Spontaneous Intracerebral Hematoma

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Abstract: An intracerebral hematoma (ICH) is a stroke which constitutes about 10–15% of all strokes. An ICH is considered an acute deadly event. Patients are characterized by the peak age between 55 and 75 years and male predominance. In 50% of cases, an ICH occurs in deep grey and white matter such as basal ganglia, thalamus and internal capsule; 35% are lobar/hemispheric, and occur due to a rupture of the Charcot–Bouchard micro-aneurysm. Even if the presenting symptoms appear mild at onset, a hematoma progression may lead to rapid neurological deterioration. Here, the etiopathological aspects of an ICH, clinical presentation and progression, imaging and interpretation, as well as principles of management with special attention to surgical management are discussed.

Abbreviations

AVM	arteriovenous malformation	BP	blood pressure
CBF	cerebral blood flow	CNS	central nervous system
CSF	cerebrospinal fluid	CT	computed tomography
EV	external ventricular drain	FFP	fresh frozen plasma
HTN	Hypertension	MRI	magnetic resonance imaging
MRA	magnetic resonance angiogram	MRV	magnetic resonance venogram
ICH	intracerebral hematoma	ICP	intracranial pressure
INR	international normalized ratio	IVH	intraventricular hemorrhage
OTC	over the counter	tPA	tissue plasminogen activator
VTE	venous thromboembolism		

1. Introduction

An intracerebral hematoma (ICH) is another variety of stroke which constitutes about 10–15% of all strokes. An ICH is considered an acute life-threatening event. It generally affects a younger age group rather than older patients, with the peak age of patients between 55 and 75 years. It is characterized by a male predominance. In 50% of cases, an ICH occurs in deep grey and white matter such as basal ganglia, thalamus and internal capsule; 35% are lobar/hemispheric, with 10% being cerebellar and 5% in the brainstem location (Figure 1). The rupture of the Charcot–Bouchard micro-aneurysm developed in small vessels (lenticulostriate, thalamostriate) is a result of longstanding chronic hypertension. Other causes of an ICH are coagulopathy (anticoagulant or chronic liver disease) and underlying vascular abnormalities such as AVM, aneurysm or a hemorrhagic tumor (glioblastoma multiforme, metastasis), drug abuse (such as cocaine, amphetamine) and cortical venous sinus thrombosis. Even if the presenting symptoms appear mild at onset, hematoma progression may lead to rapid neurological deterioration (Kirollos et al. 2019; Fewel et al. 2003; An et al. 2017).

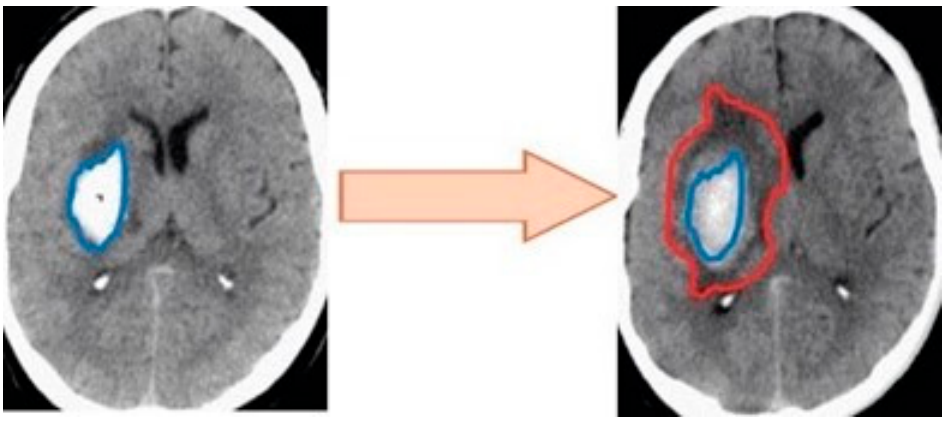

Figure 1. Putaminal hemorrhage with a mild space-occupying effect (**left side**); subsequent edema development around the primary lesion leads to compression of the midline structure (**right side**). Source: Figure by authors.

