Presentation of Case

A 50-year-old woman was seen in the emergency department at Massachusetts Eye and Ear Infirmary (MEEI), affiliated with this hospital, because of pain and decreased hearing in the left ear.

The patient had been in her usual state of health until 6 weeks earlier, when she noted decreased hearing, discomfort, and a sensation of blockage in her left ear. She saw an otolaryngologist at another hospital. On examination, there was maceration, desquamation, and minute hemorrhages on the left tympanic membrane, without perforation. An audiogram reportedly showed conductive hearing loss in the left ear, with a speech-reception threshold of 20 dB. A tympanogram was flat (indicating decreased mobility of the ear drum, a feature that is consistent with the presence of fluid in the middle ear). The ear canal was irrigated, and a crust was removed from the eardrum. The patient was treated with oral azithromycin and an otic suspension consisting of neomycin, polymyxin B, and hydrocortisone. Symptoms improved briefly but then recurred, with worsening pain, and she came to the emergency department at MEEI.

The patient did not have otorrhea, tinnitus, fever, weight loss, cough, nausea, vomiting, or abdominal pain. Tests for tuberculosis and the human immunodeficiency virus had been negative in the past. A diagnosis of essential thrombocytopenia had been made 18 years earlier; treatment had included interferon alfa and hydroxyurea and had been complicated by anemia that required intermittent transfusions. She had hypertension, anxiety, chronic diarrhea, and mild renal insufficiency. Her medications included anagrelide hydrochloride, darbepoetin alfa, and sertraline. She was allergic to hydroxychloroquine sulfate. She was married, had worked in a health care facility, and currently worked in an office; she had traveled to Europe, the Mediterranean, and in the distant past, to China and the Caribbean. She did not drink alcohol, smoke, or use illicit drugs. Her mother had had diabetes mellitus, hepatic and renal disease, and breast cancer, and her father had had heart disease and hypertension; her siblings and her child were healthy.

On examination, the blood pressure was 152/72 mm Hg and the other vital signs were normal. The left auricle was normal. The left ear canal and tympanic membrane were erythematous, and the canal contained white debris; there was a serous effusion in the middle ear. The right ear was normal. The Rinne test revealed
better hearing by means of air conduction than by means of bone conduction bilaterally; in the Weber test, sound lateralized to the left. The remainder of the examination was normal. An audiogram showed mixed hearing loss in the left ear: the hearing loss was purely conductive below 4000 Hz and mostly sensorineural above that frequency. The right ear had purely sensorineural hearing loss above 4000 Hz, paralleling the left-sided loss (Fig. 1 in the Supplementary Appendix, available with the full text of this article at NEJM.org). Azithromycin, methylprednisolone in a tapering dose, and an otic solution of neomycin, polymyxin B, and hydrocortisone were prescribed, but the patient discontinued the glucocorticoid because of insomnia. The pain resolved, but blockage persisted; on follow-up by an otorhinolaryngologist 3 days later, myringotomy with tube placement was performed and ciprofloxacin and dexamethasone drops were prescribed. The patient was advised to keep water out of the ear.

On follow-up 3 weeks later (2 months before admission), the patient reported improved hearing and no pain. On examination, abundant debris was found and removed from the left ear canal; dried serous material covered the left tympanic membrane, and granulation tissue obstructed the lumen of the tube. The speech-reception threshold was in the 35-dB range; other findings were unchanged. The tube was removed and the middle ear was aspirated, without evidence of purulence. Acetic acid drops were prescribed. Results of laboratory tests, performed later that week at the other hospital, are shown in Table 1.

Two weeks later, the patient fell while jet-skiing on a river, exposing her left ear to the water. Pain in the ear recurred, and she returned to the emergency department at MEEI 5 days later. She rated the pain at 3 to 4 on a scale of 1 to 5, with 5 indicating the most severe pain. The left auricle was tender on manipulation, without tragal pain. There was wet, white debris in the ear canal, with no perforation, effusion, or drainage. Other findings were unchanged. A 10-day course of amoxicillin–clavulanic acid was begun.

