Hypertensive intracerebral hemorrhage

- Knowledge, attitudes, and practices regarding the prevention of intracerebral hemorrhage among hypertensive patients
- Long-Term Outcomes in Patients With Hemorrhagic Moyamoya Disease Combined With Hypertension After Encephaloduroarteriosynangiosis
- Efficacy of Concurrent Utilization of Mannitol and Nimodipine in Treating Hypertensive Intracerebral Hemorrhage and Its Effects on Neurological Function
- Knowledge Graph-Enhanced Deep Learning Model (H-SYSTEM) for Hypertensive Intracerebral Hemorrhage: Model Development and Validation
- An unexpected turn: Posterior reversible encephalopathy syndrome following microsurgical resection of a brain arteriovenous malformation
- Peripheral blood immune landscape and NXPE3 as a novel biomarker for hypertensive intracerebral hemorrhage risk prediction and targeted therapy
- Questionnaire Survey of Facilities Conducted among Members of the Japanese Society for Stroke Surgery on Surgical Intervention for Hypertensive Intracerebral Hemorrhage
- Progress to Date on Cranial Electromagnetic Field Stimulation to Modulate Brain Activity

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)].

Elevated blood pressure (BP), which presents in approximately 80 % of patients with acute intracerebral hemorrhage (ICH), is associated with an increased risk of poor outcomes.

Clinical features

Patients will present depending on the region and size of the hemorrhage:

Basal ganglia hemorrhage clinical features.

Thalamic hemorrhage clinical features.

Pontine hemorrhage clinical features

Cerebellar hemorrhage clinical features

Pathology

The pathology of hypertensive ICH involves several steps:

Hypertension: The sustained high blood pressure causes damage to small blood vessels in the brain, leading to weakening and thickening of their walls. This makes them more susceptible to rupture.

Rupture: When the blood vessel ruptures, blood leaks into the surrounding brain tissue, causing inflammation and damage to brain cells.

Hematoma formation: The leaked blood accumulates and forms a hematoma, which can put pressure on surrounding brain tissue, causing further damage.

Edema: The inflammation caused by the presence of blood in the brain tissue can lead to swelling, called edema, which can worsen the pressure on the brain tissue.

Secondary injury: The pressure and inflammation caused by the hematoma and edema can lead to secondary injury to the brain tissue, which can cause further neurological deficits.

Microaneurysms of perforating arteries (Charcot-Bouchard aneurysms)

small (0.3-0.9 mm) diameter

occur on small (0.1-0.3 mm) diameter arteries

distribution matches the incidence of hypertensive hemorrhages

80% lenticulostriate

10% pons

10% cerebellum

found in hypertensive patients

may thrombose, leak (cerebral microhemorrhages), or rupture

accelerated atherosclerosis: affects larger vessels

hyaline arteriosclerosis

hyperplastic arteriosclerosis: seen in very elevated and protracted cases

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Lenticulostriate artery are particularly susceptible to damage from hypertension. They may either rupture, producing an intracerebral hemorrhage that is initially centered in the region they supply, or become occluded producing a lacunar infarct in the tissue they supply.

Guidelines

Chinese multidisciplinary guideline for the management of hypertensive intracerebral hemorrhage [2)].

Treatment

Hypertensive intracerebral hemorrhage treatment

Outcome

Characteristics of hypertensive hemorrhages that lead to poorer prognosis include :

bleed in the posterior fossa

a large amount of mass effect

extension into the ventricular system —-

Optimal recovery from intracerebral hemorrhage was observed in hypertensive patients who achieved the greatest SBP reductions (≥20 mm Hg) in the first hour and maintained for 7 days [3].

Hematomas within the basal ganglia comprise 60% of all cases with hypertensive intracerebral hemorrhage, and these cases have a particularly high morbidity and mortality despite optimized treatments [4].

Dennis MS et al. have reported that the one-year survival rate of patients with these hematomas is only 38% [5] and that most survivors are disabled [6].

see putaminal hemorrhage

Prospective cohort studies

China is one of the countries with the highest burden of hypertensive intracerebral hemorrhage (HICH), and its morbidity and mortality rates are almost twice the world average. Most survivors experience negative emotions such as anxiety and depression due to symptoms such as speech disorders, dysphagia, cognitive impairment, hemiplegia and ataxia. While evidence has emerged, supporting the acceptance of disability is a major factor in psychosocial adjustment of patients with disabilities. However, most relevant studies mainly focus on cross-sectional design, and the impact of disability on physical and mental health is a complex and comprehensive process, and its mechanism is still unclear. Therefore, Zhang et al. aimed to use the latent growth mixture model (LGMM) and the decision tree model to analyse the trajectory and predictors of disability acceptance in patients with HICH from stable hospitalisation to 2 years after discharge.

The objective of this prospective study will be to examine the 2-year trajectory of disability acceptance in a cohort of persons with HICH. 180 participants will be recruited, and baseline general data collection, disability acceptance, family caring index and self-efficacy of chronic disease will be conducted. All of them will be followed up at the stable hospitalisation period, 6 month, 1 year and 2 year after discharge using the same protocol. As a major result, disability acceptance trajectories and potential categories will be analysed using LGMM. Additionally, the independent variables with statistical significance will be included in the decision tree model, and the Classification And Regression Trees（CART） algorithm programme will be used to construct the prediction model of influencing factors of disability acceptance trajectory. The exploratory outcome will provide scientific basis for the optimal intervention time point and the formulation of rehabilitation measures for this population [7].

