuropathy (carpal tunnel syndrome, meralgia paresthetica), compartment syndrome, neuropathy, and myopathy. In particular, Wernicke encephalopathy, caused by vitamin B1 deficiency, can result in permanent neurologic deficit.[1,2] We report unusual clinical and imaging findings in a postgastric bypass patient with Wernicke encephalopathy.

Case report. A 35-year-old woman underwent gastric bypass surgery for obesity. Subsequently, she reported anorexia, nausea, vomiting, and generalized fatigue, resulting in hospitalization. The patient continued to develop progressive hearing loss, psychomotor slowing, apathy, forgetfulness, ataxia, and bilateral hand paresthesias. In the 12th postoperative week, she had lost 40 lbs, was lethargic and confused, and had difficulty walking. She was awake but not attentive, speech was fluent, and comprehension was decreased. Pupils were equal and reactive to light, extraocular movements were intact, there was no nystagmus, and hearing was diminished. Strength was 3/5 in the lower extremities and normal in upper extremities, vibratory sense was diminished in feet, deep tendon reflexes were absent, plantar reflexes were down-going, and gait was wide-based. Laboratory tests were normal for blood count, thyroid function, vitamin B_{12}, and CSF. Abnormal results included a slight elevation in liver enzymes, high serum glucose (163 mg/dL), and low serum potassium (2.6 MEq/L). EEG and head CT were normal.

The patient's mental status continued to decline despite treatment for dehydration. Upon admission to our hospital, the heart rate was 125 beats/min; she opened her eyes to nail bed pressure but followed no commands and was nonverbal. The pupils were round and fixed at 3 mm, oculocephalic and deep tendon reflexes were absent, plantar reflexes were down-going, and general muscle tone was flaccid without spontaneous movements or withdrawal to painful stimuli. Abnormal test results were as follows: serum glucose (256 mg/dL), CSF protein (90 mg/dL), and diffuse slowing on EEG. Pretreatment red blood cell transketolase and serum thiamine levels were not down. The first MRI showed bilateral symmetric hyperintense signal on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images at the floor of the fourth ventricle, periaqueductal gray matter, the medial portions of both thalami, and the premotor and motor cortices (figure, A through C). All T2 hyperintense regions demonstrated contrast enhancement. Restricted diffusion was seen in the same regions on diffusion-weighted imaging (DWI) and apparent diffusion coefficient maps.

Initially, thiamine 100 mg IV showed no clinical improvement. Subsequently, high-dose thiamine (100 mg IV every 8 hours) was administered. On the 4th hospital day, the patient became awake, pupillary reflexes and extraocular muscles returned to normal, and horizontal and gaze-evoked nystagmus became apparent. However, the patient remained areflexic and quadriparetic and unable to converse or follow commands.

Follow-up brain MRI 11 days after thiamine repletion showed interval improvement (see the figure, D through F) with interval decrease of abnormal signal on T2-weighted and FLAIR images. There was less contrast enhancement. The areas of restricted diffusion were decreased on DWI. As a major change, there was increased signal on precontrast T1-weighted images in the premotor and motor cortices, likely representing petechial hemorrhages.

Discussion. The neurologic complications after bariatric bypass surgery are diverse. Wernicke encephalopathy is a well-defined syndrome, but difficult to identify in the absence of the classic triad of oculomotor abnormalities, ataxia, and confusion, as in 20% of patients.[3] When a patient presents with unusual symptoms (in our case with progressive hearing loss, most likely secondary to thalamic involvement), then blood work (red blood cell transketolase levels) and MRI become helpful tools in making the diagnosis.[4] Typical findings on MRI correspond with the location of neuropathology: increased T2 or proton density signal around the third ventricle, the periaqueductal midbrain, and dorsomedial thalami. Contrast enhancement and symmetric cortical involvement, as were seen in our patient, have been less frequently observed.[5] A high index of suspicion for Wernicke encephalopathy is required in surgically treated obese patients.