A hematoma produces ischemic penumbra, resulting in ischemia to the regional brain. Early neurological impairment, poor prognosis and death are all linked to hematoma enlargement. Between baseline and 1-h CT scans, Brott et al. discovered that 26% of ICH patients exhibited a significant bleeding expansion (defined as

a 33% rise from the baseline hematoma volume) (i.e., within 4 h of symptom onset). In addition, between the 1-h and 20-h CT scans, 12% of the patients exhibited a hematoma expansion. The progression of a hemorrhage was linked to early neurological impairment. Regardless of how hematoma enlargement is defined, it is an independent predictor of poor outcomes and mortality. The necessity of frequent neurological examinations and early repeat CT scanning, which might alter medical patient care or prompt surgical procedures, is highlighted by the early incidence of hematoma expansion and subsequent neurological deterioration (Qureshi et al. 2009; Fallenius et al. 2019; Cruz and Hopkins 1999; Greenberg 2010). Patients receiving warfarin (or with an international normalized ratio (INR) > 1.5) have an increased tendency of a hematoma expansion compared with patients not receiving warfarin.

2. Intracerebral Hemorrhage in Adults

An intracerebral hemorrhage is the second most common form of a stroke (15–30% of strokes). It has a progressive onset over minutes to hours. The volume of the hematoma correlates highly with morbidity and mortality. Generally, it starts during an activity (seldom during sleep) that may be connected to the elevation in blood pressure (BP) or increased CBF (Fewel et al. 2003; Fallenius et al. 2019; Greenberg 2010).

3. Risk Factors

- Age: risk rises markedly after the age of 55 years and twice with every additional ten years;
- Gender: more common in men;
- Race: higher prevalence in Asians and African Americans;
- Previous stroke: (any type) increases risk to 23:1;
- Alcohol consumption;
- Cigarette smoking;
- Street drugs: cocaine, amphetamines;
- Liver disfunction: coagulopathy (An et al. 2017; Fallenius et al. 2019; Greenberg 2010).

3.1. Major Risk Factors

1. Age;
2. Male sex;
3. Hypertension;
4. High alcohol intake;
5. Race—incidence of ICH among the Black population is twice as high as in the White population;

Low serum cholesterol—Japanese population diet has a low cholesterol level (Kirollos et al. 2019; Qureshi et al. 2009; Cruz and Hopkins 1999; Greenberg 2010).

3.2. Weak Risk Factor

1. Smoking;
2. Diabetes mellitus (Kirollos et al. 2019).

Frequent sites of ICH are listed in Table 1.

Table 1. Common sites of an ICH.

Percentage (%)	Location
50	Corpus striatum (basal ganglia); putamen is the commonest; along with lenticular nucleus, globus pallidus, internal capsule.
15	Thalamus
10–15	Pons
10	Cerebellum
10–20	Cerebral white matter
1–6	Brain stem

Source: Authors' compilation based on data from Cruz and Hopkins (1999) and Greenberg (2010).

3.3. Ganglio-Thalamic ICH: Etiologies

1. Hypertension

(a) Acute hypertension (HTN);
(b) Chronic HTN: degenerative changes within blood vessels.

2. Acutely increased CBF (globally or focally) especially to areas previously rendered ischemic:

(a) Carotid endarterectomy;
(b) Repair of congenital heart defects in children.

3. Previous stroke (embolic or otherwise): hemorrhagic transformation.
4. Vascular anomalies:

(a) AVM rupture;
(b) Aneurysm rupture.

5. Venous angioma rupture.
6. Arteriopathies: amyloid angiopathy, fibrinoid, lipohyalinosis, cerebral.
7. Brain tumor (primary or met).
8. Coagulation or clotting disorders: leukemia, thrombocytopenia, thrombotic thrombocytopenic purpura, aplastic anemia.
9. Patients receiving anticoagulation, thrombolytic, aspirin therapy.
10. CNS infection:

(a) Especially fungal, which attack blood vessels;
(b) Granuloma;
(c) Herpes simplex encephalitis.

11. Venous or dural sinus thrombosis.
12. Drugs:

(a) Substance abuse (alcohol, cocaine, amphetamine);
(b) Drugs that raise BP, alpha-adrenergic agonists (sympathomimetics): phenylpropanolamine, OTC alpha agonists (phenylephrine, ephedrine, pseudoephedrine).

13. Posttraumatic.
14. Pregnancy and puerperium (up to 6 weeks post-partum) most commonly associated with eclampsia or preeclampsia.
15. Postoperative: following carotid endarterectomy, craniotomy.
16. Idiopathic (Fewel et al. 2003; An et al. 2017; Greenberg 2010).

3.4. Lobar Hemorrhage

Lobar hemorrhages have a more benign outcome than ganglionic–thalamic hemorrhages.