On follow-up examination 1 week later, 3.5 months after the onset of symptoms, the pain had resolved. The tympanic membrane was re-

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reference Range, Adults†</th>
<th>First Hospital, 2 Mo before Admission</th>
<th>First Hospital, 3 Wk before Admission</th>
<th>MEEI, 2 Days before Admission</th>
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* MEEI denotes Massachusetts Eye and Ear Infirmary. To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the values for creatinine to micromoles per liter, multiply by 88.4.
† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are for adults who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.
tracted, with an effusion but without perforation. A ballottable (mobile when palpated) subcutaneous mass, thought to be a cyst, emerged from the posterior superior quadrant of the external auditory canal and obscured the pars flaccida. Other findings were unchanged. Computed tomography (CT) of the temporal bone 1 week later revealed a hyperdense soft-tissue mass, 3.2 cm by 2.5 cm by 4 cm, against the lateral aspect of the left mastoid bone and mastoid process, medial to the auricle. The mass extended superiorly to the temporalis muscle and medi ally into the external auditory canal, narrowing the lumen to approximately 2 mm, without bony erosion. There was opacification of the left mastoid air cells and portions of the middle-ear cavity and retraction of the tympanic membrane.

One week later, the patient returned to the emergency department at MEEI because of recurrent otalgia. The left external auditory canal was narrowed by edematous skin; the tympanic membrane appeared normal. Oral amoxicillin–clavulanic acid and eardrops consisting of ciprofloxacin and dexamethasone were prescribed. The otalgia resolved, but at follow-up 1 week later, the patient reported that fullness and decreased hearing persisted. One week later, otalgia recurred. A repeat examination revealed postauricular swelling of the left ear. There was whitish debris and desquamated epithelium in the external auditory canal; the medial concha was edematous, with prominent capillaries; and the canal was narrowed posteriorly by a hypervascular soft-tissue mass, which had enlarged from previous examinations and extended along the posterior wall of the canal to the tympanic membrane. Gram’s staining of a swab from the canal revealed few polymorphonuclear cells, abundant epithelial cells, and no bacteria; routine and fungal cultures were sterile. The administration of levofloxacin was begun, and the otalgia resolved within 48 hours.

Contrast-enhanced magnetic resonance imaging (MRI) of the brain and temporal bone 1 week later revealed an enhancing soft-tissue mass, 4.5 cm by 2.8 cm by 4.5 cm, in the periauricular region of the left ear, involving the temporalis muscle and extending into the left external auditory canal, without bony erosion. There was opacification of the left mastoid air cells and middle-ear cavity, with dural enhancement along the floor of the left middle cranial fossa. Serum levels of glucose, electrolytes, albumin, globulin, calcium, and angiotensin-converting enzyme were normal, as were results of liver-function tests. Tests for Lyme disease, antinuclear antigen, and antineutrophil cytoplasmic antibodies (ANCA) were negative, as was a skin test for tuberculosis; other laboratory-test results are shown in Table 1. Two days later, a diagnostic procedure was performed.

**Differential Diagnosis**

Dr. Konstantina M. Stankovic: May we review the imaging studies?

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**Figure 1 (facing page). Imaging Studies of the Ear.**

An axial CT image, without contrast material, in soft-tissue window (Panel A) shows a hyperdense, periauricular soft-tissue mass on the left, located medial to the auricle and skin surface (white arrow). The mass involves the subcutaneous fat and extends to the margins of the external auditory canal and mastoid bone. A bulky lobular component within the external auditory canal narrows the lumen (black arrow). The mastoid air cells appear opacified (black arrowhead). An axial CT image, without contrast material, displayed in bone window (Panel B) shows that there is no bony erosion, destruction, or demineralization associated with the soft-tissue mass. The contour of the external auditory canal is preserved, without expansion, scalloping, or remodeling. The mastoid air cells are completely opacified, but the bony septations are intact, without erosion. An axial T1-weighted image from an MRI scan obtained 1 month later without the administration of contrast material (Panel C) better delineates the infiltrative soft-tissue mass. The mass is quite bulky, uplifting superficial fascial layers laterally (arrow), and has increased in size since the CT scan so that it now completely obliterates the lumen of the external auditory canal (arrowhead). Despite the increase in the size of the mass, bony margins remain preserved. The mass is hypo-intense relative to the brain. The mastoid air cells are opacified, with preserved bony septations. A coronal T1-weighted image from the same MRI scan after the administration of contrast material (Panel D) shows the periauricular mass with extension into the external auditory canal. The middle ear is opacified (arrowhead). The temporalis muscle is involved (arrow). There are no bony changes; the scutum remains sharp and un eroded (asterisk). The mass only moderately enhances. An axial T2-weighted image (Panel E) shows that the mass is heterogeneous in signal but is relatively dark overall as compared with the cerebrospinal fluid, which suggests hypercellularity. The opacity in the mastoid air cells is much more hyperintense and may represent a separate process related to inflammation or obstruction. The dark signal on an apparent-diffusion-coefficient map (Panel F) indicates reduced diffusion, which suggests that the mass is hypercellular (arrow).
Dr. Amy F. Juliano: A CT scan of the temporal bones, performed without the administration of contrast material, shows a hyperdense soft-tissue mass in the periauricular region of the left ear, medial to the auricle, that extends to the margins of the external auditory canal and mastoid bone; a lobular component in the canal causes luminal narrowing (Fig. 1A). Bony margins ap-
pear preserved, without erosion, destruction, or demineralization. The external auditory canal is preserved in contour and caliber, without expansion or remodeling of the canal (Fig. 1B).

