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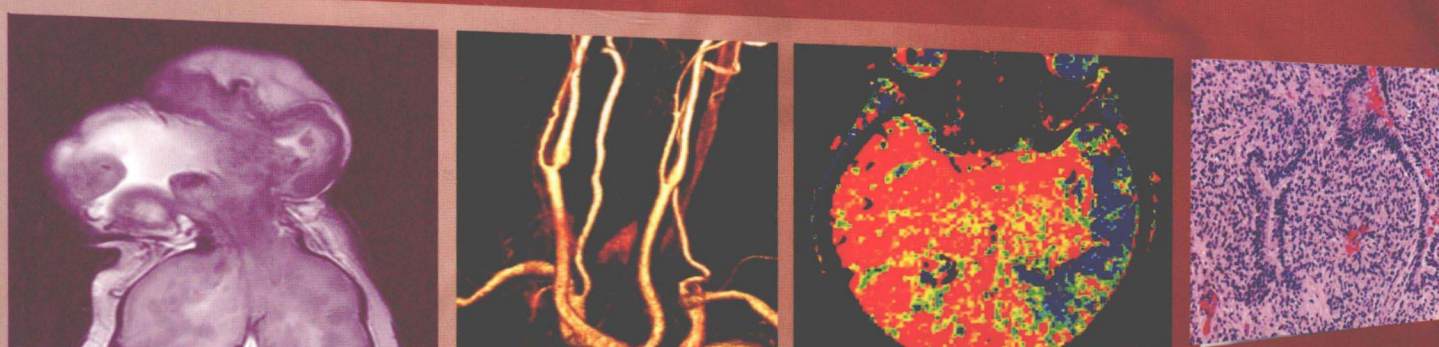
Neuroimaging

A TEACHING FILE



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A Teaching File

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Neuroimaging

A Teaching File

For Omoniyi, Olufemi, Ayokunle, Oluwadamilola, Temitope, and Opeyemi; the best children in the world.

MFO

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Teaching files are one of the hallmarks of education in radiology. When there was a need for a comprehensive series to provide the resident and practicing radiologist with the kind of personal consultation with the experts normally found only in the setting of a teaching hospital, Wolters Kluwer was proud to have created a series that answers this need.

Actual cases have been culled from extensive teaching files in major medical centers. The discussions presented mimic those performed on a daily basis between residents and faculty members in all radiology departments.

This series is designed so that each case can be studied as an unknown. A consistent format is used to present each case. A brief clinical history is given, followed by several images. Then, relevant findings, differential diagnosis, and final diagnosis are given, followed by a discussion of the case. The authors thereby guide the reader through the interpretation of each case.

Last year we made additional changes to the series. Cases have been randomized to better prepare the reader for the challenges of the clinical setting. In addition, to answer the growing demand for electronic content, we have included more cases online, which has left us, in turn, able to offer a more cost-effective product.

We hope that this series will continue to be a trusted teaching tool for radiologists at any stage of training or practice and that it will also be a benefit to clinicians whose patients undergo these imaging studies.

The Publisher

I have always maintained a teaching file from my days as a radiology resident. My teaching materials always came from these files which are updated from time to time. It is therefore a great privilege to be asked to lead a group of great contributors to put together some of these cases that we share with you here in a teaching file format. The presentation follows the new format of the teaching file in the Wolters Kluwer series, incorporating the relevant imaging findings with the clinical information in a case-based format. The Question-and-Answer segments, the Reporting Responsibilities, and the "What the Treating Physician Needs to Know" segments were the most difficult for me to write. I actually enjoyed doing them. These sections have turned out to be as informative as the Discussion section. I have tried to use the ACR communication guide as a guide in composing the Reporting Responsibilities. However, communication continues to evolve in clinical practice with the emergence of the electronic medical record. Individual practice should decide what is feasible with regard to the communication practice. Where possible we have included the specific clinical scenario leading to the diagnosis in each case so that the path to diagnosis would be clear. While this is a guide, the path to diagnosis may not necessarily be the same in your own practice. This may change from time to time depending on the available clinical information. We have collaborated with our colleagues from the clinical services and pathology where possible in some of the chapters. I believe that we get the best result through interdisciplinary collaborations in the management of our patients.

