Nontraumatic Neuroemergencies

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Several clinical presentations require emergent neuroimaging to determine the cause of the neurological deficit and to institute appropriate therapy. Time is critical because neurons that are lost cannot be replaced. Generally, the clinical symptoms are due to ischemia and compression or destruction of neural elements. The two primary imaging modalities for the CNS are CT and MRI. CT is fast and can readily visualize fractures, hemorrhage, and foreign bodies. Otherwise, in patients who can cooperate for the longer imaging study, MRI provides better contrast resolution and has higher specificity for most CNS diseases. The five major categories of nontraumatic neuroemergencies are discussed below [1].

Acute Focal Neurological Deficit

Arterial Thrombosis/Occlusion

Thrombotic strokes may occur abruptly but the clinical picture often shows gradual worsening over the first few hours. Primary causes of arterial thrombosis include atherosclerosis, hypercoagulable states, arteritis, and dissection. Secondary compromise of vascular structures can result from traumatic injury, intracranial mass effect, neoplastic encasement, meningeal processes, and vasospasm (Fig. 1).

Arterial Embolism

Embolic strokes characteristically have a very abrupt onset. After a number of hours, there may be sudden improvement in symptoms as the embolus lyses and travels more distally. The source of the embolus is usually either the heart (patients with atrial fibrillation or previous myocardial infarction) or ulcerated plaques at the carotid bifurcation in the neck.

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Arterial Dissection

Relatively minor trauma is sufficient to cause a dissection, or it can be spontaneous. The MRA may demonstrate complete occlusion or only narrowing of the arterial lumen. Spin-echo images, especially T1-weighted with fat suppression, should also be obtained because they are very sensitive for detecting the intramural hemorrhage. The typical appearance is an oval-shaped hyperintensity with an eccentrically placed flow void. The MRA is also very useful for following a dissection to look for recanalization of a complete occlusion, resolution of the vascular compromise caused by the intramural thrombus, or development of a pseudoaneurysm.

Brain Hemorrhage

Localized hemorrhages into the brain may present with a focal neurological deficit. Most of these are caused by vascular malformations. The four types of vascular lesions include AV malformation, cavernous angioma, capillary telangiectasia, and venous malformation. AV malformations are more likely to present acutely. Hemorrhage into tumors or infarcts is not uncommon, but patients usually have symptoms related to the underlying lesion. Hypertensive hemorrhages are often large and deep within the brain and produce more global neurological deficits.

Hypotension/Hypoxia

Hypotension can be cardiac in origin or result from blood volume loss or septic shock. Anoxia/hypoxia events are usually related to respiratory compromise from severe lung disease, perinatal problems, near drowning, high altitude, carbon monoxide inhalation, or CNS-mediated effects.