An MRI scan obtained 1 month later shows that the mass has increased in size and now entirely fills and obliterates the lumen. The mass is mildly hypointense relative to the brain on a T1-weighted sequence (Fig. 1C), heterogeneously hypointense relative to the cerebrospinal fluid on a T2-weighted sequence (Fig. 1E), and shows moderate heterogeneous enhancement (Fig. 1D). There is reduced diffusion (Fig. 1F), which suggests that the mass is probably hypercellular. The ipsilateral mastoid air cells and middle-ear cavity are opacified, but signal and enhancement characteristics differ from those of the periauricular mass, which suggests that the opacification is due to inflammation or obstruction, or both, rather than to extension of the mass.

Since the mass is solid and enhances, it is not a hematoma or an abscess. The absence of bony erosion argues against cholesteatoma, and the absence of canal expansion argues against keratosis obturans. The preserved integrity of bone despite such an extensive mass argues against malignant otitis externa, Wegener’s granulomatosis, tuberculosis, and epithelial cancers such as squamous-cell carcinoma. The apparent hypercellularity raises the possibility of lymphoma, although the degree of enhancement is less than that usually seen in lymphomas. The findings are not specific, and infectious, inflammatory, and neoplastic processes remain possible.

Dr. Stankovic: I cared for this patient and am aware of the diagnosis. This 50-year-old woman with no otologic history presented with a clinical picture of recurrent otitis media and otitis externa; months later, a mass in the medial external auditory canal developed that progressed laterally to include the concha and the postauricular region without causing bony destruction. The disease occurred despite the patient’s general good health and stable chronic hematologic disease. The differential diagnosis includes infectious, inflammatory, and neoplastic processes.

INFECTIONOUS DISEASES

Acute Otitis Media

Acute otitis media is characterized by otalgia, aural fullness, conductive hearing loss, a thickened or hyperemic tympanic membrane, fluid in the middle-ear space, and a flat tympanogram, all of which the patient initially had. Acute otitis media is most common in childhood, but adults can be affected, even without an otologic history. Chronic immunosuppression is a predisposing factor and could be present in this patient who has essential thrombocythemia. The most common pathogens are Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis. Infection with unusual microorganisms may occur after exposure to river water and could have occurred in this patient who had a tympanic membrane perforation after removal of an occluded tympanostomy tube.

Chronic Otitis Media

Chronic otitis media can be classified into three categories: chronic active otitis media, which implies an active and potentially progressive process, such as chronic drainage or cholesteatoma; chronic inactive otitis media, which implies a stable, nonprogressive abnormality, such as a tympanic-membrane perforation or a retraction pocket in the tympanic membrane; and chronic inactive otitis media with frequent reactivation, such as a perforated tympanic membrane with episodic purulent drainage. Pathogens associated with chronic otitis media are typically mixed and include gram-negative bacilli (e.g., pseudomonas, klebsiella, and proteus species and Escherichia coli), staphylococcus, and anaerobes. Cholesteatoma is a cystlike mass of desquamating debris in the middle ear; the mass is lined with stratified squamous epithelium. It typically arises as a consequence of rupture or retraction of the tympanic membrane (i.e., a retraction pocket) due to chronic recurrent infections. It is unlikely in this patient in view of the absence of a history of otologic problems, the absence of a retraction pocket in the tympanic membrane or keratin in the middle ear, and no radiologic evidence of ossicular or scutal erosion despite a fully opacified middle ear and mastoid.