Case series

Clinical data of 184 patients with HICH in the hospital from January 2019 to May 2021 were analyzed retrospectively. The patients were divided into mini-open craniotomy group and neuroendoscopic-assisted group. The operation time, hematoma clearance rate, intraoperative blood loss, neurological function recovery, and postoperative mortality of the two groups were compared by retrospective analysis.

Results: The operation time and intraoperative blood loss in the mini-open craniotomy group were more than those in the neuroendoscopic-assisted group, but there was no significant difference between the two groups. There was no significant difference in hematoma clearance rate between the two groups, but for the rugby hematoma, the hematoma clearance rate in the neuroendoscopic-assisted group was higher than in the mini-open craniotomy group, the difference was statistically significant. Within 1 month after the operation, there was no significant difference in mortality between the two groups. 6 months after the operation, there was no significant difference in the recovery of neurological function between the two groups.

Conclusion: Neuroendoscopic-assisted and mini-open craniotomy for the treatment of HICH has the advantages of minimal trauma with good effects, and its main reason for short operation time, reduced bleeding, and high hematoma clearance rate. Although the two surgical methods can improve the survival rate of patients, they do not change the prognosis of patients. Therefore, the choice of surgical methods should be adopted based on the patient's clinical manifestations, hematoma volume, hematoma type, and the experience of the surgeon [8].

From February 2013 to November 2018, 60 patients diagnosed as basal ganglia ICH were divided into the filled type hematoma expansion group (FTE group) and the expanded type hematoma expansion group (ETE group). we performed follow-up CT and three-dimensional reconstruction for the patients and compared the hematoma before and after the expansion of size and extent.

The regression analysis showed that the irregular sign (odds ratio, 3.64; 95 % CI, 1.46-9.12), black hole sign (odds ratio, 3.85; 95 % CI, 1.40-10.60), blend sign (odds ratio, 2.86; 95 % CI, 1.03-7.95), and early use of dehydration (odds ratio, 4.59; 95 % CI, 1.59-13.19) were possible risk factors for the ETE group, while the high systolic blood pressure (odds ratio, 1.51; 95 % CI, 1.04-2.30), early use of dehydration (odds ratio, 3.27; 95 % CI, 1.10-9.69) and low density low-density band (odds ratio, 4.52; 95 % CI, 1.54-13.28) were possible risk factors for the FTE group.

The irregular sign, black hole sign, blend sign and early use of dehydration may be the main risk factors for hematoma expansion group, whereas early use of dehydration, high systolic blood pressure, and low-density band may be the main risk factors for hematoma expansion [9].

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96 HIH patients were performed the craniotomic hematoma dissection (CHD) and the hematoma-cavity drilling drainage (HCDD), respectively. Meanwhile, the intracranial pressure and mean arterial pressure of each patient were continuously monitored for 7 days, the postoperative 1st, 3rd, 7th and 14th-day average flow velocities and pulsatility indexes of the bilateral middle cerebral arteries were monitored. CHD exhibited the significant difference in the long-term quality of life (ADL classification 6

months later) of patients with hematoma >50 ml than HCDD; furthermore, the postoperative 1st, 3rd, 7th and 14th-day TCD parameter analysis revealed that CHD exhibited better results in relieving the intracranial pressure and improving the cerebral blood flow than HCDD, and the postoperative ICP and MAP monitoring towards all patients could effectively control the blood pressure and prevent the further bleeding. The patients with hematoma >50 ml should choose CHD, and all HIH patients should be routinely performed the ICP and MAP monitoring [10].

Case reports

A 55-year-old Japanese woman with a history of hypertension and right putaminal hemorrhage developed simultaneous hemorrhages in the left thalamus and putamen and died 24 hours later. There were no vascular anomalies in the brain. Synaptophysin immunostaining and eosin azure 50 (EA50) staining identified the hematoma and the surrounding brain structures. In the right cerebral hemisphere, a cystic lesion as a sequela of the usual type of hypertensive putaminal hematoma was evident. In the left cerebral hemisphere, two fresh hematomas were evident. One was a thalamic hematoma, which had destroyed the dorsal and medial structures of the thalamus, and the other was an unusual putaminal hematoma, which had destroyed the entire putamen and crossed the internal capsule and caudate nucleus. α-Smooth muscle actin immunostaining combined with EA50 and Victoria bleu staining demonstrated three ruptured arteries associated with fibrin aggregates in the anterior thalamic nucleus and anterior putamen. Some circular structures composed of fibrin, suggesting the presence of ruptured arteries in the neighborhood, were evident in the thalamus and putamen. In the putamen, ruptured arteries and circular structures were present in the lateral to medial areas. Fibrin aggregates in the anterior thalamic nucleus were more numerous than those in the putamen. Based on these findings, we concluded that: (i) the artery with numerous fibrin aggregates in the anterior thalamic nucleus had ruptured first, followed by the arteries distributed in other parts of the thalamus and putamen; (ii) the unusual putaminal hematoma was attributable to rupture of the arteries around the center of the putamen, which are not responsible for the usual type of hypertensive putaminal hematoma; and (iii) it is suggested that even if hypertensive hemorrhage occurs simultaneously in the ipsilateral putamen and thalamus, the usual type of hypertensive mixed-type hematoma does not form [11].

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