This title should be useful to residents in radiology and the busy general radiologists who want to quickly look up a case similar to a problem case they may have. In this regard, we have elected to present each case discussion starting with the imaging findings and discussion of the differential diagnosis to be closely followed by the pathology, clinical presentation, and treatment where necessary so that our readers would be able to first compare the imaging findings with their cases before diving into the clinical findings. I also think this book will be useful to the neurology and psychiatry residents doing elective rotations in neuroimaging. Students doing observership in neuroimaging and medical students doing neuroscience rotation may also find this book useful. I have been privileged to mentor many of these residents and students. We have included some cases on normal anatomy. We have also covered how advanced imaging could be helpful in the differential diagnosis in relevant situations. We have been as comprehensive as possible in each case considering the constraint of space. I think we have covered most of the common cases in neuroimaging practice. Several cases with several imaging features are discussed in several places resulting in some form of duplication which I consider necessary in order to do justice to the more common imaging features of such cases.

Matthew F. Omojola

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My special thanks to my colleagues in the department of Neurological Sciences at the University of Nebraska Medical Center for their support throughout my time at the UNMC. They have contributed immensely to my longevity at this institution. I would like to thank the many Neurology and Psychiatry residents who took electives with me for the impetus to be a part of this project. I am grateful to all our colleagues, who took care of the patients presented here, for their contributions to the well-being of our patients.

I want to remember the late Jonathan Pine, with whom I started out this project at LWW, for his kindness, support, and dedication to duty while he was under tremendous burden, which I was not aware off until the very end. I'd also like to thank Charley Mitchell, who originated the project but left LWW before we could actualize it. Charley was very supportive of this project, and I thank him for his comforting words during the initial break in the project. This project could not have been completed without the support of many people at LWW, including Amy Dinkel and her group. I learned a lot from my development editor, Mary Beth Murphy.

Deborah Klein sought and pulled most of the references for this book. Drs. Terri Love and Lisa Wheelock supplied some images on posterior fossa congenital lesions, some of which I used in this book. I am also grateful to many other colleagues who contributed images.

I owe a debt of gratitude to my co-editor Dr. Mauricio Castillo, my mentor and a great friend without whom this work could not have been done. Mauricio has served our profession and specialty with distinction in so many ways. Those of us who have benefited from his knowledge, leadership and wisdom should be thankful for knowing him. I would like to thank Allan Fox, MD for my foundation in Neuroradiology. I am indebted to my children, Omoniyi, Olufemi, Ayokunle, Oluwadamilola, Temitope and Opeyemi; my cheerleaders for their love, constant encouragement and support. I don't know what I could have done without them! I am grateful to my wife, Jumoke, for her support.

Matthew F. Omojola

CASE 1

CLINICAL HISTORY *8-year-old male with malignant astrocytoma of the corpus callosum treated with combination therapy.*

