CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 33-2024: A 71-Year-Old Woman with Confusion, Aphasia, and a Brain Mass

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PRESENTATION OF CASE

Dr. Beatriz Rizkallah Alves (Neurology): A 71-year-old woman was transferred to this hospital for neurosurgical evaluation because of confusion, aphasia, and a mass lesion in the brain.

The patient had been in her usual state of health until 3 days before the current presentation, when headache and fatigue developed. The next day, she was typing on a computer at her workplace when she suddenly noticed that her typed words were incoherent. The episode of confusion and language production difficulties resolved after a few minutes. Confusion recurred in the evening while the patient was at dinner with her husband, and she sought evaluation in the emergency department of another hospital. Examination was reportedly normal.

Dr. Jeremy N. Ford: Computed tomography (CT) of the head, performed without the intravenous administration of contrast material, and CT angiography of the head and neck reportedly showed a large area of attenuation in the left posterior superior frontal lobe.

Dr. Alves: The patient was admitted to the other hospital.

Dr. Ford: On hospital day 2, magnetic resonance imaging (MRI) of the head (Fig. 1), performed after the intravenous administration of contrast material, revealed a heterogeneously enhancing mass in the left frontal lobe with vasogenic edema and mass effect. A diagnosis of cancer with metastases to the brain was considered, and additional imaging studies were obtained. CT of the chest, abdomen, and pelvis, performed after the intravenous administration of contrast material, reportedly showed a pulmonary nodule that measured 0.2 cm in diameter and a nodule in the right breast that measured 1.3 cm in diameter. The appearance of the breast nodule was unchanged from that observed on imaging performed 8 years before the current presentation, except for the presence of a new punctate calcification along the anterior margin.

On hospital day 3, expressive aphasia developed. CT of the head, performed without the intravenous administration of contrast material, reportedly showed increased

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N Engl J Med 2024;391:1529-38.
DOI: 10.1056/NEJMcpc2402496

CME

edema and mass effect that corresponded to the abnormality in the left frontal lobe.

Dr. Alves: Treatment with dexamethasone and levetiracetam was started. The patient received one dose of each medication before she was transferred to the neurosurgical unit of this hospital for evaluation and possible brain biopsy.

The patient had a history of myasthenia gravis, vitamin B_{12} deficiency, and hypertension. Surgical history included supracervical hysterectomy with bilateral salpingo-oophorectomy for uterine fibroids (performed 31 years before the current presentation), transsternal thymectomy and wedge resection of the right upper lobe for a cortical

