Spontaneous Intracerebral Hemorrhage Risk Factors

Hypertension, obesity, smoking, and cerebral small vessel disease were important factors associated with non-lesional spontaneous intracerebral hemorrhage in young patients. Radiologic changes corresponding to cerebral small vessel disease appeared in young patients (in their 30s) and they were associated with hypertension [1)].

Psychosocial, ethnic, and economic factors play a role in the prevalence of Spontaneous Intracerebral Hemorrhage, with ICH being twice as common in low-income and middle-income countries compared with high-income countries. Other identified risk factors for ICH include age (i.e., each decade from 50 years of age is associated with a 2-fold increase in the incidence of ICH) and an elevated alcohol intake.

Etiologies of ICH to always consider include intracranial aneurysms (typically presenting as subarachnoid hemorrhage); arteriovenous malformations (ICH is the first presentation of AVMs in 60 % of cases); cerebral venous sinus thrombosis and venous infarction; brain tumors (<5 % of all ICH cases) including cerebral metastases (e.g., lung cancer, melanoma, renal cell carcinoma, thyroid carcinoma, and choriocarcinoma) and primary CNS tumors (e.g., glioblastoma multiforme and oligodendrogliomas); and drugs of abuse (e.g., cocaine, amphetamines). Because of the differing etiologies of ICH, a rapid and accurate diagnosis of the underlying etiology of ICH is essential to direct appropriate management strategies.

Hypertensive intracerebral hemorrhage

The most important modifiable risk factor in spontaneous ICH is chronic arterial hypertension:

see Hypertensive intracerebral hemorrhage.

Amyloid angiopathy

Besides hypertension, cerebrovascular amyloid deposition (i.e., cerebral amyloid angiopathy) is associated with ICH in older patients.

Although cerebral amyloid angiopathy (CAA), which is Aβ deposition in the cerebral vessels, related cerebral hemorrhage rarely develops in young people, several patients with CAA-related cerebral hemorrhage under the age of 55 with histories of neurosurgeries with and without dura mater graft in early childhood have been reported. These patients might show that Aβ pathology is often recognized as Aβ-CAA rather than parenchymal Aβ deposition in the transmission of cerebral β-amyloidosis in humans, and Hamaguchi et al. proposed an emerging concept, “acquired CAA”. Considering that there have been several patients with acquired CAA with an incubation period from neurosurgery and

the onset of CAA-related cerebral hemorrhage of longer than 40 years, the number of cases is likely to increase in the future, and detailed epidemiological investigation is required. It is necessary to continue to elucidate the pathomechanisms of acquired CAA and urgently establish a method for preventing the transmission of cerebral β-amyloidosis among individuals [2)].

Intracranial vascular malformation

It is a common initial symptom of intracranial vascular malformations.

see Intracerebral hemorrhage from ruptured cerebral arteriovenous malformation.

see Aneurysmal intracerebral hemorrhage.

see Cerebral venous sinus thrombosis and venous infarction.

Neoplasm

see Spontaneous intracranial hematoma caused by neoplasm.

Vasculitis

Vasculitis.

AIDS

Complication of AIDS.

NPH

Shunting for NPH

Coagulopathy

Coagulopathies (i.e., the use of antithrombotic or thrombolytic agents, congenital or acquired factor deficiencies) and systemic diseases, such as thrombocytopenia, are possible causes of ICH. The use of oral anticoagulants, especially vitamin K inhibitors (i.e., warfarin), has increased coagulopathy-associated ICH in recent years, accounting for more than 15 % of all cases.

Severe thrombocytopenia can result in petechial hemorrhages or spontaneous intracerebral

hemorrhage (ICH).

Anticoagulant Related Intracerebral Hemorrhage

see Anticoagulant Related Intracerebral Hemorrhage.

Posterior fossa surgery

Remote supratentorial hematoma soon after posterior fossa surgery for the removal of a space-occupying lesion is a rare but dramatic and dreaded complication, carrying significant morbidity and mortality [3) 4) 5) 6) 7) 8) 9) 10)].

Posterior reversible encephalopathy syndrome

see Posterior reversible encephalopathy syndrome.

Ethanol

Ethanol

Cocaine

Cocaine

Inadvertent intrathecal injection of unapproved contrast agents

Inadvertent intrathecal injection of unapproved contrast agents.

Vitamin D

The evidence linking vitamin D (VitD) levels and Spontaneous Intracerebral Hemorrhage Risk Factors remains inconclusive. Szejko et al. tested the hypothesis that lower genetically determined VitD levels are associated with a higher risk of ICH. They conducted a 2-sample Mendelian Randomization (MR) study using publicly available summary statistics from published genome-wide association study of VitD levels (417 580 study participants) and ICH (1545 ICH cases and 1481 matched controls). They used the inverse variance-weighted average method to generate causal estimates and the MR

Pleiotropy Residual Sum and Outlier and MR-Egger approaches to assess for horizontal pleiotropy. To account for known differences in their underlying mechanism, we implemented stratified analysis based on the location of the hemorrhage within the brain (lobar or nonlobar). Our primary analysis indicated that each SD decrease in genetically instrumented VitD levels was associated with a 60% increased risk of ICH (odds ratio [OR], 1.60; [95% CI, 1.05-2.43]; P=0.029). They found no evidence of horizontal pleiotropy (MR-Egger intercept and MR Pleiotropy Residual Sum and Outlier global test with P>0.05). Stratified analyses indicated that the association was stronger for nonlobar ICH (OR, 1.87; [95% CI, 1.18-2.97]; P=0.007) compared with lobar ICH (OR, 1.43; [95% CI, 0.86-2.38]; P=0.17). Lower levels of genetically proxied VitD levels are associated with higher ICH risk. These results provide evidence for a causal role of VitD metabolism in ICH [11].

COVID-19 and Intracerebral Hemorrhage

COVID-19 and Intracerebral Hemorrhage

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