Etiologies are (Greenberg 2010):

1. Extension of a deep hemorrhage;
2. Amyloid angiopathy of the brain (the commonest etiology of a lobar ICH in older normotensive sufferers);
3. Trauma;
4. Hemorrhagic changing of an infarct (ischemic);
5. Hemorrhagic;
6. Cerebrovascular malformation (especially AVM);
7. Rupture aneurysm;
8. Idiopathic.

4. Clinical Presentation of an ICH

Classical presentation is sudden the onset and rapid progression of a focal neurological deficit, along with headache, vomiting and an impaired level of consciousness. The severity of presentation depends upon the volume of a hematoma. When a hematoma is significant in volume (>60 mm^3), it causes brain herniation. When the volume of hematomas is more than 85 mL, brain herniation is more prominent and patients may die even after carrying out a proper decompression.

The symptoms and signs of a spontaneous ICH vary according to the site and size. Most cases of an ICH occur during daily routine activity. The neurological signs and symptoms generally increase progressively over minutes to a few hours, in contrast to a cerebral embolism, as well as subarachnoid hemorrhage, where the neurological signs and symptoms are often the highest at onset. However, a few patients with an ICH are

comatose or obtunded upon arrival to the emergency department or when first discovered. Headache and vomiting, as well as a reduced level of consciousness develop following an adequately large ICH. Headache and vomiting occur in nearly 50% of patients with an ICH. Headaches may be due to traction on meninges, blood in the cerebrospinal fluid (CSF) or increased intracranial pressure (ICP); they are the most common with lobar and cerebellar hemorrhages. These clinical features are absent with a small ICH; the clinical features in this situation are that of a slowly progressing stroke. Neck stiffness and meningism are seen in ICH with ventricular extension. Coma or stupor in ICH is a perilous sign. The sole exception is a thalamic ICH, where involvement of the reticular activating system is the etiology of stupor or coma rather than diffuse cerebral injury; these sufferers may recover once the ICH is reabsorbed. In putaminal hemorrhage, the spread of an ICH into the putamen commonly takes place along white fiber tracts, resulting in hemiplegia, homonymous hemianopsia, stupor, hemisensory loss, gaze palsy and coma. In the small internal capsule, hemorrhage restricted to the internal capsule may result in mild dysarthria and contralateral hemiparesis, as well as a sensory deficit. Cerebellar hemorrhage generally starts in the dentate nucleus, and spreads into the hemisphere and fourth ventricle. These ICHs result in imbalance, headache, vomiting, gaze palsy, neck stiffness and facial weakness without hemiparesis. Thalamic hemorrhage may extend transversely to the posterior limb of the internal capsule, inferiorly to put pressure on the tectum of the mesencephalon or may rupture into the 3rd ventricle. Symptoms are hemisensory loss, hemiparesis and rarely transient homonymous hemianopsia. Aphasia may result if the ICH affects the dominant cerebral hemisphere, while neglect may result if the ICH affects the nondominant cerebral hemisphere. Lobar hemorrhages vary in their neurologic signs based on the location. Often, the affected lobes are the parietal and occipital lobes. These are linked with a higher frequency of seizures. An occipital ICH commonly presents with a dense contralateral homonymous hemianopsia. The frontal ICH brings about a contralateral paresis or plague of the leg with relative sparing of the arm. A pontine hemorrhage is characterized by a medial ICH that extends into the basal pons. These often lead to deep coma over the first few minutes, probably as a result of the disruption of the reticular activating system. The motor examination may reveal total paralysis. The pupils are pinpointed but react to a strong light. Horizontal gaze palsy, ocular bobbing, facial palsy, deafness and dysarthria can be found when the patient is awake (Ahangar et al. 2019).

5. Diagnostic Imaging

5.1. CT Scan

A CT scan can give the diagnosis of an ICH rapidly. It is quick and can even preform in restless patients without aggressive sedation (Figure 2A–C).