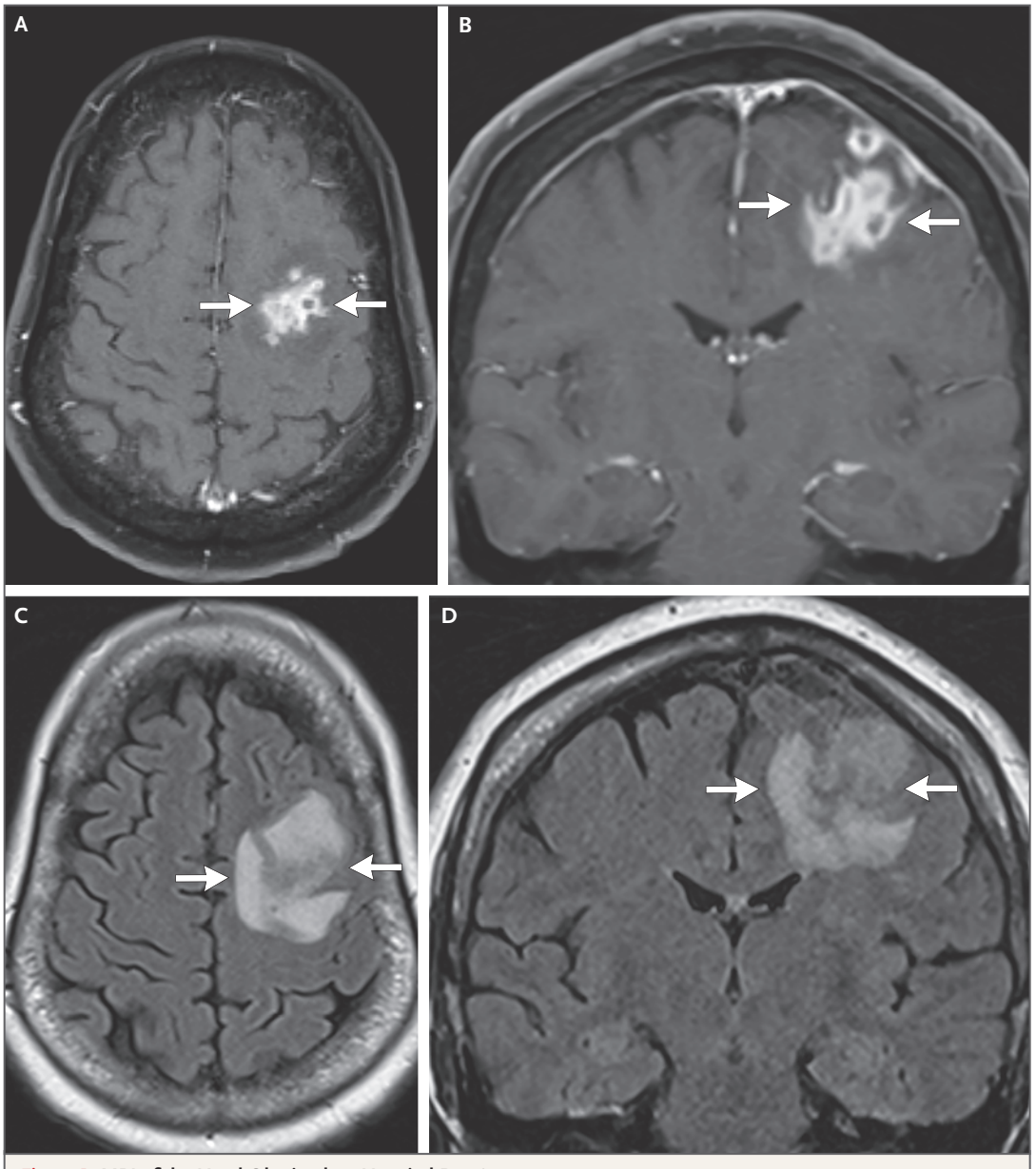

Figure 1. MRI of the Head Obtained on Hospital Day 2.

Axial (Panel A) and coronal (Panel B) T1-weighted images, obtained after the administration of contrast material, show masslike heterogeneous enhancement (arrows) centered in the left centrum semiovale and extending to the cortex. Axial (Panel C) and coronal (Panel D) T2-weighted fluid-attenuated inversion recovery (FLAIR) images show surrounding signal hyperintensity and mass effect (arrows), findings suggestive of vasogenic edema.

thymoma (22 years before the current presentation), and sigmoid colectomy for recurrent diverticulitis (7 years before the current presentation). Ocular-predominant myasthenia gravis was diagnosed 18 years before the current presentation and 4 years after thymectomy. Myasthenia gravis had been treated for 11 years with cyclosporine and pyridostigmine. Seven years before the current presentation, a flare of myasthenia gravis was complicated by respiratory failure, for which the patient received bilevel positive airway pressure and underwent plasmapheresis. After that hospitalization, cyclosporine therapy was replaced by mycophenolate mofetil, which she had been taking daily for the past 7 years.

Other medications included cholecalciferol, latanoprost ophthalmic drops, hydrochlorothiazide, polysaccharide–iron complex capsules, and monthly intramuscular vitamin B_{12} injections. Amoxicillin–clavulanate had previously caused rash. The patient lived in the northeastern United States, was married, and worked in an office. She had recently traveled to California for a wedding, and she reported no exposures to animals or fresh water. She had not had any recent dental procedures. She had no history of tobacco use or injection drug use. She rarely consumed alcoholic beverages. Her mother had systemic lupus erythematosus, and her father had lung cancer. A sister had rheumatoid arthritis.

On examination, the oral temperature was 36.6°C, the blood pressure 150/80 mm Hg, the pulse 91 beats per minute, and the oxygen saturation 96% while the patient was breathing ambient air. Severe expressive aphasia was observed, and she followed commands intermittently. Orientation could not be accurately assessed. She had disconjugate gaze at rest. Motor strength was 5/5 in the left arm and left leg and 4/5 in the right arm and right leg. The blood level of glucose was normal, as were the results of tests of kidney function. The blood level of phosphorus was 2.2 mg per deciliter (reference range, 2.6 to 4.5). The complete blood count was normal, as were the results of tests of coagulation.

Electroencephalography showed intermittent irregular delta slowing focally over the left frontal region but no epileptiform abnormalities. CT of the head, performed without the intravenous administration of contrast material, revealed ill-defined parenchymal hypoattenuation involving the left superior frontal gyrus, left middle frontal gyrus, and left centrum semiovale with associated sulcal effacement. Treatment with dexamethasone, hydrochlorothiazide, mycophenolate mofetil, and pyridostigmine was stopped, as was treatment with cholecalciferol, latanoprost drops, polysaccharide–iron complex, and vitamin B_{12}. Treatment with levetiracetam was continued, and empirical treatment with intravenous ceftriaxone and vancomycin was started.