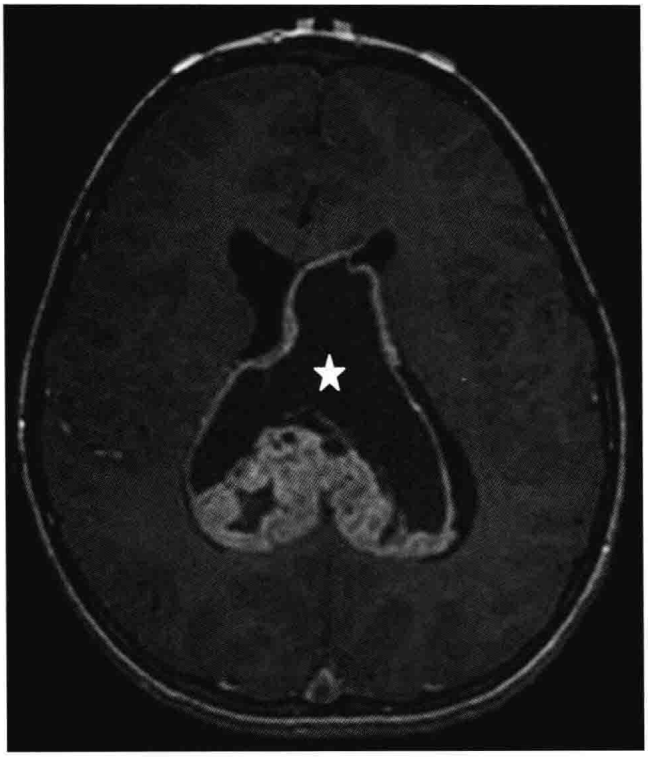

FIGURE 1-1

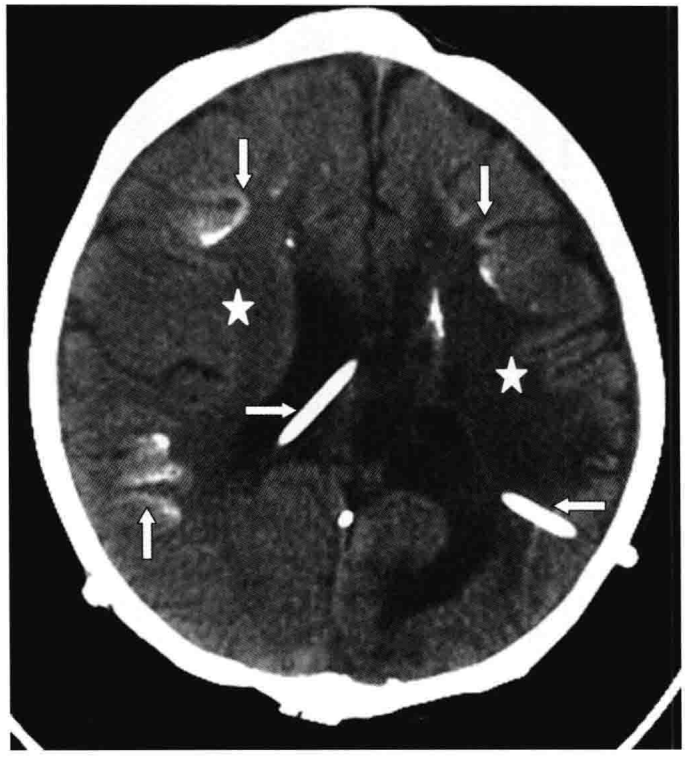

FIGURE 1-2

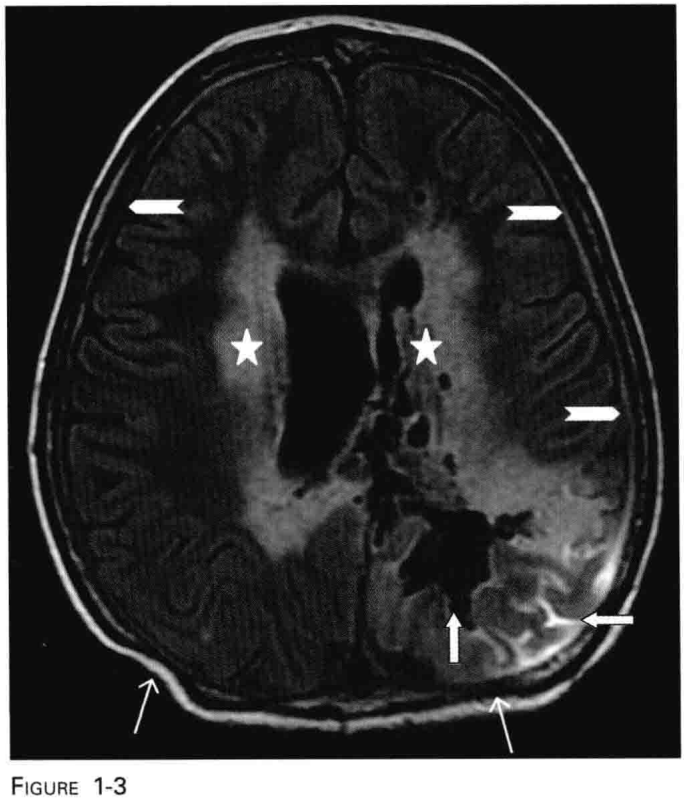

FIGURE 1-3

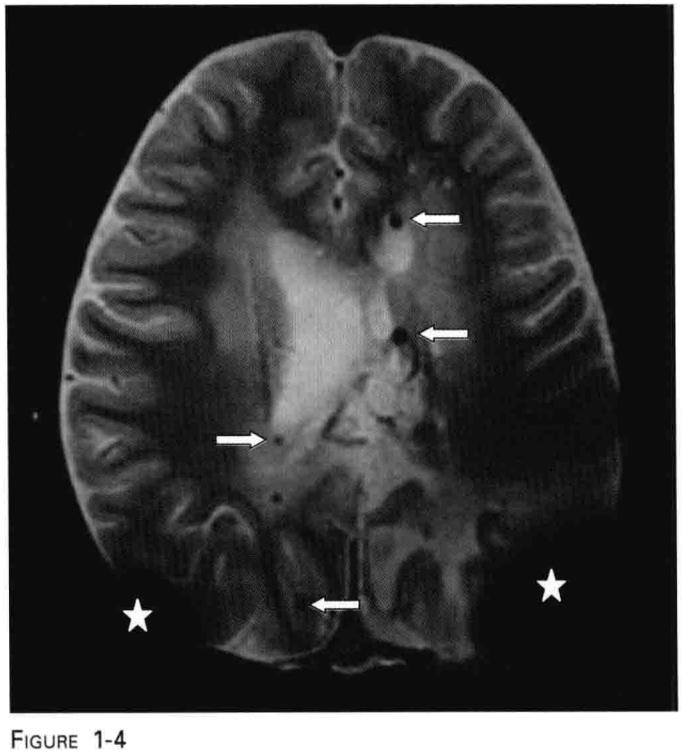

FIGURE 1-4

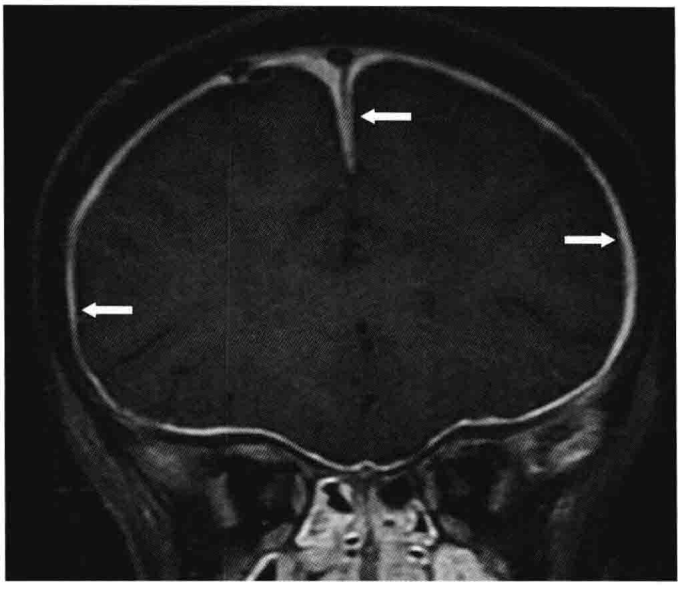

FIGURE 1-5

FINDINGS Figure 1-1. Axial treatment planning post-contrast T1WI through the lateral ventricles. There is a large partially cystic and solid mass (WHO IV) astrocytoma (star) of the corpus callosum bulging into the lateral ventricles. Figure 1-2. Axial NCCT through the corona radiata about 2 years into his treatment which included surgical removal of the tumor via bilateral parietal craniotomies, radiation treatment, and chemotherapy. There are bilateral ventriculoperitoneal (VP) shunts (transverse arrows), multifocal subcortical calcifications (vertical arrows), and bilateral periventricular smudgy white matter (WM) hypodensities (stars). Figure 1-3. Axial FLAIR through the corona radiata. There is bilateral confluent WM hyperintensity (stars). The left parasagittal parietal surgical cavity (vertical arrow) represents part of the surgical tract to the original tumor. There are hyperintensities in the left parietal sulci (transverse arrow) produced by the VP shunt susceptibility artifact. There is thickening of the dura (chevrons). The craniotomy flap is present posteriorly (line arrows). Figure 1-4. Axial GRE through the centrum semiovale. There are bilateral large signal void artifacts from the VP shunts (stars). There are multiple punctate hypointensities in bilateral centrum semiovale WM (transverse arrows) consistent with telangiectasia or microhemorrhages or calcifications. Figure 1-5. Coronal post-contrast T1WI through the frontal lobes. There is smooth pachymeningeal enhancement (arrows) which is circumferential around the cerebral and cerebellar hemispheres.