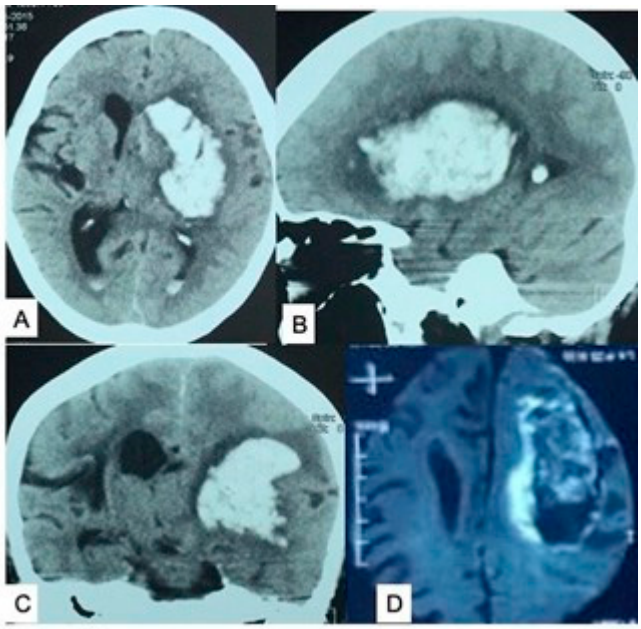

Figure 2. (**A**) CT scan of the brain: axial view showing intraventricular hemorrhage; (**B**) CT scan of the brain: sagittal view showing blood in the ventricle; (**C**) CT scan showing uncal herniation due to an ICH; (**D**) MRI showing an ICH in the frontoparietal lobe. Source: Figure by authors.

5.2. CTA

A CTA is needed in suspicious cases of an AVM and aneurysm ruptures.

5.3. MRI Scan

An MRI scan of the brain (Figure 2D) can clearly rule out bleeding from a cavernous angioma or intratumoral bleed. MRI appearance of an ICH are shown in Table 2.

Table 2. MRI appearance of an ICH in different stages.

Stage	Age	Condition of Hemoglobin	T1WI	T2WI
Hyperacute	<24 h	Oxy-Hgb	Iso	sl ↑
Acute	1–3 d	Deoxy-Hgb	sl ↓	Very ↓
Subacute				
Early > 3d		Met-Hgb	Very ↑	Very ↓
Late > 7d		Met-Hgb	Very ↑	very ↑
Centre > 14 d		Hemichromes	Iso	sl ↑
Chronic Rim		Hemosiderin	sl ↓	Very ↓

Source: Authors' compilation based on data from Greenberg (2010).

5.4. MRV

An MRV depicts the cortical and dural venous sinus and is needed in case of suspected dural venous sinus thrombosis.

Clinical symptoms of an ICH in relation to anatomical localization are shown in Table 3.

Table 3. Typical symptoms of an ICH in relation to the localization.

Typical symptoms of an ICH in Relation to the Localization
Putamen
• Contralateral hemiparesis
• Conjugate gaze deviation to the lesion side
• Homonymous hemianopia
• Aphasia if an ICH is on the dominant side
Thalamus
• Contralateral sensory symptoms
• Initial reduced conscious state progressing to coma
• Hemiparesis
• Hemiataxia (up to 20%)
• Oculomotor symptoms caused by pressure to the midbrain (i.e., Parinaud's syndrome)
• Neuropsychological deficit
Caudate nucleus
• Hemiparesis
• Often intraventricular involvement with meningism
• Pons
• Initial reduced conscious state progressing to coma
• Teraparesis
• Abnormal flexion or extension
• Bilateral cranial nerve deficits in medial lesions
• Tegmental localization: internuclear ophthalmoplegia, dilated pupil and contralateral hemiparesis
Midbrain
• Initial reduced conscious state progressing to coma
• Parinaud's syndrome cerebellum
Initial reduced conscious state progressing to coma
• Ataxia
• Dizziness
• Gaze palsies
• Signs of an elevated ICP

Source: Authors' compilation based on data from An et al. (2017), Qureshi et al. (2009) and Quiñones-Hinojosa (2012).

6. Herniation Syndrome

Uncal herniation (Figure 2C) is the most common herniation which is caused by a temporal lobe hematoma that shift the uncus medically and downward until it is herniated over the tentorium and compress the midbrain.

The earliest sign of an impending uncalled herniation is the unilateral dilated pupil secondary to the compression of the parasympathetic fiber which lies on the periphery of the 3rd nerve. When the uncalled herniation progresses, it is then associated with a rapid decline in the level of consciousness and sign of an ipsilateral or contralateral hemiplegia with an extensor planter response (due to ipsilateral or contralateral compression of cerebral peduncle, respectively). Eventually, a bilateral pupil disloyalty dilatation develops (Greenberg 2010).