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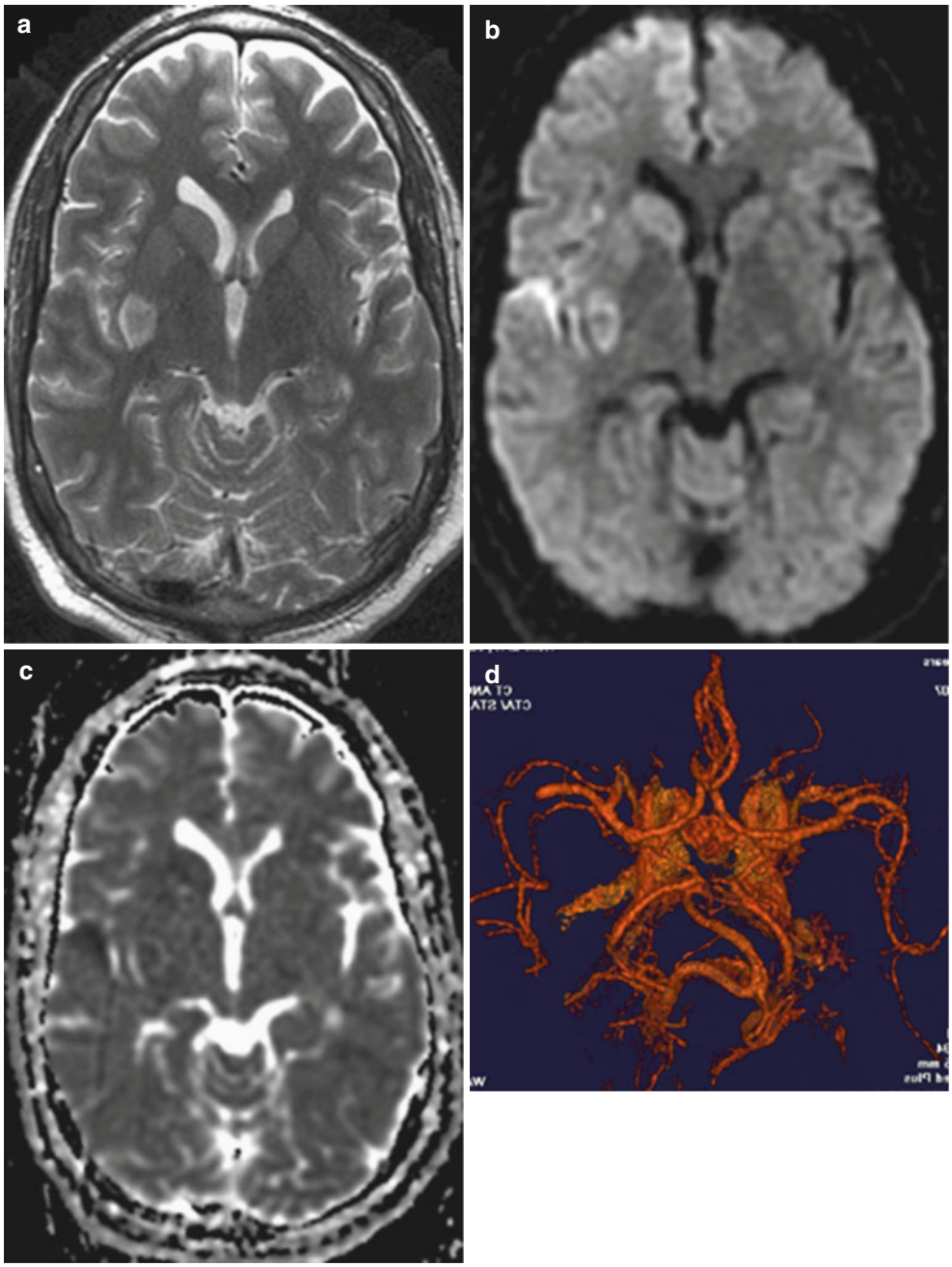

Fig. 1 Acute cerebral infarct secondary to a right MCA embolic occlusion. (**a**) Axial T2 image shows focal hyperintensity in the posterior right putamen and external capsule. (**b**, **c**) Restricted diffusion on the DWI (**b**) is confirmed by hypointensity on the ADC map (**c**). The infarct extends laterally into the peri-sylvian cortex. (**d**) CT angiogram reveals occlusion of the right middle cerebral artery

Venous/Sinus Occlusion

Thrombosis of the cerebral venous sinuses has multiple etiologies, including hypercoagulable states, pregnancy, sepsis, dehydration, paranasal sinus infection, and neoplastic invasion. Occlusion of the venous sinuses results in cerebral venous engorgement, brain swelling, and increased intracranial pressure. If the thrombosis extends retrograde and involves the cortical veins, secondary cerebral infarction can occur.

Acute thrombus is hyperdense on CT and may be detected within one of the major sinuses or cortical veins. The other classic sign is the "empty delta" sign due to nonfilling of the superior sagittal sinus on a contrast scan. Nonetheless, MR is far superior for diagnosing abnormalities of the cerebral veins and sinuses. Normally, the dural sinuses have sufficient flow to exhibit a flow void. If that flow void is missing or if the sinuses are hyperintense, thrombosis should be suspected. One must be careful to exclude the possibility of any inflow enhancement effect. The diagnosis must be confirmed with gradient-echo techniques or MR angiography. Phase-contrast or Gd-enhanced time-of-flight MRAs are the preferred techniques because they are not adversely affected by intraluminal clot [2].

Associated parenchymal infarcts are found in the areas of venous abnormalities, and the infarcts are often hemorrhagic because arterial perfusion is maintained to the damaged tissue. In cases of superior sagittal sinus thrombosis, the infarcts are typically bilateral and in a parasagittal location.

Cortical Mass Lesion

Any lesion that irritates the cortical neurons can be a source of seizures. Neoplasia, encephalitis, meningitis, abscess, and hemorrhage are the more common causes of new onset seizures.