DIFFERENTIAL DIAGNOSIS N/A.

DIAGNOSIS Treatment-related changes.

DISCUSSION This case illustrates some of the changes that can occur following intervention in the management of intracranial pathology. Multimodal interventions happen very frequently in clinical management, and we have to be familiar with the effects of these on images. Some of these may mimic abnormalities, some are abnormalities, and some are just life support instruments casting artifacts. Surgical intervention usually starts with a craniotomy (a cranial vault bone flap raised to allow entry and replaced at the end of the intervention) or craniectomy (a bone flap raised and not replaced or may be replaced at a later date). The margins of these flaps are seen as defects in the cranial vault. The craniectomy could produce a sunken effect on the scalp. Because of foreign bodies at the margins of the craniotomy, it is easier to identify on the GRE sequence where the susceptibility effect is greatest. These margins are usually easy to see on CT bone windows.

If there is hydrocephalus or one is expected, a ventricular drain or a VP shunt may be placed. The drain or shunt should pass through the lateral ventricle for it to function properly. Shunt complications may include malfunction such as overdrainage or inadequate drainage which could result in slit ventricles or hydrocephalus, respectively, shunt infection with ventriculitis and ependymal contrast enhancement with intraventricular debris, extraaxial collections particularly with overdrainage and intraventricular hematoma (IVH) during insertion or revision of the shunt. The valve of the shunt may produce signal void artifacts peripherally on the cranial vault particularly on the GRE and DWI, and sulcal hyperintensities on FLAIR images.