There are various separate types of brain herniation that assert the type of herniation taking place:

- Subfalcine herniation;
- Transellar herniation: descending and ascending;
- Transtentorial herniation;
    - Caudal: central herniation as well as uncal herniation;
    - Cranial: ascending transtentorial herniation;
- Tonsillar (cerebellar) herniation;
- Extracranial herniation.

7. Management

7.1. Medical Management

7.1.1. Hypertension Management

Antihypertensive therapy is usually not indicated if the systemic BP is <180 mm Hg. If the systolic BP is 180 to 230 mm of Hg or more and/or the diastolic is 105 to 120 mm of Hg, then oral nifedipine or injectable labetalol is required.

An intracerebral hematoma varies in amount.

Calcium channel blockers (nicardipine or nimodipine, 16.2%), combination alpha- and beta-blockers (labetalol, 14.4%), venodilators (nitroglycerin, 14.9%), a diuretic (furosemide, 12.4%) and arterial vasodilators (furosemide, 12.4%) are among the most commonly prescribed medications (nitroprusside, 12.1%; hydralazine 5.9%). The ATACH 2 experiment used an intravenous infusion of nicardipine, starting at a dose of 5 mg/h and increasing by 2.5 mg every 15 min until the target SBP is reached (maximum dose of 15 mg/h). If the SBP target is not met despite the maximum dose of nicardipine, intravenous labetalol is administered as a second-line drug. The most commonly prescribed medicines in North America are nicardipine and labetalol; both appear to be safe, but nicardipine may be more effective in achieving and maintaining the target blood pressure (Kirollos et al. 2019; Fewel et al. 2003; An et al. 2017; Qureshi et al. 2009; Fallenius et al. 2019; Cruz and Hopkins 1999; Greenberg 2010; Quiñones-Hinojosa 2012; de Oliveira Manoel et al. 2016).

7.1.2. Anticoagulant-Associated ICH

Patients having an ICH on antithrombotic drugs carry a higher threat for an ICH expansion, as well as greater risk for mortality and poor results.

A. Warfarin-Induced ICH

Warfarin is to blame for 9–11% of all ICH cases. When compared to people who do not use anticoagulants, patients on long-term warfarin had an 11-fold increased risk of an ICH. Urgent coagulopathy reversal is required for patients with warfarin-related ICH and an elevated INR (>1.4). Vitamin K can entirely reverse the warfarin effect when administered as a slow intravenous infusion (5–10 mg over 30 min) (de Oliveira Manoel et al. 2016).

The transfusion of fresh frozen plasma (FFP):

- FFP is required when the INR is more than 5.
- Recombinant activated factor VII (rFVIIa) can be utilized for the reversal of warfarin-related coagulopathy.

B. Low-Molecular-Weight Heparin (LMWH)-Induced ICH

Heparin-related ICH affects about 0.1–0.2% of patients receiving continuous infusions. The heparin infusion should be stopped promptly, and 1 mg of protamine sulfate should be given for every 100 units of heparin given in the previous 2–3 h (maximum single dose of 50 mg) (de Oliveira Manoel et al. 2016).

C. Antiplatelet Agent (Ecosprin, Clopidogril)-Related ICH

In ICH, for patients on antiplatelets (APTs), the drug should be stopped instantly and there is an advantage from platelet transfusion.

Desmopressin (DDAVP) was tested in a recent pilot research to see if it could increase platelet function in individuals with an ICH and low platelet activity who were also taking aspirin. Platelet activity was raised by desmopressin (0.4 g/kg IV over 30 min) (de Oliveira Manoel et al. 2016).

7.1.3. Management of Diabetes Mellitus

Uncontrolled diabetes mellitus is one of the prime risk factors and most often associated with an ICH. The control of diabetes is of utmost importance for treatment and also prevention.

7.1.4. Fever

Besides infection, intraventricular hemorrhage may cause a prolonged fever. To return the temperature to normal, a cold saline infusion (4 °C, 2 L at 4 L/h) vs. nasopharyngeal cooling (60 L/min for 1 h) may be needed (de Oliveira Manoel et al. 2016).

7.1.5. Preventing Venous Thromboembolism (VTE)

Patients with an ICH are at an increased risk of a VTE, which has been shown to be up to four times higher than in patients with an ischemic stroke. Intermittent pneumatic compression devices, positioned at the time of hospital admission (strong recommendation and high-quality evidence) are used as an initial prophylaxis, followed by pharmaceutical prophylaxis with LMWH (de Oliveira Manoel et al. 2016).