Dr. Gavin P. Dunn: A left frontal craniotomy with open brain biopsy was planned to allow for sufficient lesional tissue to be obtained and to enable further debulking to be performed, pending intraoperative findings and the results of pathological assessment. A small craniotomy centered at the coronal suture directly over the target lesion was performed. The lesion appeared gray, and tissue that appeared abnormal was sampled in several locations of the lesion. Direct microscopic visualization of the specimens identified areas that did not appear to be completely distinct from normal tissue, and thus, further debulking was not indicated. No purulence was noted.

Dr. Christopher W. Mount: Intraoperative examination of a frozen section (Fig. 2) revealed a cellular infiltrate consisting of atypical cells in a focal perivascular arrangement. No evidence of pathologically significant acute inflammation

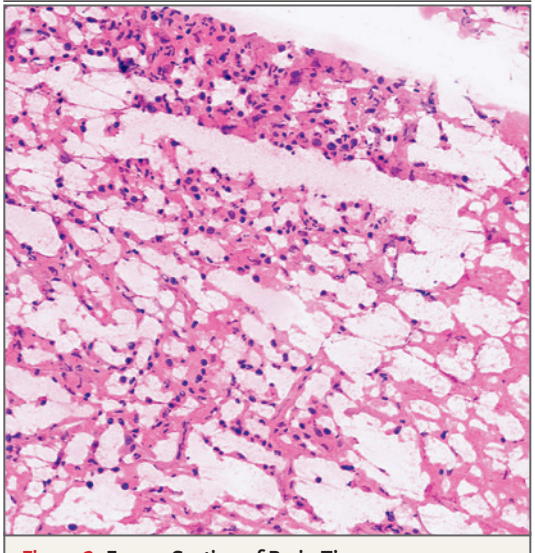

Figure 2. Frozen Section of Brain Tissue.

Hematoxylin and eosin staining of a frozen section examined intraoperatively shows brain parenchyma with atypical cells that are suggestive of cancer. No acute inflammation is present.

was present in the tissue. At this time, these features suggested the presence of a malignant infiltrate. Additional specimens were submitted to the pathology department for formalin fixation and interpretation.

Dr. Alves: A diagnosis of lymphoma was considered, and treatment with dexamethasone administered intravenously every 6 hours was started. On postoperative day 1, new facial droop on the right side developed, along with weakness with cervical flexion and monoplegia of the right arm.

Dr. Ford: Repeat MRI of the head (Fig. 3), performed after the intravenous administration of contrast material, revealed postsurgical changes in the left frontal lobe and susceptibility artifact

