

Intracerebral Hemorrhage: A Retrospective Study in Metropolitan Population

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Abstract

Intracerebral hemorrhage, or ICH, is a devastating disease. The overall incidence of spontaneous ICH worldwide is 24.6 per 100,000 person-years with approximately 40,000 to 67,000 cases per year in the United States. The 30-day mortality rate ranges from 35% to 52% with only 20% of survivors expected to have full functional recovery at 6 months. Approximately half of this mortality occurs within the first 24 hours, highlighting the critical importance of early and effective treatment in the Emergency Department.

Introduction

Intracerebral hemorrhage (ICH) is defined as bleeding in the brain parenchyma, whereas intracranial hemorrhage refers to any bleeding within the cranial vault. While traumatic ICH is by far the most common type of ICH, this review will focus on spontaneous, nontraumatic ICH, implying bleeding that occurs without trauma or known bleeding causes such as an arteriovenous malformation, cerebral aneurysm, or tumor.

Intracerebral hemorrhage is defined by its location within the brain parenchyma, with “deep” ICH being located within the basal ganglia and internal capsule (35%-70%), brain stem (5%-10%), and cerebellum (5%-10%). In contrast, “lobar” ICH (15%-30%) refers to hemorrhages located in cortical-subcortical areas and follows a “lobar” pattern across one or less often multiple lobes of the brain. Deep ICH accounts for about two third of spontaneous ICH cases, and lobar ICH accounts for the remaining one third.

Epidemiology and Risk Factors

Intracerebral hemorrhage is twice as common as aneurysmal subarachnoid hemorrhage (SAH).The incidence of ICH is 12 to 15 cases per 100 000 individuals or about 40 000 cases per year in the United States. Incidence varies among populations.

Hypertension is by far the most common attributable risk factor; it accelerates age-related “wear and tear” of cerebral arterioles at branch points. Cerebral amyloid angiopathy (CAA), a condition that increases with age, is the second most common risk factor. Cerebral amyloid angiopathy is an important cause of lobar ICH, especially in the elderly individuals.This condition results from amyloid protein deposition in cortical arterioles; such deposition is rare in the basal ganglia and brain stem (usual locations of HTN-related ICH and unusual locations of CAA-related ICH). Apolipoprotein E (ApoE) genotype plays an important role in the pathogenesis of CAA, but it is neither sensitive nor specific for the primary diagnosis of this condition. Recurrent lobar CAA-related ICH is relatively common.

Cerebral reperfusion syndrome is a rare but serious complication of carotid revascularization, seen both after endarterectomy (CEA) and angioplasty/stenting (CAS); it is a uncommon cause of ICH. It is due to increased cerebral blood flow (compared to pre-revascularization levels) in combination with impaired cerebrovascular autoregulation. It may occur up to several weeks after revascularization but usually in the first few days.

The highest risk period is at 12 hours post CAS and at 6 days post CEA. Deterioration of consciousness, confusion, and headache are the typical presenting symptoms. Risk factors for this syndrome include long-standing elevated blood pressure, diabetes, advanced age, contralateral carotid occlusion, and recent contralateral carotid revascularization within 3 months, postoperative HTN, and use of anticoagulants. Treatment strategies are directed toward regulation of blood pressure and limitation of rises in cerebral perfusion.

Pathophysiology

Nontraumatic bleeding into the brain parenchyma results from rupture of small penetrating arteries. In deep hematomas, this has been attributed to degenerative changes in the vessel wall associated with advancing age, chronic HTN, diabetes, and other vascular risk factors. Charcot-Bouchard microaneurysms and lipohyalinosis of small arterioles have been suggested as mechanisms. In some circumstances, degenerative changes of these arterioles may be associated with lacunar stroke and in other cases they may lead to ICH.Cause and effect are not always clear-cut because ischemic and hemorrhagic stroke are both age- and HTN-related. Rigorous pathological studies in the era of modern imaging with computerized tomography (CT) and magnetic resonance imaging (MRI) are lacking. When ICH does occur, most bleeding occurs at or near the bifurcation of affected arterioles.

Treatment

Hemostatic Therapy

The presence of coagulopathy (congenital or acquired) worsens the prognosis of ICH by increasing the rate of hematoma expansion and the duration of that expansion. Reversal of coagulopathy is of the essence, and specific antidotes should be used depending on the clinical scenario. Anticoagulant agents are discontinued immediately. In the case of warfarin, vitamin K, 10 mg, is administered slowly, intravenously, followed by either fresh frozen plasma (FFP) along with prothrombin complex concentrates (PCCs) or in some selected cases recombinant factor VIIa. The PCC dosage is calculated according to body weight, degree of INR prolongation, and desired level of correction; typical dosages are 25 to 50 IU/kg.

Blood Pressure

The management of acute HTN in the setting of ICH is a medical emergency.



However, the exact therapeutic blood pressure target remains one of the considerable controversies in the context of incomplete evidence. Two contradicting theories, yet unproven, illustrate this uncertainty. The first theory is that there is a region of perihematomal “penumbra” or brain tissue at risk that can become ischemic if the blood pressure is reduced precipitously. The second theory is that acute HTN actually causes worsening hematoma growth.

Anticonvulsant Therapy

Early seizures occur in about 4% of ICH cases and in about 8% within 30 days. Lobar hematomas carry a higher risk of seizures than deep ICH. Clinical seizures in the setting of ICH should be treated with appropriate antiepileptic drugs. Initial medication choices include benzodiazepines, followed by fosphenytoin or phenytoin.

Deep Vein Thrombosis Prophylaxis

Intermittent pneumatic sequential compression devices (SCDs) are indicated for patients with ICH for deep vein thrombosis (DVT) prophylaxis, assuming there is no history of DVT or leg fracture to contraindicate use. Once the intracranial bleeding has stopped and after 3 to 4 days from onset of the ICH, low-dose molecular weight heparin or unfractionated heparin may be used for DVT prevention in patients with hemiplegia.

Conclusion

Intracerebral hemorrhage diagnosis and treatment have evolved over the past decade, in the setting of increasing knowledge about risk factors, pathophysiology, and management. Neuroimaging has advanced the field. Microbleeds detected by MRI may help predict underlying pathophysiology and help determine prognosis. Wider appreciation of hematoma growth and the utility of contrast-enhanced CT “spot sign” have increased the value of CT. Primary management of ICH involves rapid clinical evaluation, correction of any coagulation defects, admission to an ICU setting, and careful control of blood pressure. More specific blood pressure targets are being evaluated within the ATACH II and INTERACT 2 trials with the hopes of further reducing morbidity and mortality. The role of surgical hematoma evacuation is uncertain and likely not beneficial for most of the patients with ICH. Studies addressing minimally invasive surgical management for ICH and IVH are underway. The studies may answer whether these techniques offer more than medical management for patients with ICH. Further trials are needed to determine the best measures to halt early ICH growth, minimize cerebral edema, and attenuate the toxic effects of blood products.

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