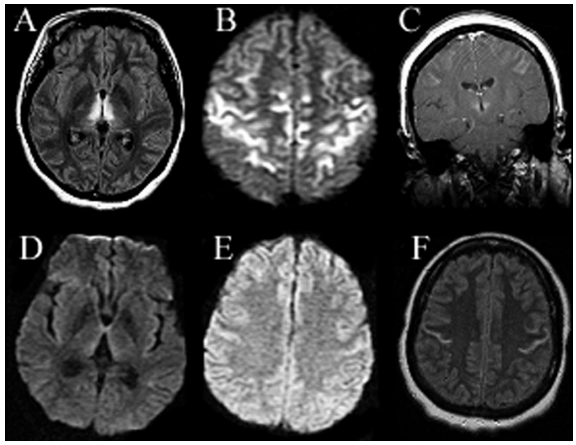

Figure. Pretreatment (A through C) and posttreatment (D through F) treatment MRI. (A) Axial fluid-attenuated inversion recovery image, showing bilateral symmetric hyperintense lesions of the periaqueductal gray matter and medial portions of both thalami. (B) Diffusion-weighted image, showing restricted diffusion symmetrically in the motor and premotor cortices. (C) Postcontrast coronal T1-weighted image, with thalamic and cortical contrast enhancement bilaterally. (D and E) Near resolution of lesions on diffusion-weighted imaging after thiamine treatment. (F) Precontrast axial T1-weighted MRI, showing petechial cortical hemorrhages bilaterally.

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MuSK Ab described in seropositive MG sera found to be Ab to alkaline phosphatase

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We previously reported finding autoantibodies to muscle-specific receptor tyrosine kinase (MuSK) in 41% of patients with acetylcholine receptor antibody (AChR Ab)–seronegative MG (SNMG),[1] as in other reports.[2-4] In addition, and in contrast with previous data,[2-4] we found a 10.5% positivity rate of MuSK Abs in AChR Ab–seropositive MG (SPMG) patients in the RIA using a recombinant MuSK fusion protein with placental alkaline phosphatase (AP). In this study, we tested MG sera for MuSK Ab in an RIA that uses His-Tag human recombinant MuSK (rMuSK) and for AP Ab using placental AP. The data presented in our earlier article on MuSK Abs in SPMG sera proved to be incorrect, and what we initially believed were MuSK Abs are Abs to AP.

Methods. Serum samples were obtained from 41 SNMG patients with severe generalized MG and from 229 SPMG patients (50 with, 179 without thymoma). The control populations comprised 70 healthy subjects and 91 patients with other neurologic or immunologic diseases.

His-Tag MuSK protein was constructed from the extracellular domain of human MuSK, including four immunoglobulin-like domains and a cysteine-rich segment, but lacked the transmembrane region and the kinase domain, and expressed in secreted form from COS 7 cells. This rMuSK protein was purified in a histidine affinity column, then labeled with $Na^{125}I$. MuSK Ab was detected by an RIA. In brief, 5 μL of each serum sample was incubated with ^{125}I-rMuSK (40,000 cpm) overnight at 4 °C, after which 50 μL of anti-human IgG was added, and the samples were incubated for another 2 hours at room temperature. After two washes of the pellets with saline, radioactivity was counted. Human placental AP was purchased (Calzyme Laboratories, CA) and further purified (95% purity by sodium dodecyl sulfate–polyacrylamide gel electrophoresis) by affinity chromatography on anti-AP monoclonal antibody coupled to agarose (Sigma, St. Louis, MO). AP Ab was assayed in 5 μL of serum samples with ^{125}I-labeled AP.

Results. The cut-off value (0.01 n*M*) calculated from the mean + 3 SD of the values for the healthy subjects showed no MuSK Ab present in the healthy subjects or patients with other neurologic or immunologic diseases. MuSK Ab was present in 13 (29%) of the 45 SNMG patients but not in any of the 229 patients with SPMG, including patients who were positive in the ^{125}I-rMuSK-AP assay.[1] The mean MuSK Ab value for SNMG was 45.0 n*M* (n = 13; 8.4 to 114.6 n*M*) (figure). Decreased binding of ^{125}I-rMuSK in different dilutions of all sera studied was dose dependent.

Next, we assayed the same samples in a ^{125}I-labeled AP assay. The cut-off value (0.01 n*M*) was calculated from the mean + 3 SD of the values for the healthy subjects. AP Ab was detected in 8.8% (20/229) of the SPMG patients, accounting for 8.9% (16/179) of those without and 8% (4/50) with thymoma. SPMG sera that were positive in the ^{125}I-rMuSK AP assay also were positive in the ^{125}I-AP assay. In contrast, no AP Ab was detected in any SNMG patients, non-MG patients, or healthy subjects. The mean AP Ab value for SPMG was 0.236 n*M* (n = 20; 0.017 to 0.8 n*M*). We also confirmed immunologically that AP Ab-positive sera had antibodies directed against AP by western blot analysis (data not shown).