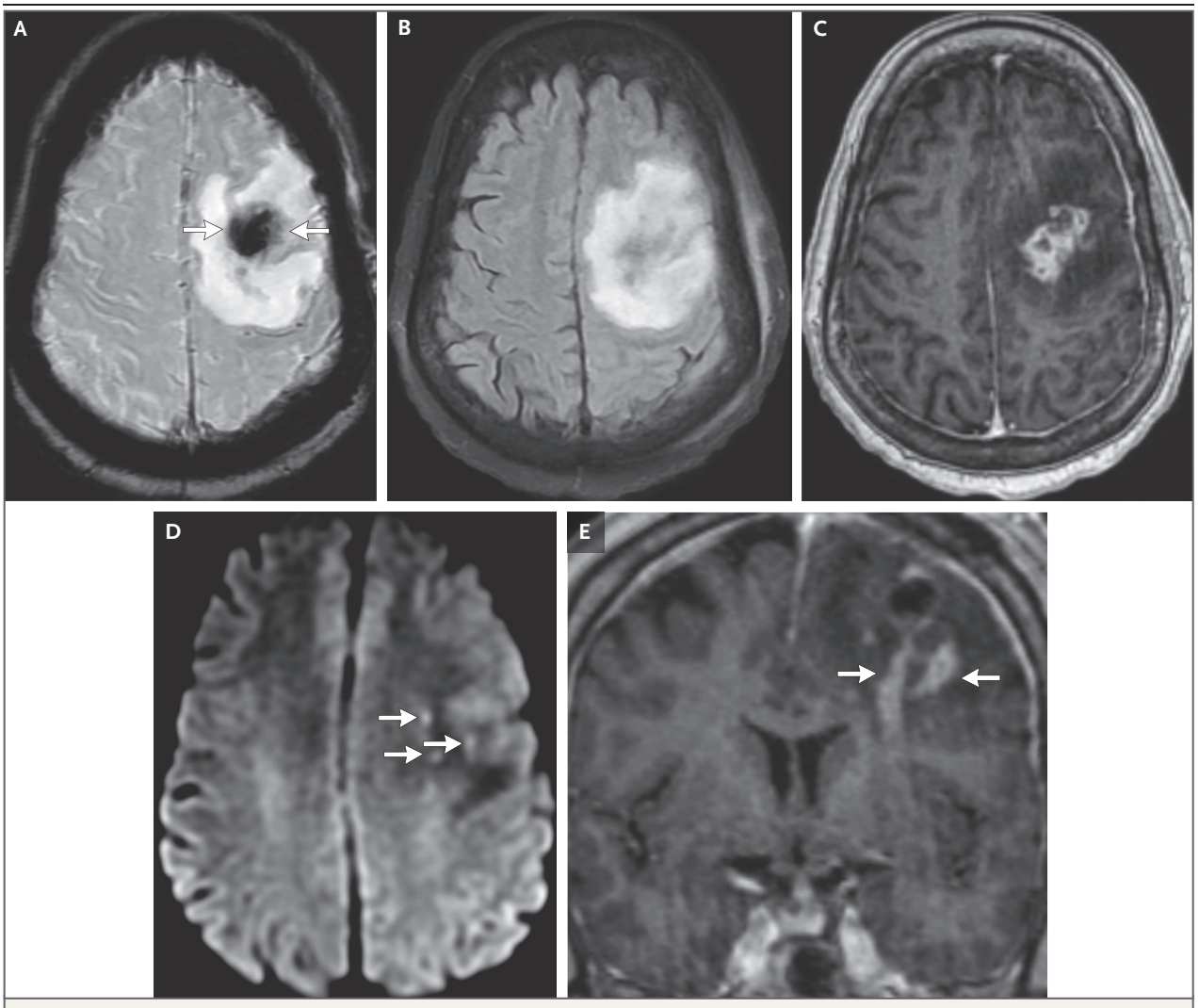

Figure 3. MRI of the Head Obtained on Postoperative Day 1.

Axial images from susceptibility-weighted imaging (SWI) (Panel A), T2-weighted FLAIR imaging (Panel B), three-dimensional T1-weighted imaging (Panel C) obtained after the administration of contrast material, and diffusion-weighted imaging (Panel D) show worsening and expansion of heterogeneous enhancement and signal hyperintensity as compared with that observed on T1-weighted images and T2-weighted FLAIR images from the preoperative MRI. Expected blooming indicating the presence of blood products is seen on the SWI (Panel A, arrows). Nonspecific punctate foci of restricted diffusion are present near the biopsy site (Panel D, arrows). A coronal three-dimensional T1-weighted image (Panel E), obtained after the administration of contrast material, shows enhancement surrounding the biopsy site (arrows).

at the biopsy site that was consistent with blood products. There was enlargement of the expansile lesion in the left frontal lobe that corresponded to an area of hyperintensity on T2-weighted fluid-attenuated inversion recovery imaging.

Dr. Alves: A diagnostic test was performed.

DIFFERENTIAL DIAGNOSIS

Dr. Isabel Arrillaga-Romany: I participated in the care of this patient, and I am aware of the final diagnosis. This 71-year-old woman with a history of myasthenia gravis that had been treated with mycophenolate mofetil presented to another hospital with a 2-day history of new headache and a 1-day history of transient word-finding difficulty and confusion. MRI of the head revealed a heterogeneously enhancing mass in the left frontal lobe with surrounding vasogenic edema and small foci of restricted diffusion. Her symptoms progressed rapidly over a period of several days and included expressive aphasia and weakness on the right side.

The differential diagnosis for any patient with a newly discovered brain mass includes cancer, infection, autoimmune disease, vascular disease, a toxic–metabolic process, and congenital lesions. Understanding the severity and time course of symptoms is essential to narrowing the differential diagnosis. The patient's laboratory test results showed no evidence of a toxic–metabolic cause for the presentation. Benign or indolent processes such as a congenital lesion can be ruled out on the basis of the patient's acute presentation and rapid progression of symptoms. Slower progression of pathologic processes in patients with indolent disease allows for recruitment of compensatory mechanisms to maintain brain function. Therefore, persons with indolent disease processes typically present with disproportionately mild and slowly progressive symptoms.[1] This patient's presentation involving severe and rapidly progressive symptoms over a period of days indicates an aggressive process.

VASCULAR DISEASE

The progressive nature of the patient's symptoms would be unusual for a vascular process. Ischemic stroke symptoms can be rapidly progressive in the context of hemorrhagic conversion, which may occur within hours to days after the initial presentation. Moreover, in the context of prodromal transient ischemic attacks, stroke may be preceded by several days of headaches or transient focal neurologic symptoms,[2] both of which were seen in this patient. In this case, however, imaging findings were not consistent with a vascular cause, although imaging of resolving central nervous system (CNS) infarctions or hematomas, performed months after the onset of symptoms, can show gyriform enhancement that can at times be difficult to distinguish from a mass.

