Approach to Imaging in Patients with Spontaneous Intracranial Hemorrhage

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KEYWORDS

• Intracranial hemorrhage • Spontaneous • Hematoma • Imaging • Brain • Hypertension
• Amyloid angiopathy

KEY POINTS

- The approach to spontaneous intracranial hemorrhage (ICH) must both address emergent considerations that may prompt or guide immediate action and identify etiology of the hemorrhage.
- Understanding anatomic differences in the deep portions of the brain and the lobar brain helps predict the etiology of the hemorrhage and guide imaging selection.
- Various imaging modalities have different strengths in identifying individual etiologies of ICH. Knowledge of these strengths helps guide appropriate imaging selection.

INTRODUCTION

Spontaneous (ie, nontraumatic) intracerebral hemorrhage (ICH) is a potentially devastating neurologic event that requires emergent diagnosis and management. It is estimated that 10% to 15% of all new clinical strokes are due to ICH, with an incidence of 40,000 to 67,000 cases per year in the United States.[1,2] Mortality from ICH is high; 30-day mortality rates are 35% to 52%, with half of those deaths occurring within the first 2 days.[1] Rapid diagnosis and aggressive management are critical to reducing morbidity and mortality. Imaging plays a pivotal role in establishing the diagnosis of ICH, identifying the cause of hemorrhage, and identifying complications of acute ICH that guide management.

When a patient presents with spontaneous ICH, the role of imaging is 2-fold: first, imaging must rapidly provide information that immediately establishes the diagnosis and guides interventions intended to stabilize the patient, and, second, imaging helps determine the etiology of the hemorrhage, which can be important in both acute management and preventing rebleeding. It is, therefore, useful to have a conceptual approach to imaging that addresses both of these goals. The authors advocate the use of a primary survey intended to define factors that are expected to immediately guide the emergent stabilization and management of the patient and a secondary survey aimed at determining the etiology of the hemorrhage.[3]

This review describes anatomy, imaging techniques, and the diagnostic approach relevant to spontaneous ICH within the brain parenchyma. Additionally, it reviews the information that needs to be communicated emergently as part of the primary survey. Finally, it summarizes imaging features of the most common causes of spontaneous ICH and discusses how these features combine with clinical information to produce a patient-centered approach to the selection of imaging, as part of the secondary survey.

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NORMAL ANATOMY AND IMAGING TECHNIQUE

Anatomy

Hemorrhage tends to occur in specific and sometimes predictable locations in the brain, depending on the etiology of the hemorrhage. Understanding how various anatomic factors contribute to the risk of hemorrhage, therefore, is instructive in assigning the etiology of spontaneous ICH. The most fundamental distinction to understand in terms of cerebral anatomy when it comes to ICH is the difference between deep brain and lobar hemorrhage.

The deep brain structures are composed of the basal ganglia (including the caudate nucleus, putamen, and globus pallidus), the thalamus, pons, and dentate nuclei of the cerebellum. Hypertension, the most common cause of spontaneous brain hemorrhage, has a predilection for the deep brain structures. It is believed that this predilection is related to the particular anatomy of the blood vessels that perfuse these regions. Specifically, the deep structures are supplied by small penetrating arteries that arise directly off of medium-caliber vessels.[4] As a result of this rapid transition in caliber of the vessels, the perfusion pressure in the small perforators is higher than the pressure in the vessels that irrigate the lobar portions of the brain, which originate more distally in the arterial supply. This difference in perfusion pressure becomes even more exaggerated in the setting of hypertension.[5]

In the penetrating vessels of the deep brain structures, chronic exposure to high intra-arterial pressure results in destruction of the smooth muscle cells in the vascular wall and results in the formation of microaneurysms, termed *Charcot-Bouchard aneurysms*.[4,6] Together, these changes make the vessel fragile and more prone to rupture. The lenticulostriate perforating vessels that supply the basal ganglia are 1 example of such vessels and are 1 of the most frequent sites of hypertensive hemorrhage.

Although the basal ganglia are commonly involved in cases of hypertensive hemorrhage, it is important to realize that infratentorial locations are vulnerable to hypertensive hemorrhage as well. In particular, the pons and the dentate nuclei of the cerebellum are frequent sites of bleeding. The pons is perfused by very small penetrating arteries that arise directly off of the relatively larger basilar artery and thus are vulnerable to the same perfusional stresses exerted on the lenticulostriate perforating vessels.

The dentate nucleus is a deep gray nucleus located in the medial cerebellum notable for its convoluted, serrated margin (**Fig. 1**). As a metabolically active gray matter structure, it too has an abundant blood supply supplied by small perforating vessels, which predispose it to hypertensive hemorrhage. Because of its proximity to the fourth ventricle, hypertensive hemorrhages centered in the dentate nucleus often extend into the ventricle and can be mistaken for a primary subarachnoid or intraventricular hemorrhage (IVH) if the parenchymal component is not appreciated.

In contrast, the supratentorial white matter in the more peripheral or lobar portions of the brain is irrigated by penetrating long medullary arteries. These long medullary arteries arise further from the circle of Willis compared with the perforating vessels in the deep brain structures and, therefore, experience lower perfusion pressures.[7] They course from their origins in the pial vasculature into the deep white matter of the centrum semiovale and periventricular white matter, where they are provide end-arterial supply.[8] Exposure to chronic hypertension is associated with increasing vascular tortuosity, which increases the overall vessel length, in turn further decreasing perfusion pressures distally.[5,7,9] These factors combine to make hypertensive hemorrhage less likely in the lobar portions of the brain. Rather, hemorrhages here are more likely to be due to other entities, including amyloid angiopathy, arteriovenous malformations (AVM), tumors, and other etiologies. Generally speaking, then, imaging is more likely to reveal underlying lesions in lobar hemorrhages and less likely to identify lesions in the deep portions of the brain, where hypertensive hemorrhages predominate.

Imaging Technique

As first-line imaging, noncontrast computed tomography (NCT) is by far the most common modality used to diagnose ICH and is highly sensitive for the detection of acute blood products.[10] NCT provides very rapid imaging and is widely available. The American College of Radiology Practice Parameters recommend that brain computed tomography (CT) obtained for new focal neurologic deficit or suspected parenchymal hemorrhage include at minimum contiguous or overlapping axial slices of no greater than 5-mm thickness.[11]

MR imaging has also been shown sensitive for the detection of acute hemorrhage and has the benefit of being more sensitive for acute ischemia and chronic hemorrhage than NCT.[12,13] Nevertheless, its use as an initial, first-line imaging test is limited to a few centers because of issues of scanner availability, scan time, patient contraindications to MR imaging, and challenges associated