Discussion. We have repeated the prior assay[1] using both a His-Tag MuSK and AP as antigens. We detected MuSK Abs in 29% of SNMG patients but not in any of SPMG patients. AP Abs were found in 8.8% of SPMG patients but not in any of SNMG patients. As a result, we found that the antibody with a 10% positivity rate detected in SPMG by the prior assay were AP Abs, not MuSK Abs, and thereby correct our previous report.[1] In addition, we found that this AP Ab is present only in SPMG patients, not in SNMG patients, non-MG patients, or healthy subjects. Our results may also add AP as a novel antigen to the variety of autoantigens found in MG patients without further evaluation of its pathogenic role in MG.

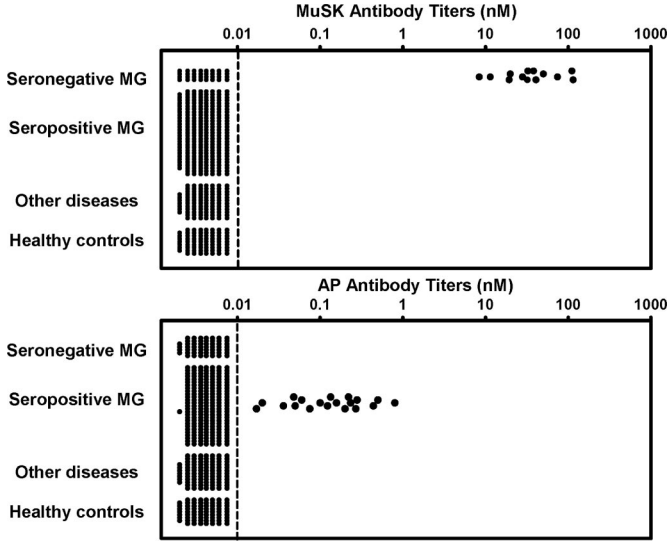

Figure. MuSK and alkaline phosphatase (AP) antibody titers detected by ^{125}I-rMuSK and ^{125}I-human placental AP assays. Antibody titers were measured by RIA in 45 patients with seronegative MG, 229 patients with seropositive MG (179 without, 50 with thymoma), 91 patients with other neurologic or immunologic diseases (5 Lambert–Eaton myasthenic syndrome, 6 polymyositis, 10 muscular dystrophy, 15 thyroiditis, 10 type 1 diabetes mellitus, 5 rheumatoid arthritis, 10 multiple sclerosis, 5 spinal progressive muscular atrophy, 5 chronic inflammatory demyelinating polyneuropathy, 10 ALS, 10 patients with epilepsy), and 70 healthy subjects. The broken line denotes the cutoff value (0.01 nM) calculated from the mean + 3 SD of the values for the 70 healthy sera.

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A trochlear stroke

Achraf A. Makki, MD, MS; and Nancy J. Newman, MD

A 51 year-old right-handed male physician had diplopia. His past medical history included refractive error, hypertension, dyslipidemia, and reactive hypoglycemia. He was involved in a motor vehicle accident in 1996, with cervical whiplash, but no associated head trauma or loss of consciousness.

In 1996, the same year of his accident, he started experiencing binocular, vertical diplopia, although he did not relate it clearly to the accident. He also noted some associated dizziness, but no definite oscillopsia and no other neurologic complaints.

During ophthalmologic evaluation for refractive surgery in 1997, vertical nystagmus was noted. MRI in 1997 and again in 1998 were interpreted as normal.

Neuro-ophthalmologic evaluation in 2004 revealed a left head tilt, 70% of normal depression of the right eye when adducted, right hypertropia that decreased in up-gaze, increased in down-gaze, and increased with right head tilt, and a variable up-beat nystagmus in primary gaze, which worsened on up-gaze. His neurologic examination was otherwise normal. Examination was diagnostic of a right superior oblique palsy, with associated up-beat nystagmus.