Chronic tuberculous otitis media may present as insidious, scant, typically painless otorrhea and is characterized by the presence of multiple minute perforations of the tympanic membrane and pale granulation tissue. Tuberculous otitis can occur in the absence of pulmonary involvement; in one series, less than 50% of the cases had abnormal chest-imaging studies. Tuberculous otitis media is a consideration in this case,
but the tympanic-membrane perforation was iatrogenic, the characteristic granulation tissue was absent, and a tuberculin skin test with purified protein derivative was negative.

**Otitis Externa**
Otitis externa is characterized by otalgia, auricular and tragal tenderness, an edematous and erythematous external auditory canal, conductive hearing loss, and possibly blunting of the post-auricular crease, all of which this patient had. The major cause is *Pseudomonas aeruginosa* infection acquired from water during swimming (swimmer's ear). Malignant or necrotizing otitis externa is a skull-based osteomyelitis seen primarily in patients with diabetes, characterized by progressive ear pain that develops over a period of weeks to months. It is caused by *P. aeruginosa* in nearly all cases. A characteristic finding in malignant otitis externa is granulation tissue at the junction of the bony and cartilaginous external auditory canal, which was lacking in this case. Advanced osteomyelitis is characterized by cranial-nerve palsies and evidence of bony destruction on CT, which were not present in this case.

This patient's pain improved each time she took antibiotics, which suggests that the pain was, at least in part, due to recurrent acute otitis media and otitis externa. However, the eventual persistence of pain, fullness, and hearing loss and the ultimate appearance of a soft-tissue mass suggest that the mass was obstructing the canal, the eustachian tube, or both and was the underlying cause of the pain and other symptoms. The mass could be inflammatory or neoplastic.

**Inflammatory and Other Diseases**
Autoimmune inflammatory and granulomatous diseases represent a small but important subgroup of ear diseases and should be considered in this case. Wegener's granulomatosis is a vasculitis that typically has systemic manifestations, especially pulmonary and renal, and is rarely isolated to the middle ear. Otologic manifestations are nonspecific and include serous exudates, otalgia, and conductive hearing loss. The eardrum is thickened and often perforated. Lack of destructive effects on the temporal bone and lack of granulation tissue in the middle ear make this diagnosis unlikely. Testing for ANCA was negative; however, this test may be negative in 40% of patients with limited disease.

**Neoplastic Diseases**
In this patient with an enlarging mass, a neoplasm must be considered. Neoplasms of the temporal bone can be primary tumors, metastases from local tumors (e.g., tumors of the parotid gland or skin of the auricle), or metastases from distant carcinomas (e.g., carcinoma of the breast, lung, or kidney). This patient did not have evidence of a primary cancer in another site.

**Adenoma**
Adenoma of the ear is a benign neoplasm arising from pluripotent cells of the middle ear. Otologic findings include hearing loss, tinnitus, and a soft-tissue mass in the middle ear. Typically, there is no bony destruction, and MRI reveals brain-
tissue intensity on T₂-weighted and T₁-weighted images, similar to this case. However, extension of the mass beyond the middle ear and mastoid is a feature that is not consistent with adenoma.

Langerhans’-Cell Histiocytosis
Langerhans’-cell histiocytosis (eosinophilic granuloma) may present with unifocal or multifocal bone involvement, including the temporal bone. It is most common in children and young adults but has been reported in persons of all ages. Otologic findings include otorrhea, a perforated eardrum, and failure to respond to antibiotic treatment. The absence of a lytic bone lesion makes this diagnosis unlikely.

Squamous-Cell Carcinoma
The most common primary malignant tumor of the temporal bone in adults is squamous-cell carcinoma. It typically presents as escalating otalgia in a patient with previously painless chronic otorrhea. Bleeding, hearing loss, and cranial neuropathies, such as facial-nerve paresis may occur. The absence of chronic otorrhea and bony destruction makes this diagnosis unlikely. Other carcinomas, such as adenocarcinoma, acinic-cell carcinoma, and adenoid cystic carcinoma, are also unlikely because of this patient’s intact temporal bone.