Worst Headache of Life

Subarachnoid Hemorrhage

The incidence of congenital aneurysms in the general population is about 1–2 %. Clinically, a ruptured aneurysm presents as sudden onset of severe headache. In cases of subarachnoid hemorrhages, the most common aneurysms are posterior communicating, 38 %; anterior communicating, 36 %; and middle cerebral, 21 %. These three locations account for 95 % of all ruptured aneurysms. The basilar artery accounts for only 2.8 % and posterior fossa aneurysms are even less common.

The CT scan is important, first of all, to document the subarachnoid hemorrhage and to assess the amount of blood in the cisterns (Fig. 2). Detection of subarachnoid blood is very dependent on how early the scan is obtained. Data in the literature vary from 60 to 90 %. If the scan is obtained within 4–5 days, the detection rate is very high. Secondly, the CT helps localize the site of the aneurysm. This can be done by the distribution of blood within the cisterns. If conventional angiography is not available or is not planned immediately, CT angiography is very good for detecting and characterizing intracranial aneurysms. Thirdly, the CT is important to evaluate complicating factors such as cerebral hematoma, ventricular rupture, hydrocephalus, cerebral infarction, impending uncal herniation, and rebleed.

Conventional MR sequences are very insensitive for detecting subarachnoid hemorrhage. Clots within cisterns can be detected, but in general, MR is not the procedure of choice in the work-up of patients with subarachnoid hemorrhage. Due to the flow void phenomenon, aneurysms about the circle of Willis can be identified on spin-echo MR images [3]. With fluid-attenuated inversion recovery (FLAIR) sequences, the CSF is dark, so that subarachnoid hemorrhage can be seen more easily. These sequences may be helpful for detecting subarachnoid blood in the posterior fossa where CT has difficulty [4].

Acute Meningitis

Bacterial meningitis is an infection of the pia and arachnoid and adjacent cerebrospinal fluid. The most common organisms are *Haemophilus influenza*, *Neisseria meningitidis* (*meningococcus*), and *Streptococcus pneumoniae*. Patients present with fever, headache, seizures, altered consciousness, and neck stiffness. The overall mortality rate ranges from 5 to 15 % for *H. influenza* and meningococcal meningitis and as high as 30 % with streptococcal meningitis. In addition, persistent neurological deficits are found in 10 % of children after *H. influenza* meningitis and in 30 % of patients with streptococcal meningitis.

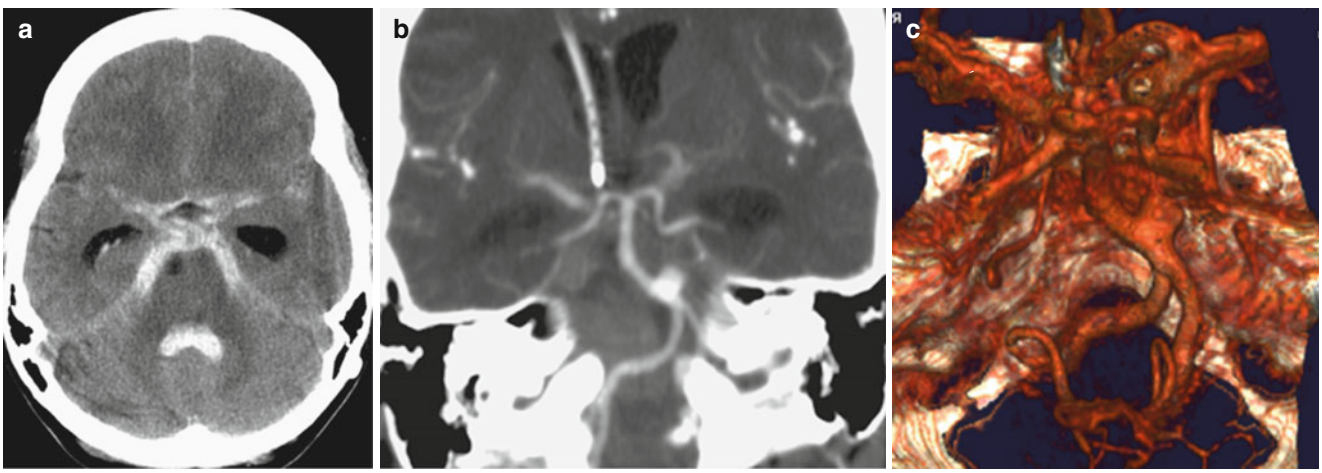

Fig. 2 Subarachnoid hemorrhage due to basilar aneurysm. (**a**) Axial CT scan reveals acute blood in the basal cisterns and fourth ventricle. Temporal horn dilatation indicates hydrocephalus. (**b**, **c**) CT angiography and a surface-shaded reconstruction demonstrate a wide-necked aneurysm at the left vertebrobasilar junction