AUTOIMMUNE DISEASE

Persons with a fulminant autoimmune disease such as tumefactive multiple sclerosis can present with lesions in the brain that exert mass effect and are associated with heterogeneous enhancement. It is important to note that the pattern of ring enhancement seen in patients with autoimmune diseases of the CNS is most often that of an incomplete or open ring.[3] This patient's imaging did not show an open-ring pattern of enhancement. Although this difference from the classic imaging pattern does not rule out the possibility of an autoimmune disease, the patient's previous years of treatment with mycophenolate mofetil and lack of clinical improvement after treatment with high-dose glucocorticoids make this diagnosis unlikely.

CNS LYMPHOMA

CNS cancers are often associated with weeks to months of slowly progressive, mild symptoms followed by more severe and rapidly progressive symptoms. Although this patient did not have symptoms before the 2 days of headache at presentation, a CNS cancer remains high on the differential diagnosis. The patient's immunocompromised state, along with the imaging findings and initial interpretation of biopsy results from the tissue sample submitted for frozen-section examination, strongly suggest CNS lymphoma.

Immunocompromised persons are at increased risk for several hematologic cancers, including lymphoma. Although the exact mechanism of increased risk remains unclear, it is thought to be associated with a defective immunosurveillance system that allows cancer cells to escape host immunity.[4] A weakened immune system may be especially important in the development

of lymphomas associated with Epstein–Barr virus (EBV).[4] In keeping with this observation, restoration of immune function can be an effective management strategy for EBV-associated lymphomas.

Head imaging findings in patients with CNS lymphoma are typically characterized by intraparenchymal, multifocal, homogeneously enhancing lesions with associated restricted diffusion. This patient's MRI revealed a heterogeneously enhancing mass with associated foci of restricted diffusion that would not be typical of classic lymphoma. However, in immunocompromised persons with CNS lymphoma, the appearance of a lymphomatous lesion may be atypical and can show ring enhancement with central necrosis that closely resembles high-grade glioma or abscess.[5]

INFECTION

Infection is another important consideration in this immunocompromised patient. She did not have fever, an elevated white-cell count, or a history of recent dental procedures — factors that would increase the possibility of infection. However, brain abscess and CNS lymphoma can often be difficult to distinguish in immunocompromised persons because of similar risk profiles and similar features on MRI.[6] CNS lymphoma was our working diagnosis after the brain biopsy; however, because the patient's condition worsened despite treatment with high-dose glucocorticoids, we expanded our differential diagnosis to include the possibility of fulminant CNS infection such as a bacterial abscess. To obtain additional information, we recommended repeating the head MRI.

DR. ISABEL ARRILLAGA-ROMANY'S DIAGNOSIS

Lymphoma of the central nervous system or infection (possible brain abscess).

CLINICAL COURSE AND DISCUSSION OF NEUROSURGICAL MANAGEMENT

Dr. Dunn: When the patient was initially transferred to this hospital, her examination was notable for severe expressive aphasia, which was considered to be out of proportion to the imaging findings. Moreover, we strongly considered the possibility that the imaging findings were consistent with lymphoma, particularly because the patient was immunosuppressed. Although we needed to minimize glucocorticoid administration before a diagnostic surgical procedure so that the pathological findings would not be obfuscated, we were highly motivated to initiate treatment with high-dose glucocorticoids. Therefore, we reviewed the clinical urgency with the patient and her family and planned a sampling procedure. The intraoperative histopathological assessment was highly suggestive of lymphoma, and treatment with high-dose glucocorticoids was initiated postoperatively. Because the findings on the intraoperative frozen-section examination were interpreted as probable lymphoma, no additional specimens were obtained for microbiologic evaluation. After the procedure, despite the administration of glucocorticoid treatment, the patient's neurologic status continued to decline, and on postoperative day 1, repeat MRI of the head revealed disease progression and worsening edema. On postoperative day 2, our team was alerted by the pathology consultant about new findings regarding the specimen that had been sent for histologic evaluation of a permanent section.

