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Neurologic Emergencies on Computed Tomography of the Head

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The purpose of this article is to provide the reader, a seasoned radiologist, a budding resident, or a curious practitioner, the background necessary to accurately and swiftly interpret a head computed tomography in the emergency setting. At the very least, being able to generate a reasonable differential diagnosis is the aim and will be accomplished by describing not only features of classic neurologic emergencies but also the possible traps to which one may fall prey. Images will be used to illustrate cases, and the reader will be instructed when other imaging modalities may be required to clarify diagnoses.

Introduction

Evaluation of the brain with computed tomography (CT) has become an indispensable tool in the emergency setting, providing accurate diagnosis of patients in a timely manner. Any radiologist, radiology resident, and even certain clinicians should be able to quickly and precisely differentiate various emergent processes. Several topics will be addressed in the following pages with a goal of learning to identify key diagnostic imaging findings in the most common emergent neurologic processes so that patients can be treated quickly and appropriately. Multiple cases will be provided to illustrate key learning points, and the limitations of CT will also be discussed, as well as when further testing such as angiography or magnetic resonance (MR) may be warranted.

Brain (Infarction)

Cerebral vascular accidents (CVAs) are the second leading cause of death worldwide, and the number one cause of morbidity in the United States[1] occurring secondary to either parenchymal ischemia or hemorrhage. Overall, 85% of strokes are ischemic in etiology, with the remaining hemorrhagic in nature. Ischemia is defined as an inadequate perfusion to tissue resulting in dysfunction of those tissues. If corrected in a timely manner, ischemia can be potentially reversed. Infarction is the result of prolonged ischemia resulting in cell death and irreversible damage. Approximately two-thirds of ischemic CVAs are due to thrombotic events, with embolic etiology comprising the other one-third.[2] Less common etiologies include dissection or vasculitis. For embolic events, most are cardiac in origin, though rarer entities such as air or fat paradoxical emboli in those who have a patent foramen ovale are also possible.

Stroke symptoms comprise one of the most common reasons for ordering a CT of the brain in the emergency setting. Initial evaluation of suspected CVA should be undertaken with a noncontrast CT of the head with an initial focus in assessing for the presence of hemorrhage as this drastically alters treatment. Intracranial hemorrhage will be addressed in subsequent sections as will further imaging with MR.

Once the reader is assured that no intracranial hemorrhage is present, the focus should turn to assessing for signs of acute ischemia. First, the reader should always be provided with the proper clinical history as the presenting neurologic deficits will clue the reader into the suspected area of pathology. Depending on the onset of symptoms, the initial head CT in the setting of ischemia can have varying findings. If symptoms have been present for several hours, or there is not a last known "well" time, sufficient time has usually passed to result in areas of confluent hypodensity in the brain parenchyma related to cytotoxic edema. In the presence of acute infarction, cell death results in disruption of the adenosine triphosphate-dependent sodium-potassium pumps in the cell membrane, leading to cellular retention of sodium and water. This cellular swelling manifests on CT as parenchymal hypodensity with loss of gray-white differentiation confined to a vascular territory. Narrow windowing is imperative in assessing for early ischemia to

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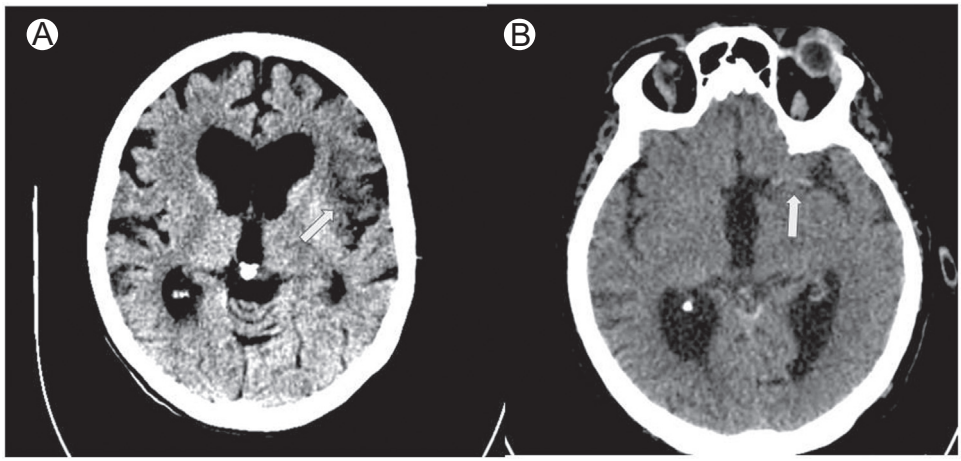