The ability of nonenhanced MR to image meningitis is extremely limited, and a majority of cases is normal or has mild hydrocephalus. In severe cases, the basal cisterns may be completely obliterated, with high signal intensity replacing the normal CSF signal on FLAIR images. Intermediate signal intensity may be seen in the basal cisterns on T1-weighted images in these cases. Meningeal enhancement often is not present, unless a chronic infection develops [5].

Fungal organisms can start as a meningitis or cerebral abscess or can invade directly from an extracranial compartment. Coccidioidomycosis is endemic to the central valley regions of California and desert areas of the southwestern United States. Infection occurs by inhalation of dust from soil usually heavily infected with arthrospores. Primary coccidioidomycosis, a pulmonary infection, is followed by dissemination in only about 0.2 % of immunocompetent patients. Central nervous system involvement most often represents a meningitis, but cerebral abscess and granuloma formation can also occur [6]. Other fungal infections are primarily found in immunocompromised hosts.

Migraine

Migraine headaches can be severe and unrelenting. At presentation, the severity of the headache may raise the clinical question of possible subarachnoid hemorrhage or acute meningitis. Also, patients with known migraine may develop atypical headaches.

Acute/Increasing Confusion and Obtundation

Obstructive Hydrocephalus

Acute obstructive hydrocephalus is caused by compression of the ventricular system to the point of obstructing the outflow of CSF. The common locations of blockage are at the foramina of Monroe, the cerebral aqueduct, and the outlets of the fourth ventricle. Possible causes include tumor, abscess, ventriculitis, and hemorrhage. Brain injury or cerebral infarction with massive vasogenic edema can also cause obstructive hydrocephalus.

Brain Stem or Basal Ganglia Hemorrhage

Most large deep hemorrhages in the brain are associated with hypertension. The criteria for hypertensive hemorrhage include a hypertensive patient, 60 years of age or older, and a basal ganglia or thalamic location of the hemorrhage. A CT or MR scan is the procedure of choice for evaluating these patients. Arteriography is necessary only if one of these criteria is missing. Hypertensive hemorrhages are often large and devastating. Since they are deep hemorrhages and near ventricular surfaces, ventricular rupture is common. One-half of hypertensive hemorrhages occur in the putamen; the thalamus in 25 %; pons and brain stem, 10 %; cerebellum, 10 %; and cerebral hemispheres, 5 %.

Brain Herniation

As with hydrocephalus, any large mass lesion or process with prominent vasogenic edema can produce brain herniation. With large frontal or parietal lesions, subfalcine herniation is common. Also, any large hemispheric lesion can result in medial migration of the temporal lobe and subsequent inferior herniation through the tentorial incisura. Subfalcine herniation can compress the ipsilateral anterior cerebral artery, leading to brain infarction, whereas temporal lobe herniation commonly compresses the contralateral posterior cerebral artery, causing an occipital infarct. Diffuse brain swelling or posterior fossa masses can result in herniation of the cerebellar tonsils and brain stem inferiorly through the foramen magnum.

Encephalitis

Encephalitis refers to a diffuse parenchymal inflammation of the brain. Acute encephalitis of the non-herpetic type presents with signs and symptoms similar to meningitis but with the added features of any combination of convulsions, delirium, altered consciousness, aphasia, hemiparesis, ataxia, ocular palsies, and facial weakness. The major causative agents are arthropod-borne arboviruses (Eastern and Western equine encephalitis, St. Louis encephalitis, California viral encephalitis). Eastern equine encephalitis is the most serious but fortunately also the least frequent of the arbovirus infections. The enteroviruses, such as Coxsackie virus and echoviruses, can produce a meningoencephalitis, but a mild aseptic meningitis is more common with these organisms. MR reveals hyperintensity on T2-weighted scans within the cortical areas of involvement, associated with subcortical edema and mass effect.