7.1.6. Dysphagia

After a stroke, dysphagia is widespread, with a reported incidence ranging from 37 to 78%, depending on the method employed to identify it. Dysphagia is linked to a higher risk of pneumonia/pneumonitis (RR 3.17, 95% CI 2.07–4.87). Aspiration pneumonia can be prevented with NG tube feeding (de Oliveira Manoel et al. 2016).

7.1.7. Seizure Prophylaxis

In patients with an ICH, seizure frequency has been found to range between 8.1 and 10.6%, with status epilepticus occurring in 1–2% of cases. Phenytoin has been linked to a higher risk of adverse effects and poorer outcomes. Anticonvulsant prophylaxis is not recommended by the current AHA ICH recommendations (de Oliveira Manoel et al. 2016).

7.1.8. ICP Management

ICP management techniques include the head of the bed elevation of 30 to 45 degrees, CSF draining by an EVD, analgesia and sedation, normocapnic breathing and hypertonic solution administration (e.g., hypertonic saline or mannitol). Barbiturates therapy is indicated in resistant cases of hypothermia (de Oliveira Manoel et al. 2016).

7.2. Surgical Management

Commonly, we need surgical removal +/− decompressive surgery of the brain for hematoma removal (Box 1).

Box 1. Hematoma volume measurement.

The result of the ABC/2 formula is as below: The volume of an ellipsoid is **4/3π(A/2)(B/2)(C/2)**, where A, B and C are the three diameters. If π is estimated to be 3, then the volume of an ellipsoid becomes ABC/2, where A is the greatest diameter on the largest ICH section, B is the diameter right angle to A, and C is the number of axial cuts with ICH multiplied by the section thickness. Frequently, we categorize them according to the volume: Mild—>10 mL Moderate—30 to 60 mL Severe—more than 60 mL of ICH

7.2.1. Surgical Indication of an ICH

(a) Lesions with a marked mass effect, edema or midline shift on imaging;
(b) Symptoms (e.g., hemiparesis/plegia, aphasia, or sometimes just confusion or agitation) due to an increased ICP or mass effect;
(c) Volume: surgery for a moderate and large volume of hematomas;
(d) Continued raised ICP despite therapy;
(e) Quick deterioration (signs of brainstem compression), regardless of the location in a patient considered to be salvageable;
(f) Favorable location (lobar, cerebellar, external capsule, non-dominant hemisphere);
(g) Young patient (especially age $\leq$ 50 years) with a moderate-to-large lobar hematoma who is clinically worsening;
(h) Early intervention: surgery after 24 h from onset of symptoms or deterioration may be of less benefit;
(i) Patients having cerebellar hemorrhages larger than 3 cm in diameter, hydrocephalus or neurological deterioration;
(j) ICH linked with a surgically accessible structural lesion, such as an AVM or tumor (Greenberg 2010).

7.2.2. ICH Score

ICH score can be used for assessment of outcome (Table 4).

Table 4. ICH score.

Component	Points	Total ICH Score	30-Day Mortality (%)
Glasgow Coma Scale			
3–4 5–12 13–15	2 1 0	0	0–10
Age (years)			
≥80 <80	1 0	1	7–13
ICH volume (mL)			
≥30 <30	1 0	2	30–44
Presence of intraventricular hemorrhage			
Yes No	1 0	3	56–78
Infra-tentorial origin of ICH			
Yes No	1 0	4	70–100
Total ICH score	0–6	5–6	100

Source: Authors' compilation based on data from de Oliveira Manoel et al. (2016) and Hemphill et al. (2001).

The first column shows the five independent predictors of 30-day mortality based on the original ICH score (Glasgow Coma Scale, age, ICH volume, intraventricular hemorrhage, and infra-tentorial location of ICH). The total score, which ranges from 0 to 6, is the sum of the five components (column 3). The expected 30-day mortality increases as the total score (column 3) rises (column 4). The key factors in selecting the appropriate surgical candidate is the size of the hematoma, age of the patient, comorbidity, location of hematoma (lobar vs. deep) and clinical course of the patient (Greenberg 2010; Hemphill et al. 2001). In our experience, young patients need surgical decompression more than older patients. Some neurosurgeons prefer only bony and dural decompression, keeping the deep-seated hematoma intact. Some prefer hematoma removal, as well as bony and dural decompression.

