Diagnostic Evaluation for Nontraumatic Intracerebral Hemorrhage

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KEYWORDS

- Intracerebral hemorrhage • Deep perforating vasculopathy
- Cerebral amyloid angiopathy • Diagnostic evaluation • MRI
- Digital subtraction angiography

KEY POINTS

- The incidence of intracerebral hemorrhage (ICH) ranges from 15 to 40 per 100,000 person-years. ICH is a devastating condition, with a 30-day mortality ranging from 35% to 52% with only 20% of survivors achieving full functional recovery at 6 months.
- Several different underlying vessel diseases may result in ICH. The aim of the diagnostic evaluation is not only to confirm the ICH diagnosis but also to establish the cause of the bleeding.
- Noncontrast computed tomography is highly sensitive for the detection of clinically relevant acute brain hemorrhage but the most useful tool for the etiologic evaluation of ICH is MRI. Besides being highly sensitive, MRI provides clues to the underlying vessel disease. Other neuroimaging techniques, such as CT angiography, magnetic resonance angiography and magnetic resonance venography, and digital subtraction angiography, can also be used to assess the intracranial vessels (arteries and veins).
- Diagnostic algorithms may vary according to the suspected underlying vessel disease.

INTRODUCTION

Intracerebral hemorrhage (ICH) is defined as a focal collection of blood within the brain parenchyma that may extend to other compartments of the brain (ventricular,

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subarachnoid, or subdural spaces). It is a heterogeneous condition resulting from several distinct underlying vasculopathies. The overall incidence of ICH ranges from 15 to 40 per 100,000 person-years.[1,2] ICH accounts for 10% to 15% of all strokes, but this proportion may be higher in Asian and African populations.[2] Despite a significant improvement in the management of ischemic strokes, ICH treatment has not significantly evolved and this condition remains associated with a very high case fatality rate in the first month, ranging from 13% to 61% of patients, with a median of 40% across studies.[2] The poor prognosis of ICH may be partly caused by poor understanding of this heterogeneous condition.

The clinical and epidemiologic scenario of ICH has been changing in the last decades.[3,4] Despite an overall stable incidence of ICH, the incidence among people younger than 60 years has decreased, whereas in those older than 75 years it has increased in association with increasing premorbid use of antithrombotic drugs at this age. This trend may suggest that some bleeding-prone vasculopathies in the elderly are more likely to bleed when antithrombotic drugs are used, as illustrated by the rise in the incidence of lobar ICH in the elderly, in which cerebral amyloid angiopathy (CAA) may be strongly implicated.[4]

WHAT ARE THE CAUSES TO SEARCH FOR IN PATIENTS WITH INTRACEREBRAL HEMORRHAGE?

Importance of Intracerebral Hemorrhage Location to Tailor Diagnostic Work-Up

ICH location can be classified as deep, lobar, and infratentorial (involving the cerebellum and/or the brainstem). The anatomical distribution of the hemorrhage and its extension to other compartments (subarachnoid, subdural, intraventricular) may contribute to identify the underlying cause of the bleeding.

Deep locations represent about 45% of all ICH, whereas lobar locations account for 30% to 40%, cerebellar for approximately 10%, and brainstem for approximately 5%. These proportions are subject to bias because most estimates are based on hospital series in which referral is less often considered for moribund patients or for patients with mild deficits, and on population studies that contain such a small proportion of ICH that any subdivisions are subject to chance effect.[5,6] The pooled 1-year survival estimate in nine population-based studies was 45.4% to 59.1% after lobar ICH and 45.4% to 59.5% after deep ICH.[7]

Causes of Intracerebral Hemorrhage

The two most frequent causes of ICH are deep perforating vasculopathy and CAA.

Deep perforating vasculopathy

Deep perforating vasculopathy accounts for nearly 50% of ICH worldwide.[8] Chronic arterial hypertension is the most frequent risk factor associated with deep perforating vasculopathy.[5] Deep perforating vasculopathy results probably from a reactive hyperplasia and microscopic degenerative changes of vessel wall components leading to reduced vascular reactivity and enhanced vessel wall fragility.[9] Deep perforating vasculopathy usually occurs in lenticulostriate arteries originating from middle cerebral artery, in small thalamic arteries arising from the posterior communicating and posterior cerebral arteries, and perforating arteries that arise from the basilar artery. Given the absence of anatomic demonstration of perforating arteries in the cerebellum, the implication of deep perforating vasculopathy in cerebellar ICH remains controversial.

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