Herpes simplex is the commonest and gravest form of acute encephalitis with a 30–70 % fatality rate and an equally high morbidity rate. It is almost always caused by Type 1 virus except in neonates where Type 2 predominates. Symptoms may reflect the propensity to involve the inferomedial frontal and temporal lobes – hallucinations, seizures, personality changes, and aphasia. MR demonstrates positive findings in viral encephalitis as soon as 2 days after symptoms, more quickly and definitively than CT. Early involvement of the limbic system and temporal lobes is characteristic of herpes simplex encephalitis. The cortical abnor-

malities are first noted as ill-defined areas of high signal on T2-weighted scans, usually beginning unilaterally but progressing to become bilateral. Edema, mass effect, and gyral enhancement may also be present [7].

Meningitis

As describe above, in addition to severe headache, patients with acute meningitis commonly present with fever, seizures, altered consciousness, and neck stiffness. Most of these cases are bacterial in origin, but tuberculosis and fungal infections can also present acutely (Fig. 3).

Metabolic/Toxic Disorders

Whenever a patient presents to the emergency department, the possibility of ingestion of drugs or other toxic substances must be considered. The narcotics and sedatives generally produce respiratory depression, which can lead to global cerebral hypoxia. Some toxic agents specifically target the basal ganglia or the white matter. In diabetic patients, the possibility of an insulin overdose and hypoglycemia must be considered. Cocaine and methamphetamine also cause vasospasm, so these patients may present with an acute focal neurological deficit.

Acute/Progressive Visual Deficit

Monocular Deficit

Monocular visual loss can be caused by anything anterior to the optic chiasm that blocks light from the retina or compresses the optic nerve. Ocular diseases, such as retinal detachment and ocular hemorrhage, are generally first evaluated by direct visualization with fundoscopy or by ultrasound. A mass compressing the optic nerve or causing severe proptosis can cause a visual deficit. Severe proptosis and stretching of the optic nerve can compromise the arterial supply to the nerve. Finally, intrinsic optic nerve lesions, such as tumors, ischemia, and inflammation, are other causes of visual loss. Intraorbital diseases are evaluated equally well by CT or MRI (Fig. 4). For intracranial disease MRI is the imaging procedure of choice.

Bitemporal Hemianopsia

This visual deficit is caused by chiasmatic compression, usually by a mass in the suprasellar cisterns. Differential diagnosis includes all tumors and inflammatory conditions that can occur in the suprasellar region.

Homonymous Hemianopsia

The most common cause of a homonymous hemianopsia is ischemia in the distribution of the posterior cerebral artery that supplies the calcarine cortex of the occipital lobe. Also, mass lesions can compress the geniculate ganglion or the optic radiations in the temporal-occipital region.

Acute/Progressive Myelopathy

Epidural Hemorrhage

Most epidural hemorrhages are post-traumatic or postoperative. Also, patients who are anticoagulated are at greater risk for epidural hemorrhage. The introduction or presence of an epidural catheter also increases the risk of both hemorrhage and infection.

