Basal ganglia hemorrhage

- Left Spontaneous Basal Ganglia Hemorrhage With Transcommissural Extension Via the Canal of Gratiolet and Left-Sided Dystonia: A Case Report
- The causal relationship between enlarged perivascular spaces and intracerebral hemorrhage: A 2-sample Mendelian randomization study
- Moyamoya-related intracerebral hemorrhage associated with hyperemesis gravidarum
- Cavernoma of the left ventral striatum, anatomical and microsurgical implications of the ipsilateral transcallosal transrostral pathway
- An unexpected turn: Posterior reversible encephalopathy syndrome following microsurgical resection of a brain arteriovenous malformation
- miR-210 Regulates Autophagy Through the AMPK/mTOR Signaling Pathway, Reduces Neuronal Cell Death and Inflammatory Responses, and Enhances Functional Recovery Following Cerebral Hemorrhage in Mice
- Ring hemorrhages in the central nervous system of severely anemic cynomolgus monkeys
- Automated Computer Vision Methods for Image Segmentation, Stereotactic Localization, and Functional Outcome Prediction of Basal Ganglia Hemorrhages

Epidemiology

Basal ganglia hemorrhage is a relatively common type of stroke, accounting for approximately 10-15% of all strokes. The incidence of BGH increases with age, and the majority of cases occur in people over the age of 60. Men are more likely than women to experience BGH. Certain medical conditions, such as hypertension (high blood pressure) and diabetes, increase the risk of BGH. Lifestyle factors such as smoking and excessive alcohol consumption have also been associated with an increased risk of BGH. There may be some differences in the incidence of BGH between different ethnic and racial groups, but the data on this is somewhat limited. It's worth noting that these epidemiological factors can vary somewhat depending on the specific population being studied and the methodology used to collect and analyze the data.

Classification

Basal ganglia hemorrhage classification.

Mechanism

Rzepliński et al. injected 40 anatomic specimens of the basal ganglia with a contrast medium, scanned them with a micro-computed tomography scanner, and analyzed the results of radiological studies, and direct and histological examinations.

In 9 cases, micro-computed tomography and histological examinations revealed contrast medium extravasations mimicking intracerebral hematomas. The artificial hematomas spread both proximally and distally along the ruptured perforator and its branches in the perivascular spaces and detached the branches from the adjacent neural tissue leading to the destruction of the tissue and secondary

extravasations. Moreover, some contrast extravasations skipped to the perivascular spaces of unruptured perforators, created further extravasation sites, and aggravated the expansion of the artificial hematoma. There was no subarachnoid extension of any artificial hematoma.

They postulate that forming basal ganglia intracerebral hematoma spread initially in the perivascular space, detaches the branches from the neural tissue, and causes secondary bleeding. It can also skip to the perivascular space of a nearby perforator. The proposed mechanism of hematoma initiation and formation explains the extent of damage to the neural tissue, variability of growth in time and space, creation of secondary bleeding sites, and limited usefulness of surgical interventions. The model is reproducible, the extent of the artificial hematoma can be easily controlled, the rupture sites of the perforating arteries can be determined, and the preparation of the model does not require specialized, expensive equipment apart from the micro-computed tomography scanner [1)].

Scales

ICH score

Clinical features

Basal ganglia hemorrhage clinical features

Diagnosis

Basal ganglia hemorrhage diagnosis.

Treatment

see Basal ganglia hemorrhage treatment.

Case series

see Basal ganglia hemorrhage case series.

Case report from the HGUA

Q12488

Title: Hypertensive Intracerebral Hemorrhage with Complicated Course: A Clinical and Radiological Evolution

Abstract: We report the case of a 51-year-old woman presenting with sudden neurological deterioration due to hypertensive intraparenchymal hemorrhage (ICH) involving the left basal ganglia. Her clinical course was complicated by seizure, hydrocephalus, and progressive mass effect. This case highlights the critical role of timely neuroimaging, intracranial pressure (ICP) monitoring, and surgical interventions in managing severe hypertensive ICH.

Introduction Spontaneous intracerebral hemorrhage, a life-threatening neurological emergency, accounts for approximately 10–15% of all strokes. Poorly controlled hypertension remains a significant modifiable risk factor. We describe a case with a complex presentation and evolution requiring multidisciplinary management.

Case Presentation Patient History Age/Sex: 51-year-old female Comorbidities: Poorly controlled hypertension Surgical History: None Allergies: None known Substance Use: Non-smoker, occasional alcohol Current Medications: Not on antihypertensive therapy Clinical Presentation The patient experienced an abrupt onset of neurological deficits, including language impairment and right hemiparesis, prompting activation of the stroke code and urgent hospital transfer.

Initial Assessment Vital Signs: BP 207/131 mmHg Neurological Examination: Glasgow Coma Scale (GCS): 14 Global aphasia, right hemiparesis, somnolence NIH Stroke Scale (NIHSS): 22 Imaging and Diagnosis Initial CT Scan:

Left basal ganglia intraparenchymal hemorrhage (5.5 x 2.2 cm) Vasogenic edema with midline shift (4 mm) Partial collapse of perimesencephalic cisterns Hospital Course Immediate Progression Neurological decline: During transfer to ICU, the patient experienced a generalized tonic-clonic seizure treated with IV midazolam (4 mg). Airway management: Intubation was performed without complications. Repeat CT Scan:

Hematoma expansion (7.0 x 2.6 cm) Increased edema and midline shift (7 mm) Hydrocephalus with partial collapse of perimesencephalic cisterns Decision: Placement of an external ventricular drain (EVD) and ICP monitoring Subsequent Imaging: Stable hematoma (7.0 x 2.6 cm) Persistent midline shift (7 mm), hydrocephalus, and mass effect No additional hemorrhagic changes Current Status Consciousness: Sedated, GCS pre-sedation: 8 (O: 2, M: 4, V: 2) Motor findings: Right hemiparesis: plegia in the upper limb, weakness in the lower limb Left extremities: no deficits Other findings: Persistent global aphasia, cranial nerve deficits Management and Interventions Neurocritical Care: Strict ICP monitoring; target ICP < 20 mmHg Surgical Intervention: External ventricular drain placement for hydrocephalus Seizure Prophylaxis: Levetiracetam Supportive Care: Sedation and close hemodynamic control Discussion Hypertensive ICH involving the basal ganglia is associated with high morbidity due to critical location and potential for mass effect, hydrocephalus, and herniation. This case underscores the importance of aggressive ICP management, neuroimaging, and timely surgical interventions to optimize outcomes. Despite hematoma stability, persistent neurological deficits reflect the extent of damage and the need for multidisciplinary rehabilitation.

Conclusion This case highlights the complexities of managing hypertensive ICH, emphasizing the importance of timely diagnosis, intervention, and ongoing monitoring. Further studies are warranted to improve prognostication and therapeutic strategies for similar cases.

References Hemphill JC, Greenberg SM, Anderson CS, et al. Guidelines for the Management of Spontaneous Intracerebral Hemorrhage. Stroke. Broderick JP, Adams HP Jr, Barsan W, et al. Guidelines for the Management of Spontaneous Intracerebral Hemorrhage: Update 2021.

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