Confluent WM T2 hyperintensity or leukoencephalopathy is a fairly common complication of radiation treatment and chemotherapy. In the acute phase, this probably relates to edema, while on the long term, there are demyelination, axonal loss, spongiosis, gliosis, and vasculopathy. Decreased *N*-acetyl aspartate (NAA) has been demonstrated on MR spectroscopy within the WM hyperintensity. Brain necrosis presenting with contrast enhancement could mimic neoplasm. MRI perfusion study may be useful for distinguishing radiation necrosis (low to poor relative Cerebral Blood Volume [rCBV]) from tumor (high rCBV). Brain volume loss with widening of the sulci and enlargement of the ventricles is not uncommon following treatment. Punctate GRE hypointensities within the WM may represent calcifications, microhemorrhage, or telangiectasia. CT usually differentiates calcifications (dense on CT) from microhemorrhages and telangiectasia (usually not seen on CT). Mineralizing angiopathy resulting in calcifications is a common complication of tumor treatment. All these changes begin as early as within 3 months of completion of treatment. Mild pachymeningeal enhancement may be seen following surgery, and this could disappear subsequently. Avid and robust pachymeningeal enhancement may suggest intracranial hypotension particularly if there is associated hind brain sagging or pseudo Chiari I. Nodular pachymeningeal configuration may indicate tumor seeding. Some of these changes may or may not be symptomatic.

Question for Further Thought

1. Which is the best imaging technique for the evaluation of treatment changes?

Reporting Responsibilities

Significant changes following brain tumor treatment should be reported directly. Complications of shunts usually require immediate attention. New tumors or new areas of contrast enhancement usually require further evaluation with DWI if one has not been obtained, perfusion studies, and MR spectroscopy.

What the Treating Physician Needs to Know

- What is new? Where and how severe?
- Are life support tubes doing what they should be doing?
- What other investigations should be done to clarify the new findings?

Answer

1. While CT may be able to demonstrate all the changes discussed here, MRI offers the best imaging technique to evaluate posttreatment changes. If the consideration is whether a shunt or EVD is functioning or not, CT is adequate. Parenchymal changes, however, are best evaluated by MRI particularly with other available procedures such as MR spectroscopy, perfusion, and the multiplanar capability of MRI.