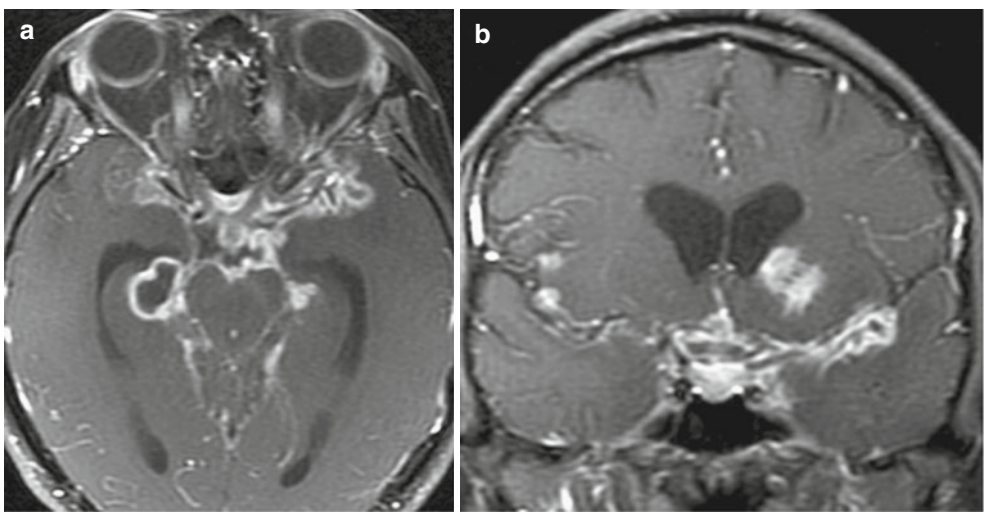

Fig. 3 Tuberculous meningitis. (**a**, **b**) Axial and coronal Gd-enhanced T1-weighted images show diffuse meningeal enhancement in the suprasellar, sylvian, and peri-mesencephalic cisterns. An enhancing mass is also present in the left basal ganglia

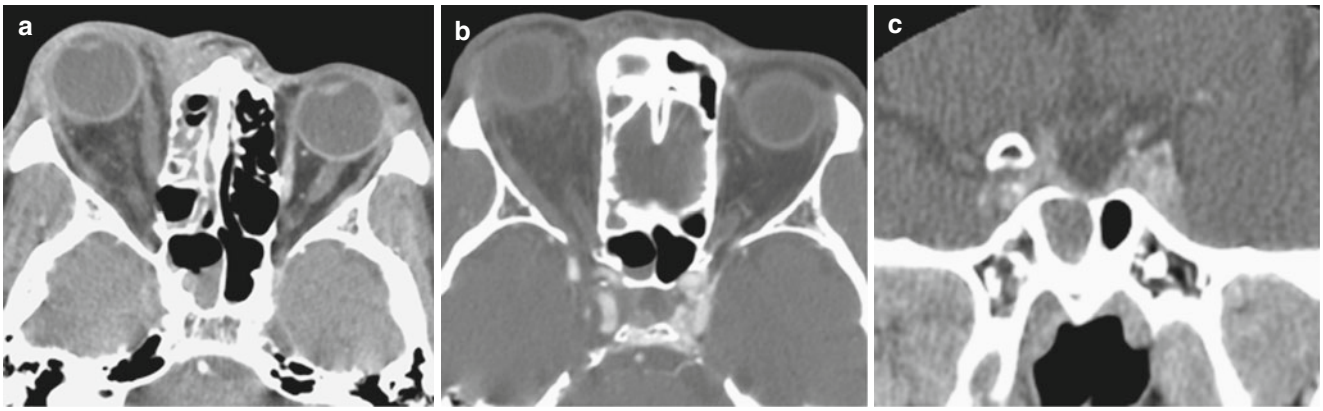

Fig. 4 Aspergillosis with cavernous sinus thrombosis. Contrast-enhanced CT scans. (**a**) The right ethmoid and sphenoid air cells are partially opacified. Right proptosis is present, and abnormal soft tissue has infiltrated the extraconal and intraconal compartments and the preseptal tissues. (**b**, **c**) Axial and coronal scans show no enhancement of the right cavernous sinus, due to septic thrombosis

Epidural Abscess

Most epidural abscesses are associated with diskitis or osteomyelitis; however, isolated infections of the epidural space can occur. The diagnosis of epidural abscess can be a challenge for both the clinician and radiologist. Patients may present with back pain or radicular pain. Fever and leukocytosis may be mild. Early diagnosis and prompt therapy are critical for favorable patient outcomes.

The imaging findings can be quite subtle on plain T1- and T2-weighted images. During the cellulitis stage, the first sign of infection is thickening of the epidural tissues, which is initially isointense on T1-weighted images and moderately hyperintense on T2-weighted images. When liquefaction occurs, the abscess cavity becomes hypointense and more hyperintense on T1- and T2-weighted images, respectively. Detection of the infectious process is easier on gadolinium-enhanced scans. The inflamed tissues (phlegmon) are highly vascular and enhance with gadolinium. On both the T2-weighted images and the enhanced T1-weighted images, fat suppression increases the contrast between the infectious process and normal tissues. The abscess cavity does not enhance and appears as a linear or elongated region of hypointensity surrounded by the enhancing cellulitis on sagittal images. The abscess cavity has an oval configuration on axial images [8].

Tumor

Epidural tumor usually extends from the spine, and the vast majority of spine tumors are metastases. The common primaries are lung, breast, and prostate. Occasionally, the epidural space may be directly seeded by lymphoma or leukemia (Fig. 5).