7.2.3. Surgical Approaches

1. Trans-Sylvian approach: Commonly, we use the trans-Sylvian approach (Figures 3 and 4) to remove a basal ganglionic hematoma. It is the shortest and most manageable trajectory for hematoma removal. The cortical opening is very minimal and the insular incision is given depending on the maximum location of the hematoma. Following insular incision and by gentle sucker manipulation, the hematoma commonly comes out spontaneously. The surgeons need controlled suction for gentle decompression. Often, the brain also becomes slack when a hematoma comes out. Interestingly, blood pressure also declines rapidly at this time. Therefore, the surgeons

often do not keep the patient antihypertensive before surgery. The surgeons also try not to administer mannitol if early surgery can be done. Following hematoma evacuation, it is needed to visualize the floor of the hematoma cavity to see the bleeding or oozing point. It needs proper coagulation if there is a bleeding point. Blood pressure needs to be raised at this level for proper hemostasis. Following hemostasis, a dural closure and bone placement are carried out, and the wound is closed in multiple layers.

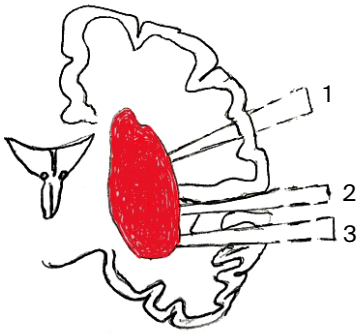

Figure 3. Surgical approaches to an ICH in basal ganglia. Source: Figure by authors.

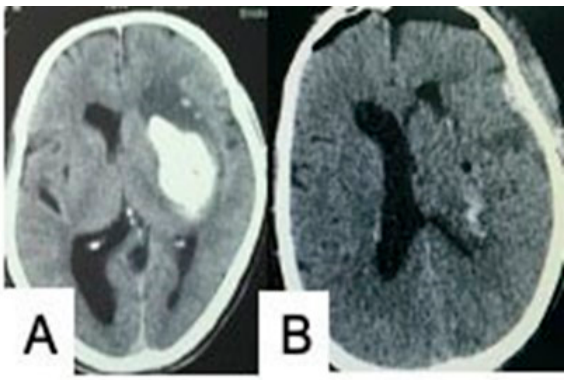

Figure 4. (**A**) CT scan showing a left-sided huge basal ganglionic hematoma. (**B**) Postoperative CT scan showing good removal of the hematoma, with no midline shift. Source: Figure by authors.

Sometimes, in young patients, if the surgeon suspects the patient may have developed brain swelling, he may offer some more bone removal in the temporal area and keep the dura open, with placement of an artificial dura to cover the exposed brain. Following decompression, commonly the bone is harvested in the subcutaneous plane of the abdomen just below the umbilicus. In some cases, the surgeons also offer a resection of the temporalis muscle and keep the bone over the temporal fascia just beneath the scalp to accommodate brain swelling. Figure 4 shows an ICH evacuation through trans-Sylvian approach.

2. Keyhole endoscopic removal: In this approach, a 0-degree endoscope and a sheath measuring about 10–17 mm in diameter and 5–10 cm long are needed. An endoscope can be held by an assistant or by an endoscope holder, and the surgeon removes the hematoma by direct endoscopic vision. This approach is carried out along the long trajectory of the hematoma cavity to get the parallel vision of the hematoma cavity by a 0-degree telescope.

It is a minimal, invasive procedure. It also needs proper hemostasis before the removal of the sheath. A hematoma cavity is properly irrigated and any bleeding point needs to be cautery by either monopolar or bipolar cautery. A hematoma cavity can be packed by surgical or gel foam following proper hemostasis to avoid recurrent hemorrhage. Wound is closed in layers keeping dura open and placement of bone commonly not needed as small opening. A hematoma evacuation can be successfully performed by direct endoscopic vision using a 0-degree endoscope. The trajectory of hematoma removal is considered along the longitudinal direction of the hematoma volume. A sheath with an obturator is mandatory to protect the brain as the trajectory is a little long. There are various types and lengths of the plastic sheath with an obturator available. The suction tube must have a teardrop for controlled suction. Fine unipolar or bipolar cautery is required for a proper hemostasis of the hematoma cavity (Kalangu et al. 2009).