Review of his previous MRI revealed an abnormality in the region of the left fourth (trochlear) cranial nerve nucleus extending into the adjacent superior cerebellar peduncle (SCP). The lesion was dark (hypointense) on T1, bright (hyperintense) on T2, with no enhancement and no mass effect, most suggestive of an old microvascular infarction (figure, A and B). Examination of the coronal T1-weighted MRI of the orbits after gadolinium administration revealed substantial asymmetry in the size of the superior oblique muscles, with the right smaller and fibrotic (see figure, C), as well as increased signal intensity on T1-weighted images, all consistent with denervation atrophy.

The trochlear nucleus is situated in the central gray matter of the midbrain, close to the median plane, in the vicinity of the medial longitudinal fasciculus (MLF) and decussating fibers of the SCP. The emerging fibers of the trochlear nucleus pass laterally and posteriorly round the central gray matter, decussate in the superior medullary velum, and leave the midbrain below the inferior colliculi. Hence the innervation of the superior oblique muscle is crossed.[1-5]

The trochlear nucleus integrates inputs from vestibular centers via the MLF, from burst cells of the rostral interstitial nucleus of the MLF sending saccadic commands, and from the interstitial nucleus of Cajal. Up-beat nystagmus is not exquisitely localizing but can occur from lesions of the medulla as well as from lesions involving the SCP (brachium conjunctivum).[3,6] The explanation for up-beat nystagmus in SCP lesions relates to the anatomy of the vestibulo-ocular reflex in which the excitatory projections from the anterior semicircular canal (which generate upward eye movements) pass through the SCP, the crossing ventral tegmental tract (CVTT), and MLF.[3] Hence, a SCP lesion might be expected to cause a vertical vestibular imbalance in which the eyes drift down (unopposed posterior canals) and corrective quick phases produce up-beat nystagmus. It is also possible that some vertical gaze-holding or smooth-pursuit signals pass up the SCP from the neural integrator or y-group.[3] In our case, disruption of the adjacent left SCP most likely underlies the observed up-beat nystagmus, although we cannot rule out involvement of the CVTT and MLF.

The blood supply of the trochlear nucleus is through the paramedian branches of the basilar bifurcation.[1,2] These very fine branches are very susceptible to shear injury from trauma, the most likely mechanism for our patient's lesion.

The earliest MRI abnormalities seen with muscle denervation may appear by 4 days after injury, and are typically decreased intensity on T1-weighted images with variable enhancement with gadolinium.[7] After several months, increased signal intensity on T1-weighted images and muscle atrophy is seen, as in our case of long-standing trochlear nuclear injury.

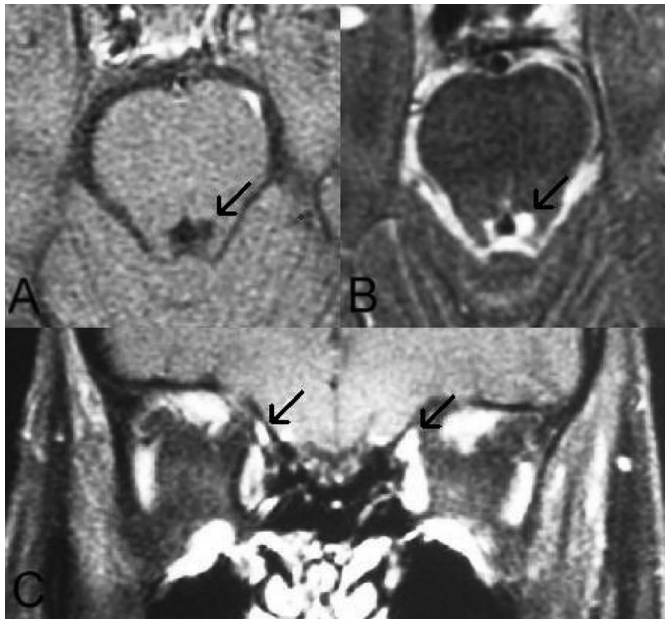

Figure. (A) Axial T1-weighted MRI of the brainstem showing a hypointense lesion (arrow) in the region of the left trochlear nerve nucleus. (B) Same lesion (arrow) is hyperintense on T2-weighted images. (C) Coronal T1-weighted MRI of the orbits after gadolinium administration showing substantial asymmetry in the size of the superior oblique muscles (arrows), consistent with denervation atrophy of the right superior oblique muscle.