Spinal cord tumors and other intradural tumors (schwannoma and meningioma) may present with a progressive myelopathy.

Inflammatory Diseases

Several demyelinating diseases are associated with a transverse myelitis and acute myelopathy. In addition to classic multiple sclerosis, post-viral syndromes and Guillain-Barré are in the differential diagnosis. In HIV patients the two primary diseases to consider are epidural abscess and CMV polyradiculopathy.

Ischemia

Spinal cord ischemia is rare. It is usually associated with spinal and paraspinal tumors or surgical procedures on the spine and aorta that may compromise the blood supply to the cord.

Cervical or Thoracic Disk Extrusion

Disk extrusions in the cervical and thoracic spine, if sufficiently large, can compress the spinal cord and produce a myelopathy. Accompanying cord edema can exacerbate the problem. Emergent laminectomy and diskectomy may be necessary to relieve the cord compression.

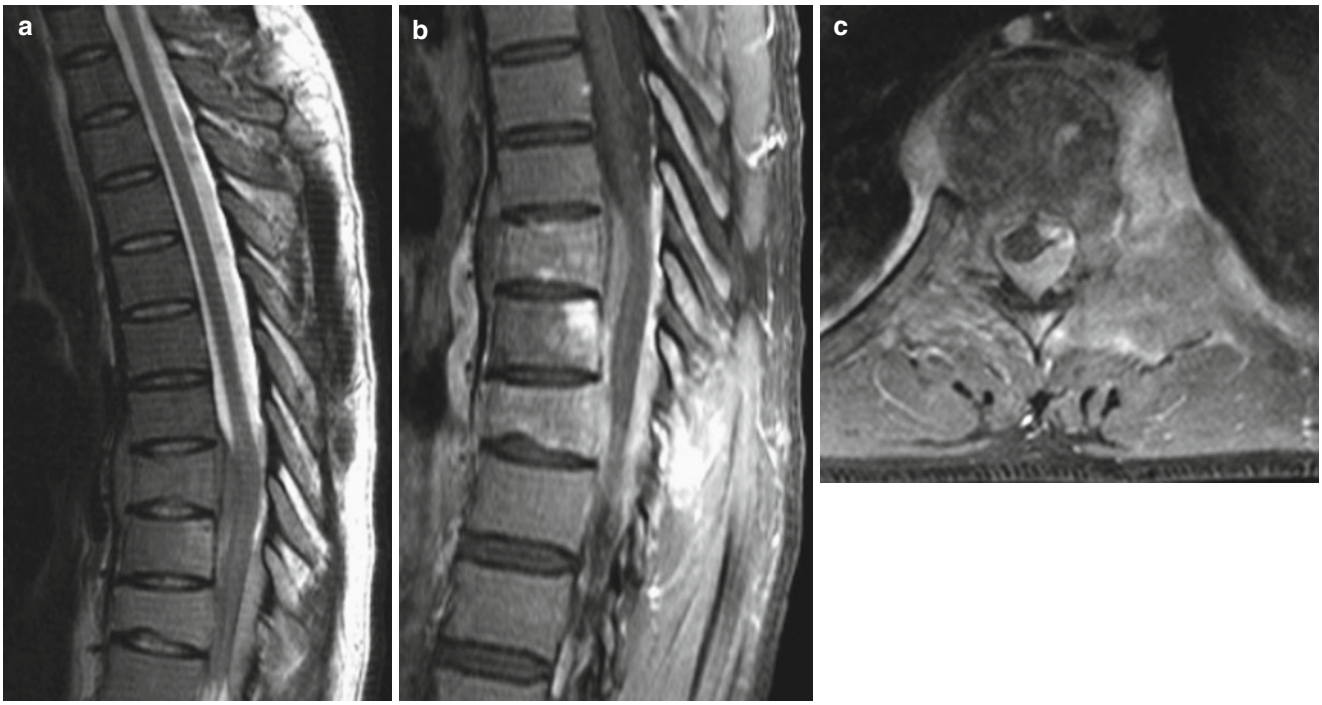

Fig. 5 Epidural spinal lymphoma. (**a**) Sagittal T2-weighted image shows epidural mass compressing the thecal sac. (**b**, **c**) Sagittal and axial Gd-enhanced T1-weighted scans reveal enhancing epidural and paraspinal masses, as well as involvement of three lower thoracic vertebrae

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