CASE 2

CLINICAL HISTORY *22-year-old male with mental status changes.*

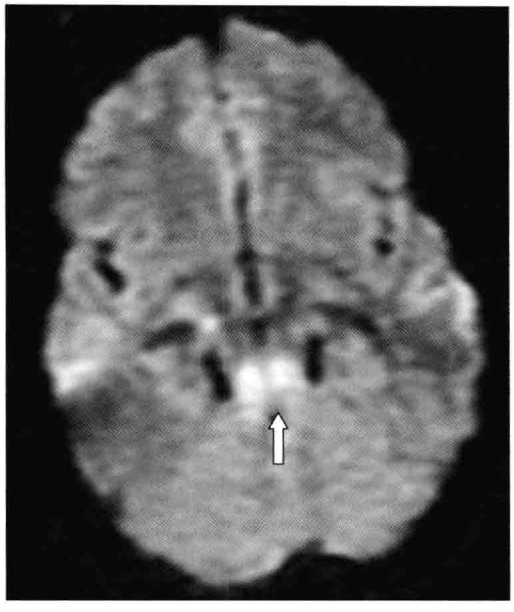

FIGURE 2-1

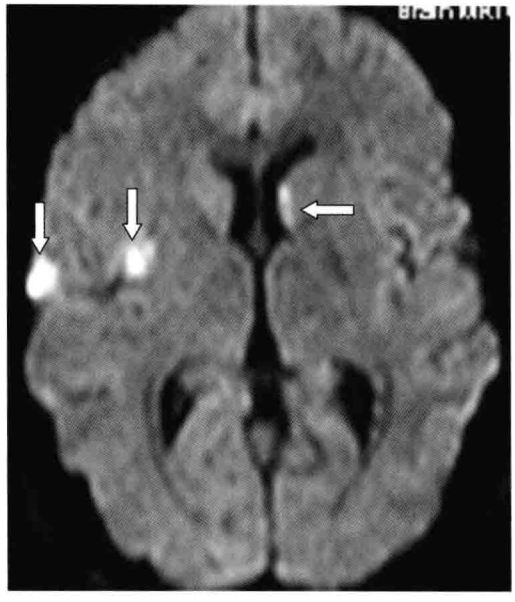

FIGURE 2-2

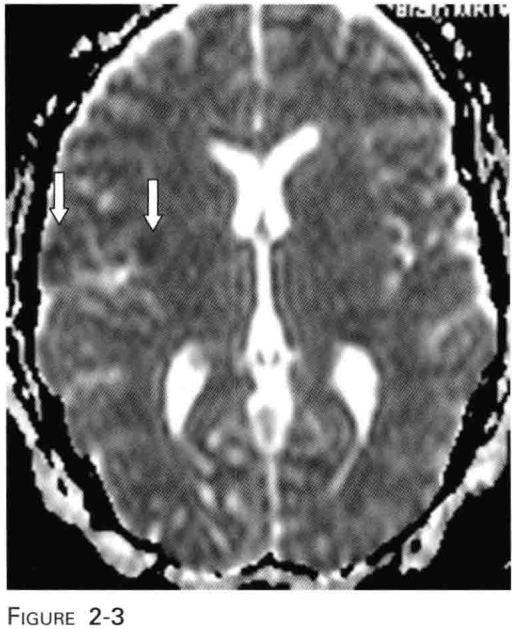

FIGURE 2-3

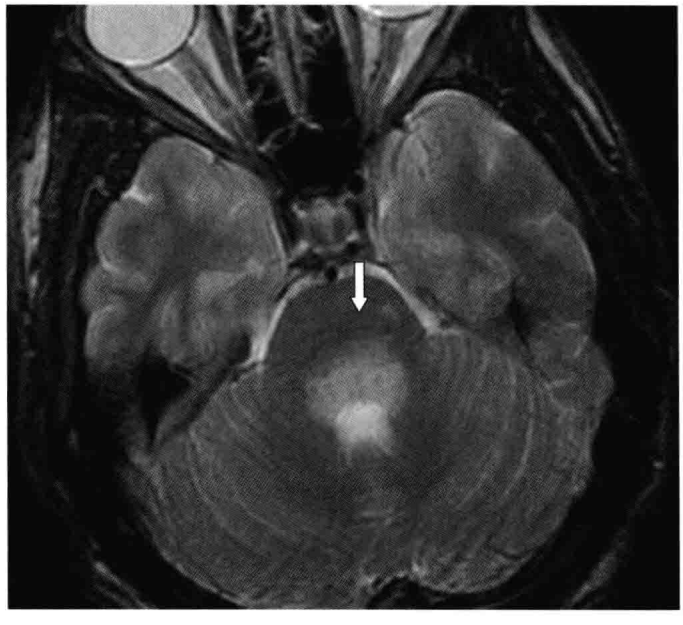

FIGURE 2-4

FINDINGS Figure 2-1. Axial DWI through the midbrain. There is hyperintensity posteriorly in the midbrain (arrow). ADC map (not shown) did not show low ADC. Figures 2-2 and 2-3. Axial DWI with corresponding ADC map through the basal ganglia. There are multifocal areas of restricted diffusion (arrows) in the left caudate nucleus, right insula, and operculum. Figures 2-4 and 2-5. Axial T2WI through the pons and midbrain, respectively. There is confluent hyperintensity in the pontine tegmentum extending into midbrain tegmentum and tectum (arrows). Inferior extension of the lesion to the medulla is present (not shown). Figure 2-6. Axial FLAIR through the pons 4 months later. There is minimal residual hyperintensity in the tegmentum (arrow). There is no contrast enhancement on the initial and follow-up images in any of the areas involved.

DIFFERENTIAL DIAGNOSIS Multifocal infarcts, demyelinating process, encephalitis, brainstem glioma, osmotic demyelination.