Lymphoma and Leukemia
Primary lymphoma of bone is a rare disease that accounts for 1 to 2% of all cases of malignant lymphoma; the temporal bone is a very uncommon site, with fewer than 20 cases reported in the literature. The most common presenting symptoms are otalgia, hearing loss, and ear fullness. Some patients present with facial-nerve palsy, a localized swelling, or a mass. Bone lymphoma may extend into the soft tissue of the ear canal but tends to be destructive of bone. Nonetheless, this diagnosis is a consideration.

Myeloid sarcoma, also known as granulocytic sarcoma or chloroma, is a solid extramedullary collection of myeloid leukemia cells. It may precede acute myeloid leukemia (AML) by months or years; more commonly, it occurs concurrently with or during remission from leukemia. In the English literature, I found only five cases (two in adults) of myeloid sarcoma of the temporal region in patients without a history of leukemia. Presenting symptoms include auricular or external-canal lesions, hyperemia and thickness of the eardrum, middle-ear effusion, otitis media, and hearing loss, all of which this patient had. A major risk factor is a history of a myeloproliferative neoplasm, which this patient also had. In addition, her latest blood count showed rare immature myeloid cells and blasts. Myeloid sarcoma is typically not associated with bony destruction, and characteristics on MRI are similar to the features found on MRI in this case.

SUMMARY
In this patient with a history of a myeloproliferative neoplasm and an enlarging mass in the ear canal, with the involvement of bone, I ultimately thought that myeloid sarcoma was the most likely diagnosis, and I performed a biopsy of the postauricular mass. At operation, a firm, infiltrative subcutaneous mass, hypervascular markings in the concha, and narrowing of the external auditory canal were apparent (Fig. 2A).

Dr. Konstantina M. Stankovic’s Diagnosis
Myeloid sarcoma of the ear, with recurrent otitis media and externa, in a patient with essential thrombocytopenia.

Pathologic Discussion
Dr. Robert P. Hasserjian: Histologic sections from the postauricular biopsy specimen show a cellular neoplasm composed of large, dyshesive cells with oval or folded vesicular nuclei and moderate amounts of pale cytoplasm (Fig. 2B). The tumor infiltrates adipose tissue and surrounds peripheral nerves. Immunohistochemical stains reveal that the tumor cells are negative for cytokeratin, leukocyte common antigen (CD45), the B-cell markers Pax-5 and CD20, and the T-cell marker CD3. The tumor cells express the myeloid markers lysozyme and myeloperoxidase (Fig. 2C) and are positive for CD34 (Fig. 2D) and CD117, both associated with myeloid blasts. These findings confirm a diagnosis of myeloid sarcoma.

Myeloid sarcoma is a mass-forming infiltrate of myeloblasts in a tissue site other than the bone marrow. It usually occurs concurrently with a diagnosis or relapse of AML that involves the bone marrow, but in about one third of cases, it occurs without evidence of AML. The most common sites of involvement are the skin and lymph nodes, but it has been reported in many other
anatomical sites, including the testes, gastrointestinal tract, bones, soft tissues, and orbits.\textsuperscript{18,19} Myeloid sarcoma is a manifestation of a systemic myeloid neoplasm; a diagnosis of myeloid sarcoma is considered equivalent to a diagnosis of AML. Even if the bone marrow is uninvolved on histologic examination, localized therapy is inadequate. A bone marrow–biopsy specimen from this patient revealed AML (Fig. 3A). Cytogenetic analysis of the bone marrow revealed a complex abnormal karyotype including monosomies of chromosomes 5, 15, and 22 and additional material on chromosomes 7q and 17p.

Hematopoietic stem-cell neoplasms (myelodysplastic syndromes, myeloproliferative neoplasms, and overlapping myelodysplastic–myeloproliferative neoplasms) can progress to AML. Among the myeloproliferative neoplasms, the risk of AML varies by disease type (Table 2). This patient had essential thrombocythemia, which carries a relatively low risk. Cytotoxic chemotherapy and radiotherapy used to treat hematologic neoplasms can also predispose patients to the development of therapy-related AML. This patient was treated with hydroxyurea, a ribonucleotide reductase inhibitor that inhibits DNA synthesis, to control her elevated platelet count. It is controversial whether hydroxyurea increases the risk of a patient’s developing AML.\textsuperscript{20-22} However, in one series, 17p deletions or unbal-