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Spontaneous bilateral carotid artery dissection and posterior reversible encephalopathy syndrome

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Spontaneous bilateral internal carotid artery dissection (sCAD) is rare. About 5 to 10% of carotid artery dissections are bilateral, but they are often revealed by unilateral symptoms, including the following: headache (up to 90%), neck pain (20%), Horner syndrome (50%), pulsatile tinnitus, and cranial nerve palsies (12%).[1,2] Dissection may be caused by extrinsic factors, such as trauma or hypertension, or by intrinsic factors, such as primary disease of the arterial wall seen in rare connective tissue disorders such as Ehlers-Danlos or Marfan syndrome.[1,3,4,5] However, the etiology of the arterial wall dysfunction that leads to sCAD in the majority of patients is unclear. There is evidence that sCAD may be caused by a transient arteriopathy.[4] The development of a transient arteriopathy may make the arterial wall of large and small vessels susceptible to triggering factors for dissection and possibly to arterial wall dysfunction that can be seen in disorders like posterior reversible encephalopathy syndrome (PRES). Clinical manifestations of PRES include headache, decreased alertness, altered mental functioning, seizures, and visual disturbance.[6] A recent study has shown that pathologic impairment of the stability of the arterial wall can be observed in other arteries, particularly the superficial temporal artery, in patients with sCAD in close temporal relation to the event.[4] We report a patient who had spontaneous bilateral carotid artery dissection and PRES.

Case report. A 44-year-old woman with a history of relapsing-remitting multiple sclerosis (MS) presented with a 1-day history of headaches, blurry vision, confusion, and a single generalized tonic-clonic seizure soon after being treated with one dose of 1 g IV methylprednisolone for a presumed MS exacerbation. The blood pressure in the emergency room was elevated at 148 to 170/78 to 96. Her normal blood pressure was 110/60. There was no prior history of hypertension. Head CT in the emergency department was normal. Brain MRI/MRA revealed findings consistent with PRES and bilateral carotid artery dissections (figure, A through C). Sedimentation rate and workup for a connective tissue disorder was normal. Her symptoms resolved completely over a period of 48 to 72 hours. She was treated with warfarin. A follow-up MRA obtained 3 months after the event revealed no evidence of dissection (figure, D).

Discussion. We describe a patient with bilateral sCAD and PRES. To our knowledge, no prior case of bilateral sCAD and PRES has been reported and there have been no case reports to link MS with sCAD or PRES. It is plausible that sCAD and development of PRES are related to arterial wall instability and dysfunction that can be caused by a generalized arteriopathy. This generalized susceptibility of the vasculature likely manifested itself clinically in this patient as bilateral sCAD and PRES. A recent study showed that microbleeds are found close to the tunica media/tunica adventitia junction, which implies that hemorrhage in sCAD is not the result of an intimal tear, but of hemorrhage in the wall itself from possibly the vasa vasorum.[4] The instability of the arterial wall can contribute to the development of PRES. The pathophysiology of PRES is thought to be related to a leaky blood–brain barrier, which is the result of an alteration of vascular reactivity related to profound endothelial dysfunction.[6] Endothelial health and function is dependent on signals from healthy, normally functioning smooth muscle cells (SMC) in the arterial wall.[7] It has also been reported that there is a preponderance of immature and vacuolated SMCs in the study population rather than the contractile SMCs typical for a healthy artery.[4] As a result, our patient probably developed PRES because of endothelial dysfunction, which may be the result of the changes in the arterial wall caused by the dysfunction of the SMCs in the related transient generalized arteriopathy. It is also reasonable to conclude that the changes in the vessels and the elevated blood pressure created an environment conducive to the development of PRES. The fact that the patient was given methylprednisolone does not contribute to the complexity of the case. Steroids are a potent vasoconstrictor used to treat this syndrome. Given that she developed PRES in spite of the treatment suggests that another mechanism is the cause.

Although the pathogenesis of spontaneous carotid artery dissection and posterior reversible encephalopathy syndrome is unclear, in this case it is logical to conclude that the development of a transient arteriopathy affecting the stability and function of the arterial wall predisposed this patient to bilateral spontaneous carotid artery dissections and PRES.