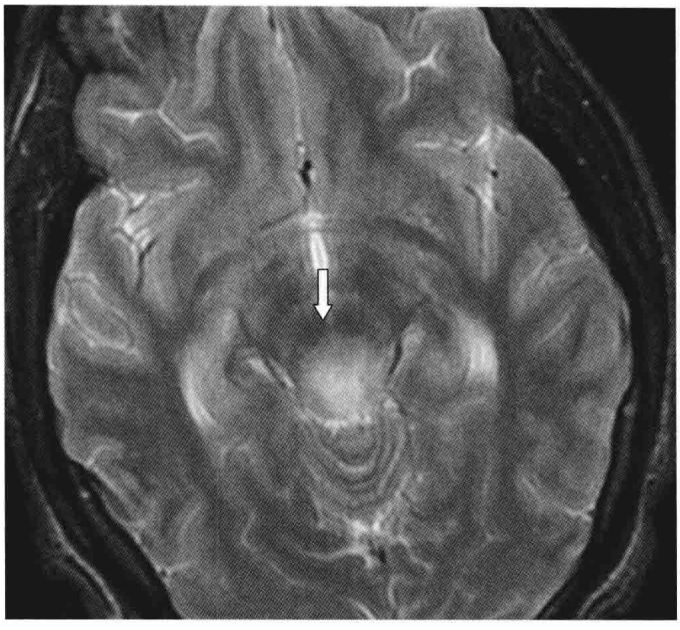

FIGURE 2-5

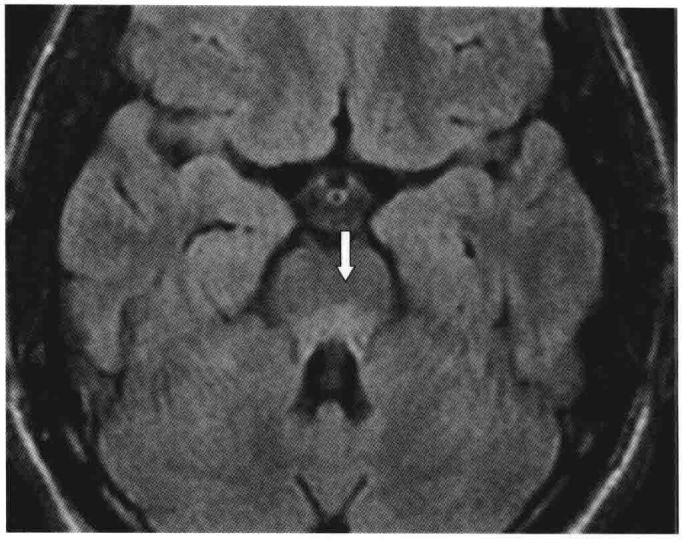

FIGURE 2-6

DIAGNOSIS Brainstem encephalitis (presumed Bickerstaff brainstem encephalitis [BBE]).

DISCUSSION BBE is a rare autoimmune brainstem encephalitis of unknown etiology. The MRI changes of BBE which is present in about 30% of the patients are a longitudinal confluent T2 hyperintensity in the brainstem extending from the medulla to the midbrain, usually without restricted diffusion or contrast enhancement, but some expansion of the brainstem. It has been described by some as vasogenic edema, while some have reported areas of restricted diffusion in some cases. In most of the images published, the tegmentum has been the region most affected. Concomitant involvement of the cerebellum and supratentorial structures has been reported. A negative MRI does not exclude the diagnosis if the clinical criteria are met. The diffuse nature of the changes and lack of restricted diffusion exclude brainstem infarct. It may be difficult to completely exclude other demyelinating processes. The wide age range does not fit with brainstem glioma. Osmotic myelinolysis usually spares the periphery and is mostly confined to the pons. Usually there is complete recovery with resolution of the signal changes in BBE as in this case.

The clinical criteria for BBE include ataxia with bilateral ophthalmoplegia. There is usually disturbed consciousness, bulbar palsy, pupillary changes, facial weakness, and hyperreflexia. Tetraparesis could be present in about 50% of the patients. The diagnosis is supported by the presence of anti-GQ1b antibodies and an abnormal brain MRI. However, absence of these does not exclude the diagnosis. There is clinical overlap between BBE, Miller Fisher syndrome (MFS), Guillain-Barré syndrome (GBS), and acute demyelinating encephalomyelitis (ADEM). There is usually a preceding infection resulting in a prodromal stage. Treatment is by corticosteroids, airways management, intravenous immunoglobulin, and plasmapheresis.

Question for Further Thought

1. Which are the organisms implicated in the prodromal phase of BBE?

Reporting Responsibilities

Direct reporting is indicated in view of the differential and the necessity for aggressive management in this otherwise curable illness.

What the Treating Physician Needs to Know

- Extent of disease
- Reasonable differential diagnosis

Answer

1. BBE has been reported following infection with several organisms including *Mycoplasma pneumoniae*, *cytomegalovirus*, *Haemophilus influenzae*, and *Campylobacter jejuni*. The exact pathogenesis is unknown. BBE is associated with the presence of antiganglioside antibody, (anti-GQ1b), which is also present in other diseases with similar prodromal phase such as MFS, ADEM, and GBS indicating similar pathogenetic mechanism of these diseases.

CASE 3

CLINICAL HISTORY *54-year-old female presenting with headache and right facial nerve weakness.*