**Figure 2. A Subcutaneous Mass in the External Ear.**

On intraoperative clinical examination (Panel A), the concha had hypervascular markings and the external auditory canal was narrowed by a subcutaneous mass (T denotes tragus, and H helix). Pathological examination of the biopsy specimen of the postauricular mass showed that the soft tissue contained a diffuse infiltrate composed of large cells with oval, vesicular nuclei and moderately abundant pale cytoplasm (Panel B, hematoxylin and eosin). On immunohistochemical staining, the large cells were positive for myeloperoxidase (Panel C), an enzyme indicating myeloid lineage. The cells were also positive for CD34, a marker of hematopoietic-cell precursors (Panel D).
Ancestral translocations were frequent in AML that developed in patients with essential thrombocytemia who were treated with hydroxyurea and were not observed in such patients who did not receive hydroxyurea.\textsuperscript{22} This patient's AML contained a 17p cytogenetic abnormality, which may implicate hydroxyurea in its cause.

AML is classified according to the World Health Organization scheme, based on a combination of morphologic, clinical, and cytogenetic features that defines disease entities with different clinical behaviors. AML with myelodysplasia-related changes is a type of AML with a very poor prognosis. It is characterized by the presence of morphologic dysplasia in the nonleukemic hematopoietic elements, a history of a myelodysplastic syndrome or a myelodysplastic–myeloproliferative neoplasm, or any of several specific findings on routine cytogenetic analysis.\textsuperscript{26} Although a previous myeloproliferative neoplasm (which this patient had) is not included in the spectrum of this type of AML, the complex karyotype qualifies this patient's neoplasm as AML with myelodysplasia-related changes. Aside from the karyotype, other adverse prognostic features of this type of AML include its presentation as myeloid sarcoma and the antecedent essential thrombocytemia.\textsuperscript{27} In one recent series, the median survival of patients with myeloproliferative neoplasms (excluding chronic myelogenous leukemia) in whom AML developed was only 5 months after the diagnosis of AML, and allogeneic stem-cell transplantation represented the only therapy associated with long-term survival.\textsuperscript{28}
Myeloid and erythroid elements matured normally, and there was no increase in the number of blasts. The complete blood count at this time showed leukocytosis, anemia, and thrombocytosis, indicating remission from AML but persistent essential thrombocythemia. The postauricular mass resolved; it recurred 3 months later and there was no increase in the number of blasts. The complete blood count at this time showed leukocytosis, anemia, and thrombocytosis, indicating remission from AML but persistent essential thrombocythemia. The postauricular mass resolved; it recurred 3 months later and was treated with high-dose cytarabine and external-beam radiation, with resolution. The patient received a bone marrow transplant related changes.

This case was discussed at the Otolaryngology Grand Rounds, MEEI, September 10, 2009. We thank Drs. Joseph Nadol, Marlene Durand, and Neil Weiner for assistance in preparing the case history and Dr. Durand for helpful comments on the manuscript.

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

MANAGEMENT AND FOLLOW-UP
This patient received standard AML induction therapy with idarubicin and cytarabine. Examination of a specimen from a bone marrow biopsy obtained 6 weeks after induction therapy revealed an increased number of megakaryocytes, occurring in clusters, with enlarged, hyperchromatic nuclei and complex nuclear lobation (Fig. 3B). Myeloid and erythroid elements matured normally, and there was no increase in the number of blasts. The complete blood count at this time showed leukocytosis, anemia, and thrombocytosis, indicating remission from AML but persistent essential thrombocythemia. The postauricular mass resolved; it recurred 3 months later and was treated with high-dose cytarabine and external-beam radiation, with resolution. The patient received an allogeneic bone marrow transplant from a HLA-matched, unrelated donor at the Dana–Farber Cancer Institute 4 months after her initial admission. Within 2 months, myeloid sarcoma recurred in the breast, sciotic notch, and left temporal region, and blasts appeared in the blood. She was referred to hospice and died 8 months after the initial diagnosis.

Dr. Stankovic: Although myeloid sarcoma as a cause of recurrent otitis media is highly unusual, this case does remind us that when an adult with no history of ear problems has recurrent or refractory otitis, evaluation for evidence of an anatomical abnormality or possibly immune-system compromise should be considered.

ANATOMICAL DIAGNOSES
Myeloid sarcoma of the ear in a patient with essential thrombocythemia.

Acute myeloid leukemia with myelodysplasia-related changes.

REFERENCES

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