Dr. Mount: The remnant of the specimen that

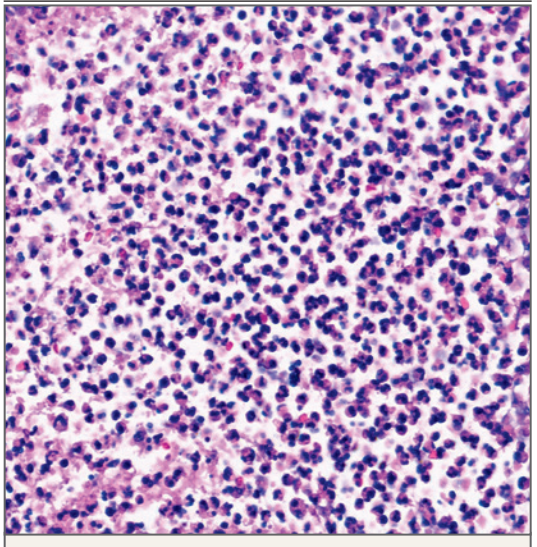

Figure 4. Permanent Section of Brain Tissue.
Hematoxylin and eosin staining of a permanent section shows dense collections of neutrophils in microabscesses.

had been sampled for intraoperative frozen-section examination was processed for permanent-section examination (Fig. 4). On histologic examination of the specimen, the densely cellular lesion appeared to be predominantly lymphohistiocytic. Additional tissue that had been submitted for permanent-section evaluation was found to have abundant microabscesses with multifocal, dense collections of neutrophils and surrounding lymphohistiocytic inflammation. No granulomas were identified. Immunohistochemical staining of a specimen of the original densely cellular lymphoid focus seen at the time of frozen-section examination revealed predominantly mature-appearing CD3+ T lymphocytes, with only rare CD20+ B lymphocytes — findings that were consistent with reactive inflammation.

Dr. Dunn: Owing to the patient's worsening neurologic status despite treatment with glucocorticoids, evidence of progression on imaging, and new data suggesting a different pathologic process, we immediately performed a second brain biopsy to obtain more tissue for microbiologic testing. The base of the lesion was firm and clearly different from normal tissue, and a more lateral area of the lesion harbored two areas of a liquid material. These areas were sampled for microbiologic testing. The patient's condition remained stable after the procedure.

INITIAL MICROBIOLOGIC EVALUATION

Dr. Katherine A. Latham: The diagnostic specimen submitted to the microbiology laboratory was an aspirate from the second brain biopsy. Gram's staining of the specimen revealed few gram-negative rod-shaped bacteria, with a background of extensive cellular debris (Fig. 5A).

CLINICAL IMPRESSION AND DISCUSSION OF INITIAL MANAGEMENT

Dr. Camille N. Kotton: The infectious disease service was consulted because Gram's staining of the specimen obtained during the second neurosurgical procedure showed few polymorphonuclear cells and few gram-negative rods. Given this patient's immunocompromised status, we considered the possibility that the presence of atypical pathogens was causing brain abscess. I was specifically concerned that the stained material could be over-decolorized — a common technical problem with Gram's stain interpretation wherein a gram-positive organism (purple) appears gram-negative (pink). To account for this possibility, I also considered the presence of organisms such as listeria, mycobacteria, and nocardia, as well as various enteric and nonenteric gram-negative organisms. On the basis of the Gram's staining results, opportunistic fungal or parasitic infection seemed unlikely.

With regard to the source of infection, approximately half of brain abscesses result from contiguous spread of infection, and approximately a third result from hematogenous spread[7]; this patient had no history of recent infection that would confer a predisposition to brain abscess. We decided to treat this patient with intravenous ampicillin, vancomycin, ceftriaxone, and metronidazole while we awaited additional microbiologic results. Although the laboratory reported gram-negative rods, we wanted to be sure to provide broad treatment in case the patient was ultimately found to have a polymicrobial infection. We were mindful of the need for the antibiotic agents to effectively cross the blood–brain barrier[8] and confirmed the appropriate antimicrobial doses with an expert pharmacist. We reviewed the Gram's staining findings with microbiology laboratory personnel. In addition, we requested routine bacterial culture, acid-fast bacilli staining and culture, fungal staining and culture, and modified acid-fast bacilli staining. We also requested that a specimen be held for next-generation sequencing in case the cultures were negative.

DIAGNOSTIC MICROBIOLOGIC EVALUATION

Dr. Latham: Given the concern that the organism may have been over-decolorized, repeat Gram's staining of the aspirate was performed. The subsequent Gram's staining revealed gram-positive rods (Fig. 5B), for which the differential diagnosis includes infection with listeria, corynebacteria, bacillus, and clostridium species. The organism grew on nutrient agar, and colonies that were small, smooth, and blue-gray in appearance were observed. The organism had flagella and moved

with a tumbling motion, which is characteristic of organisms with such structures. A narrow zone of β-hemolysis, colloquially termed "candlelight" hemolysis, was also present. The presence of growth on solid media allowed for matrix-assisted laser desorption ionization–time-of-flight (MALDI-TOF) mass spectrometry to be performed; this tool established the definitive diagnosis