Figure 1 (A and B) A 71-year-old female patient presenting with right hemiparesis. Axial nonenhanced CT of the head demonstrates subtle hypoattenuation in the left insula with narrowed stroke windows (arrow in A). A hyperdense left middle cerebral artery (arrow in B) consistent with hyperacute thrombus is also demonstrated.

accentuate gray-white matter differentiation (Fig. 1A). Cytotoxic edema must be differentiated from another primary cause of white matter hypodensity—vasogenic edema. Vasogenic edema is seen in entities such as tumors, abscesses, and parenchymal hemorrhage, in which the blood-brain barrier is disrupted allowing for extracellular water accumulation, which primarily affects the white matter. Unlike cytotoxic edema, vasogenic edema spares the cortex and manifests as hypodensity with finger-like projections extending along white matter tracts.

At our institution, all head CTs are acquired with thin axial 1 mm slices. Despite being slightly grainy, these images are greatly helpful in assessing the hyperacute finding of a "hyperdense vessel" sign. The hyperdense vessel sign is the presence of a high density vessel that represents an area of acute thrombus (Fig. 1B). This may be the only sign of an acute event if there has not been sufficient time for even subtle hypodensity to occur in the vascular distribution of the dense vessel, although in this setting the patient should have clear clinical neurologic deficits. One of the most common vessels involved is the middle cerebral artery (MCA). On every head CT, attention should be paid to the suprasellar cistern where the carotid terminus bifurcates into the anterior and MCAs. The most common mimic of a hyperdense vessel is calcific atherosclerosis. Atherosclerotic calcification should typically be peripherally oriented, though this can be difficult to determine in heavily calcified or small caliber vessels. In this case, having prior comparison studies can be helpful. One of the even more subtle signs is the "hyperdense dot" sign, which represents thrombus in a more distal cerebral artery (Fig. 2). This is typically seen in a distal MCA branch in the sylvian fissure, and should always be sought. Depending on the timing of symptoms, patients will often have a subsequent CT angiogram (CTA) as soon as the lack of intracranial hemorrhage is confirmed, discussed separately in the CTA head and neck section of the journal.

The decision whether to administer systemic thrombolytics in the setting of acute ischemia is primarily based on the presence of a new onset of focal neurologic deficit and lack of intracranial hemorrhage. However, when the diagnosis of acute infarction is made on CT, the volume of ischemic tissue should still be estimated as this can alter the treatment. Studies have shown that when less than one-third of the MCA vascular territory is involved, there is a lower risk of hemorrhagic conversion after thrombolytic therapy is administered.[3] Conversely, if greater than one-third of the MCA vascular distribution is involved, not only is there a higher rate of hemorrhagic transformation after thrombolytic therapy but also increased morbidity and mortality. The alteplase thrombolysis for acute noninterventional therapy in ischemic stroke (ATLANTIS) study from 1999 demonstrated that there was no additional benefit from treating focal neurologic deficits with intravenous thrombolytics that had an onset of 3-5 hours. However, subsequent studies have shown that benefit can be obtained in those treated within 6 hours of symptom onset.[4]

A less common though equally important cause of acute ischemia is venous infarction. Cerebral venous infarcts (CVIs) are the result of venous thrombus, most commonly involving

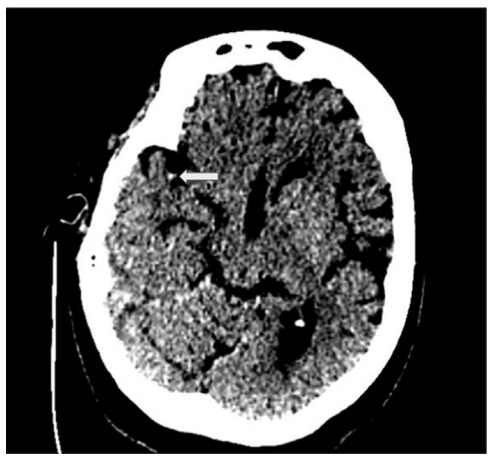

Figure 2 (A and B) An 82-year-old female patient presenting with left-sided weakness. Nonenhanced axial CT of the head shows a hyperdense focus in the right sylvian fissure representing a hyperdense right MCA dot sign (arrow).