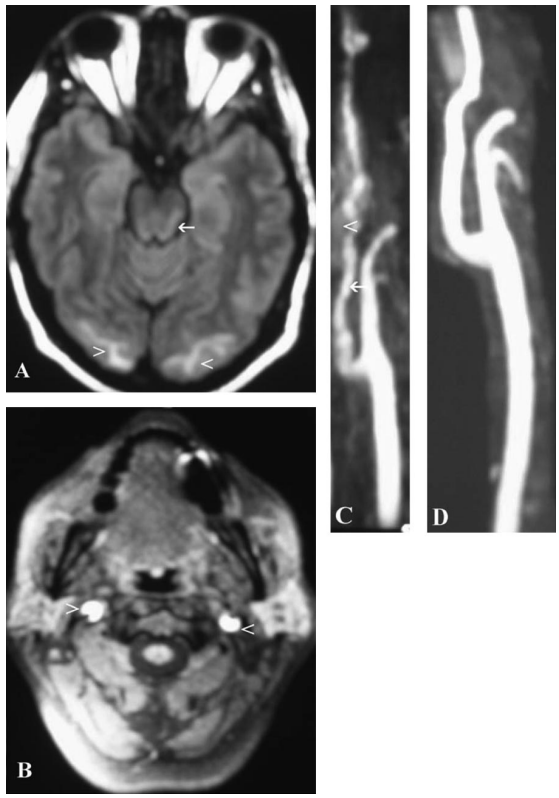

Figure. (A) MRI FLAIR image demonstrating posterior reversible encephalopathy syndrome in occipital lobes (arrowheads) with active MS plaques in pons (arrow). (B) MRI T1 fat suppressed image showing bilateral carotid artery dissections with bright signal in false lumen (arrowheads). (C) MRA of left internal carotid artery with false lumen (arrowhead) and true lumen (arrow). (D) Follow-up MRA of left carotid artery showing no evidence of dissection.

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The therapeutic paradox in the diagnosis of tuberculous meningitis

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The diagnosis of tuberculous meningitis (TBM) is challenging because of the low mycobacterial yield in the CSF and the lack of adequate diagnostic alternatives.[1,2] This often places the clinician facing patients with subacute-chronic meningitis in the situation of starting empirical antituberculous therapy while ruling out other etiologies. Adenosine deaminase in the CSF is increased in TBM but has false negatives, and PCR has a less than ideal sensitivity.[3] Once anti-mycobacterial therapy is started, some patients with TBM may develop a paradoxical reaction that may give a clue to the correct diagnosis.

In this report we characterize a paradoxical response in a patient with subacute lymphocytic meningitis in whom the microbiologic diagnosis remained elusive for almost 4 weeks.

Case report. A 70-year-old woman was admitted with a 1-week history of general malaise, disorientation, and fever of 37.7 °C. A cranial CT scan (with and without contrast) was normal, and a spinal tap yielded a mononuclear pleocytosis with increased proteins, low glucose, and normal adenosine deaminase levels (table). Her history was relevant for idiopathic epilepsy since childhood treated with phenytoin, primary hypothyroidism on replacement therapy, and mild hypertension.

An initial tuberculin test (RT 23, 2 tuberculin units) was negative. Chest x-ray was normal, as was a thoracoabdominal CT scan. Search for occult cancer and systemic infection was negative. Additional samples of 10 mL of CSF were processed almost weekly for 8 weeks (see table). A second tuberculin test with 2 tuberculin units, 1 week after the first, became positive (induration, 15 × 15 mm). From time of admission, she was treated with ceftriaxone and ampicillin (a 10-day course was completed), as well as empirical antituberculous therapy with pyrazinamide, isoniazid, and rifampin. Dexamethasone, 16 mg daily, was added on the third day and tapered off over 4 weeks. A CSF sample taken on the eighth day after admission grew *M tuberculosis*, although growth was evident on the fourth week. Sequential CSF analysis yielded a persistent low glucose level, moderate increased protein content, and lymphocytic pleocytosis until the third week, when the CSF showed a shift in the differential to polymorphonuclear (PMN) predominance (58% PMNs and 42% lymphocytes) with a moderate increase in the total cell count to 126 cells/μL. One week later, the composition of the cellular infiltrate in the CSF became reversed with a lymphocytic predominance. No eosinophils were detected in the CSF. Her condition progressively improved over the following weeks. CSF adenosine deaminase was always below normal value (<5 U/L). Brain MRI and intracranial MR angiography were normal on two occasions (2 and 20 days after admission), without evidence of meningeal enhancement or parenchymal or vessel involvement. CSF cultures were subsequently negative and she completed therapy uneventfully.