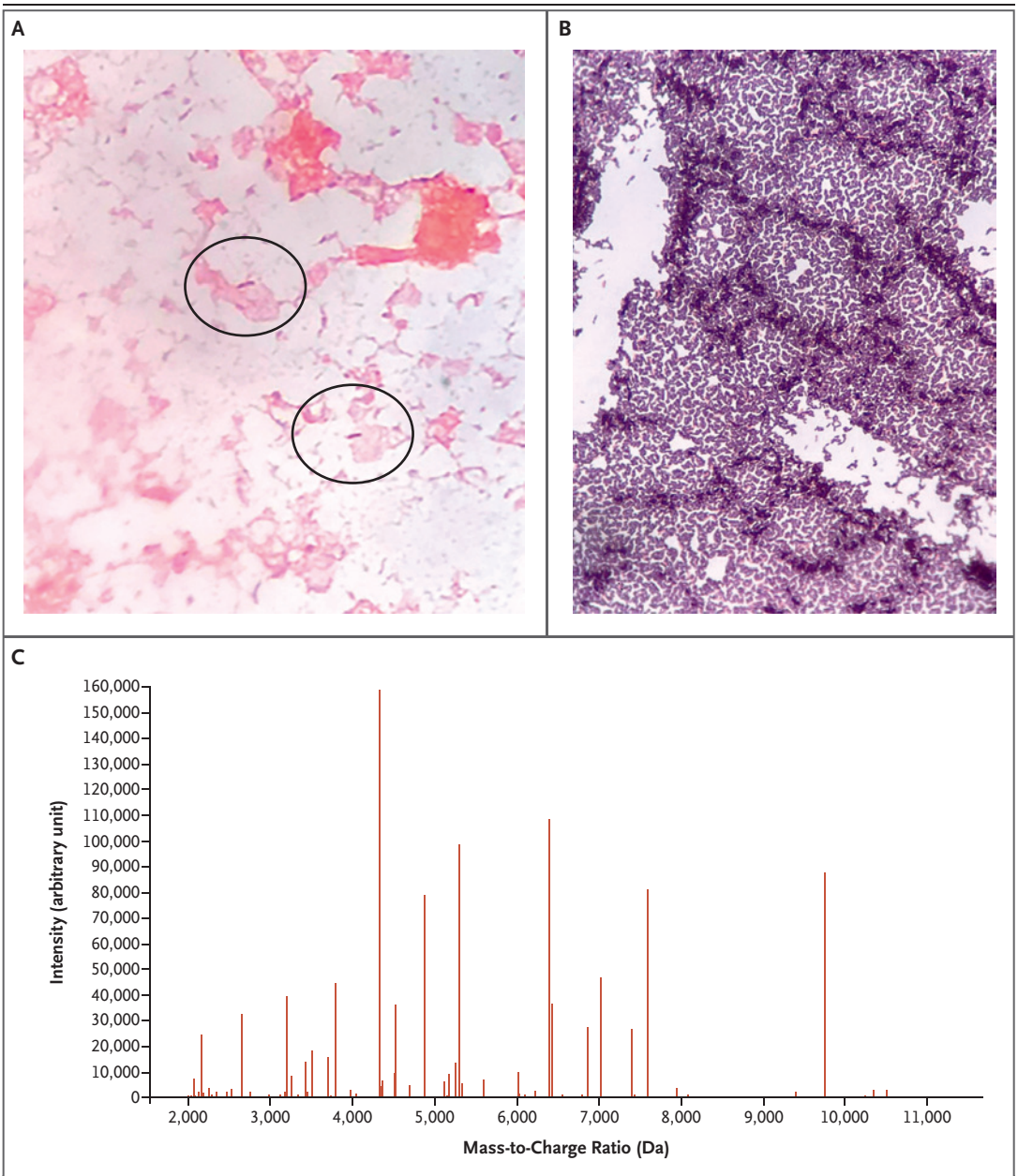

Figure 5. Microbiologic Results from the Brain Aspirate Specimen.

Initial Gram's staining of the specimen shows few gram-negative rods (Panel A, circled). Repeat Gram's staining of the specimen shows gram-positive organisms (Panel B). The results of matrix-assisted laser desorption ionization–time-of-flight (MALDI-TOF) mass spectrometry establish a definitive diagnosis of *Listeria monocytogenes* infection (Panel C).

and speciation as *Listeria monocytogenes* infection (Fig. 5C).

MICROBIOLOGIC DIAGNOSIS

Listeria monocytogenes infection.