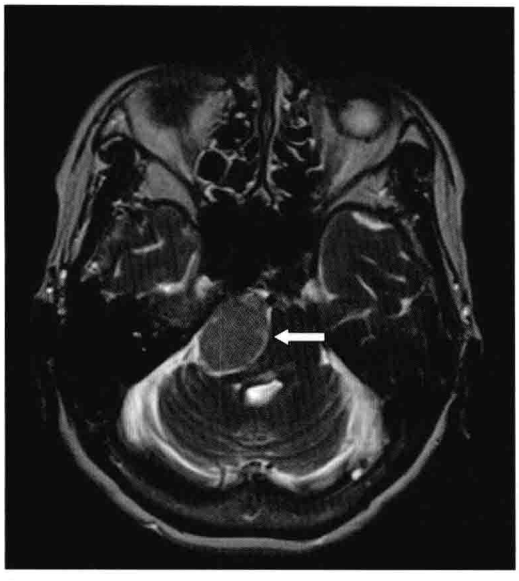

FIGURE 3-1

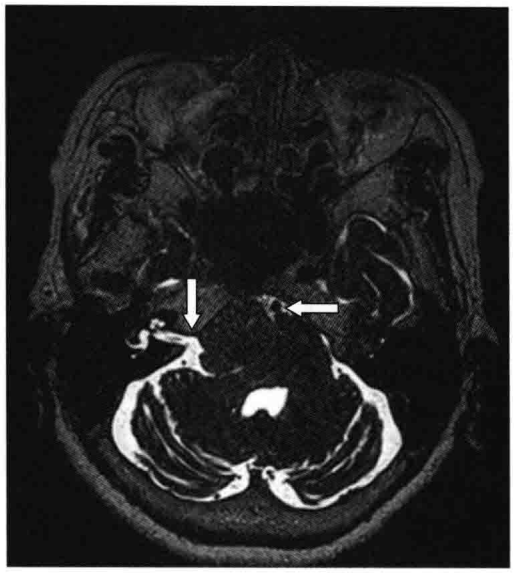

FIGURE 3-2

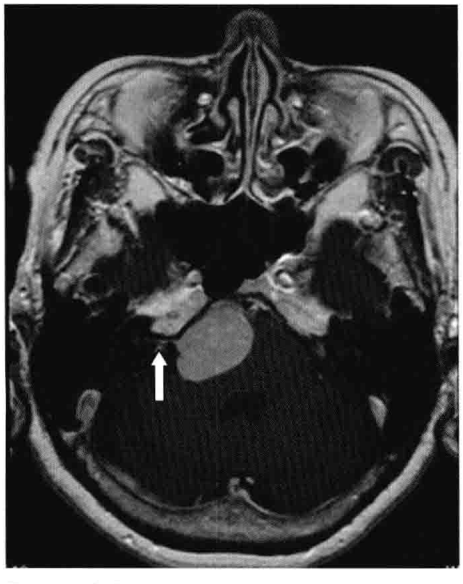

FIGURE 3-3

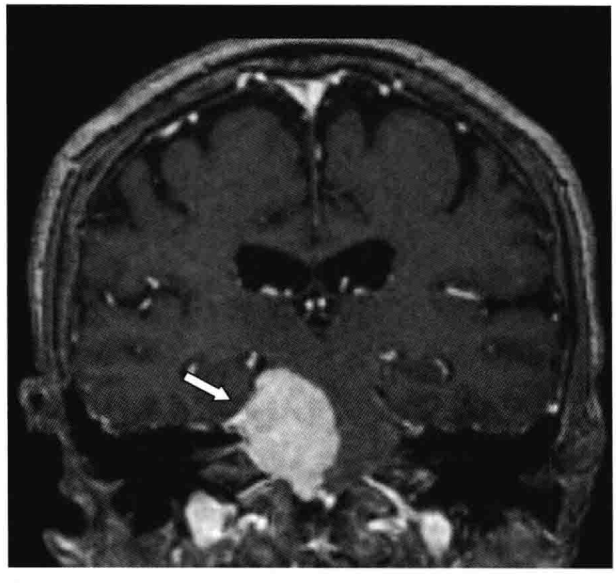

FIGURE 3-4

FINDINGS Figure 3-1. Axial T2WI through the cerebellopontine angle (CPA). There is a mass at the right CPA with a cleft of cerebrospinal fluid (CSF) separating it from the brainstem (arrow), confirming its extraaxial location. The adjacent brainstem is compressed and displaced to the left, demonstrating mild edema. The mass is ovoid, sharply circumscribed, and hypointense. Figure 3-2. Axial 3D volumetric heavily T2WI. This better demonstrates the relationship of the mass to the adjacent neural and vascular structures including cranial nerves VII and VIII (vertical arrow) and the basilar artery (BA) (transverse arrow). Figure 3-3. Axial post-contrast T1WI reveals a broad-based mass with intense, homogeneous enhancement with a dural tail extending into the right internal auditory canal (IAC) (arrow). Figure 3-4. Coronal post-contrast T1WI confirms the dural-based lesion along the tentorium cerebelli (arrow).

DIFFERENTIAL DIAGNOSIS Vestibular schwannoma, dural metastasis, hemangiopericytoma, meningioma and epidermoid.