Discussion. Several paradoxical responses in CNS tuberculosis have been described, but their frequency and timing of appearance are unclear. More often, a paradoxical response is interpreted as a clinical deterioration occurring several weeks after starting therapy, sometimes associated with an increase in CSF pleocytosis, more commonly lymphocytic. Occasionally, as in our patient, initial lymphocytic response may change transiently in the direction of polymorphonuclear predominance when therapy is initiated, a phenomenon known as the therapeutic paradox and regarded as highly suggestive of tuberculous meningitis.[1] Our systematic follow-up of this patient showed such a shift on the third week after therapy, and was not associated with clinical worsening or development of brain tuberculomas. Interestingly, brain MRI was normal from the outset and so were the CSF levels of adenosine deaminase. A commercial PCR kit for *M tuberculosis* was repeatedly negative. The PMN predominance appeared on the third week after therapy and lasted less than a week. This syndrome is thought to represent an uncommon hypersensitivity reaction to the massive release of mycobacterial proteins into the subarachnoid space.[4,5] Initial tuberculin test was negative and became positive, which may reflect a recent exposure to *M tuberculosis* rather than the more common reactivation of an old meningeal focus.[1] She was not malnourished and had no evidence of immunosuppression.

Another form of a paradoxical reaction consists of the development of intracranial tuberculomas, or the enlargement of preexisting ones during the first weeks or months of treatment for tuberculous meningitis.[6]

Finally, paradoxical reactions also occur as tuberculoma development or progression during tuberculosis treatment in the HIV-infected patient when antiretroviral therapy restores immune function.[7]

Repeated spinal punctures in a patient with suspected tuberculous meningitis of uncertain origin increases the chances of positive microbiologic results, but also of revealing a therapy-induced shift in the differential cell count that may give a clue to the etiologic diagnosis.

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Table *Evolution of CSF measures during admission*

	Days from admission							
CSF measures	Day 0 (admission)	Day 1	Day 8	Day 17	Day 22	Day 30	Day 42	Day 51
Cells/μL	72	61	116	51	126	61	50	18
Lymphocytes, %	84	92	64	74	42	94	NA	98
PMNs, %	16	8	27	26	58	6	NA	2
Glucose, mg/dL	30	26	25	21	25	31	32	34
Proteins, mg/dL	90	74	35	61	45	52	36	37
Adenosine deaminase, U/L, normal < 5	4.3	5.2	4	4.9	3.2	3.5	2	2.2
Auramine stain	NA	Negative	Negative	Negative	NA	NA	Negative	NA
Mycobacterial culture	NA	Negative	Positive	Negative	NA	NA	Negative	NA
PCR	NA	Negative	Negative	Negative	NA	NA	NA	NA

PMNs = polymorphonuclear; NA = not available.

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Corrections

The reproductive effects of beta interferon therapy in pregnancy: A longitudinal cohort

In the article "The reproductive effects of beta interferon therapy in pregnancy: A longitudinal cohort" (*Neurology* 2005;65:807–811) by R. Boskovic, R. Wade, J. Wolpin, D.J. Bauer, and G. Koren, the second author's name was incorrect (originally listed as R. Wide). The correct listing is R. Wade.

Also in the same article, there is an error on Table 2, "Comparison of maternal and neonatal characteristics and pregnancy outcome among the three groups." Under Group I: Interferon for Weight gain in pregnancy, lb, mean SD, the correct value should be 25.7 ± 15.0.

Diagnosis and management of dementia with Lewy bodies: Third report of the DLB consortium

The article "Diagnosis and management of dementia with Lewy bodies: Third report of the DLB consortium" by I.G. McKeith et al. was published as an Expedited E-Pub on October 19, 2005, and appears in this issue on pages 1863–1872. Reference 60 in the Expedited E-Pub was incorrect. The correct reference is as follows:

Walker Z, Costa DC, Walker RWH, et al. Differentiation of dementia with Lewy bodies from Alzheimer's disease using a dopaminergic presynaptic ligand. J Neurol Neurosurg Psychiatry 2002;73:134–140.

The authors apologize for this error.

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