DISCUSSION OF INFECTIOUS DISEASE MANAGEMENT

Dr. Kotton: Once we had a positive identification of *L. monocytogenes*, we tailored the antibiotic regimen to include ampicillin and linezolid. Given the potential severity of the infection, along with the fact that antibiotics may have decreased CNS penetration, we opted for dual therapy, anticipating a long course of ampicillin. We decided against treatment with gentamicin owing to its relatively low CNS penetration and our concern that it could affect the neuromuscular junction and exacerbate myasthenia gravis. Antimicrobial susceptibility testing showed excellent susceptibility to ampicillin and linezolid. Our review of the literature, which suggested that the best approach to brain abscess due to *L. monocytogenes* was neurosurgery in combination with a long course of antimicrobial therapy,[9] helped guide our management strategy. The patient received a total of 6 weeks of intravenous ampicillin, combined with 2 weeks of oral linezolid therapy concurrent with the first 2 weeks of ampicillin treatment, and then 2 additional weeks of oral linezolid therapy after the completion of ampicillin treatment.

L. monocytogenes penetrates the CNS by hematogenous spread from the gastrointestinal tract. The incubation period for CNS disease is approximately 9 days.[10,11] This patient did not have any obvious high-risk food exposures in that timeframe. Her case was investigated by the Massachusetts Department of Public Health and the Centers for Disease Control and Prevention (CDC). *L. monocytogenes* whole-genome sequencing was performed at the CDC for identification.[12] This case had some features that were similar to those of a previously identified cluster, but no clear linkages with food products were established and no recalls occurred.

The patient's myasthenia gravis had been treated for 7 years with mycophenolate mofetil, which was discontinued after the brain abscess was diagnosed. She had been treated perioperatively with dexamethasone for 3 days owing to concerns about possible lymphoma, then with prednisone for 14 days for management of myasthenia gravis. She was transitioned to monthly treatment with high-dose intravenous immune globulin for long-term management of myasthenia gravis, given concerns about her risk of infection if she were to continue to receive immunosuppressive therapy.

Dr. Simran Gupta (Medicine): After a 3-week hospitalization, the patient was discharged to a rehabilitation facility, where she continued intensive rehabilitation that included physical therapy, occupational therapy, and speech therapy. She had a neurologic recovery, with improvement in motor function, coordination, and strength on the right side, as well as improvements in speech. After 6 weeks in the rehabilitation facility, the patient was discharged home. Nine months after the initial diagnosis, she continues to engage in speech therapy and has had a gradual improvement in speech fluency.

PATIENT PERSPECTIVE

The Patient: I want to thank my husband, my amazing family, and dear friends who have loved and supported me throughout my health journey.

I manage a public health communications agency. The irony is not lost on me: the head of a public health communications agency who promotes Covid-19 vaccines, HIV and AIDS prevention, cancer prevention, and other health initiatives infected by listeria. Go figure!

Two days earlier, I had just had my annual physical and received a clean bill of health. So, I was excited to celebrate 43 years of marriage with my beloved husband. At work that day, I was typing an email, and my brain became fuzzy. My business partner asked what the problem was, and she came to assist me. I was confused and could not focus, and she had to finish the email for me. After a while, the confusion went away, and I gathered up my things and said goodbye.

I went to my car and drove to the restaurant across town to meet my husband. I was able to speak normally and chatted with another family

member we ran into. After we sat at our table, I went to the restroom. I felt fatigued and could not clear my head. I made it back to our table, and I told my husband I wasn't feeling so well. Concerned, my husband paid our bill, and we headed to the nearest hospital.

In the emergency room, I was feeling OK. But after I laid my head down on my husband's shoulder, everything was a blur from that point on.

The Patient's Husband: The doctors were undeterred, determined, and dedicated to figuring out what the problem was, and thank goodness they did. This miracle diagnosis could not have been made without the care provided by the neurosurgery, infectious disease, radiology, and anesthesiology team of physicians. The rehabilitation doctors, nurses, and therapists nursed my wife back to health, and she's still improving.

FINAL DIAGNOSIS

Brain abscess due to infection with *Listeria monocytogenes*.

CASE RECORDS EDITORS' NOTE — LESSONS LEARNED

1. Infection with *L. monocytogenes* can cause devastating CNS disease in high-risk persons, particularly in aging and immunosuppressed adults.

2. Interpretation of findings on intraoperative frozen-section examination of biopsy material provides insight into the underlying disease process but is only a presumptive diagnosis and must be confirmed by review of permanently fixed and stained specimens.

3. Specimens that undergo testing with Gram's staining are subject to technical error and may be over-decolorized or under-decolorized during the staining procedure, which can lead to an erroneous result. Over-decolorization results in a gram-positive organism (purple) appearing pink, like a gram-negative organism. Conversely, under-decolorization results in a gram-negative organism (pink) appearing purple, like a gram-positive organism.

This case was presented at Neuroscience Grand Rounds.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank Dr. Matthew Frosch for assistance with the pathology portions of the conference and case presentation.

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