3. Burr hole drainage and irrigation with urokinase (Macdonald 2018).

4. EVD for an intraventricular hemorrhage: External ventricular drains (EVDs) are used to treat ICH patients with an IVH, who have developed an obstructive hydrocephalus. Unfortunately, when there is a lot of IVH

present, the EVD fills up with blood, which clots and obstructs the EVD regularly. The surgeon can provide 5 mg of intraventricular tissue plasminogen activator (tPA) twice a day for 5 days in these instances. If the intracranial pressure (ICP) transduced by the EVD does not rise after each dosage, the EVD is clamped for 30 min to prevent the tPA from leaking out of the ventricle (Quiñones-Hinojosa 2012).

7.2.4. Complication of Surgical Management

1. Rebleed: There may be rebleed in a keyhole hematoma removal approach. Some patients may also develop brain swelling, especially those who are young and hence need to go for decompressive surgery.
2. Residual hematoma (Figure 5A).
3. Brain swelling (Figure 5B).

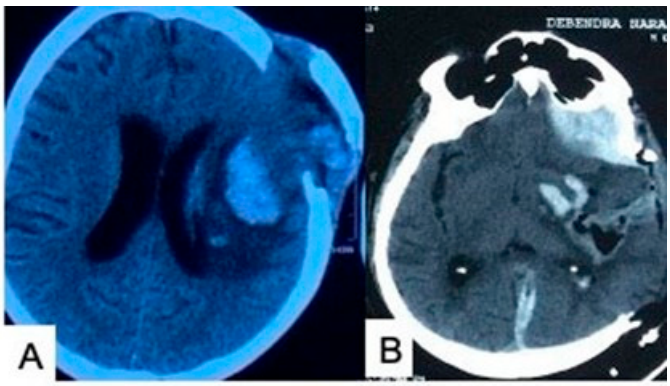

Figure 5. (**A**) CT scan showing brain swelling following surgery. (**B**) CT scan showing a residual hematoma with a perilesional edema, causing a raised ICP and extracalvarial herniation. Source: Figure by authors.

The earliest sign of an impending brain herniation from massive brain swelling is a depressed level of consciousness and a contralateral hemiparesis or ipsilateral hemiparesis (Kernohan's notch phenomenon) with a fixed and dilated ipsilateral pupil. Medical therapy by hyperosmolar therapy, hyperventilation, diuretic therapy, CSF drainage and barbiturate therapy is recommended. Surgical decompressive craniectomy is often required when the medical theory is failed or cannot be initiated.

4. Infection—meningitis and post-meningitis hydrocephalus.

7.2.5. Contraindications of ICH Surgery

1. Elderly patient (age more than 80 years);
2. Poor neurological status (GCS 5 or below);
3. Poor general condition (unable to tolerate general anesthesia);
4. Asymptotic patient with a small blood clot (<3 cm in cerebellar hematoma and <4 cm in lobar location).

7.2.6. 30-Day Mortality

An ICH has a 30-day mortality rate for 44%. Half of the deaths occur within first 2 days.

For pontine and other brainstem ICH, the mortality rate is 75% within 24 h.

For all ICH locations, the volume of an ICH is the highest predictor of 30-day mortality. Patients with a parenchymal bleeding volume of 60 cm^3 or more on their initial CT scan and a GCS of 8 or less had a predicted 30-day mortality of 91% in one research involving 188 cases. Patients with a GCS of 9 or above and a volume less than 30 cm^3 had a predicted 30-day death rate of 19% (Der-Yang Cho et al. 2008).

In individuals with spontaneous intracerebral hemorrhage, the volume of an ICH combined with the initial GCS score is a powerful and simple predictor of 30-day death and morbidity. The basal ganglia are the most prevalent site of a hypertensive ICH, accounting for 60% of all cases, and a basal ganglion ICH is linked with a 50% death risk (Quiñones-Hinojosa 2012). When there is a third ventricular outlet obstruction, a thalamic ICH is nearly invariably treated medically, with the installation of an external ventricular drain. The only study that examined surgical and medicinal treatment for a thalamic ICH found that an endoscopic evacuation had no advantage over medical treatment.

Because of the difficulties in gaining safe surgical access to the brainstem and the morbidity associated with brainstem manipulation required for a hematoma evacuation, most patients with a pontine hematoma are handled conservatively. A pontine ICH is expected to have an 18% death rate during hospitalization and a 69% mortality rate after a year (Quiñones-Hinojosa 2012).

A cerebellar ICH, when large in volume, causes brainstem compression and rapid fatal worsening. A second cause of mortality as well as morbidity for these patients is an ICH causing compression of the 4th ventricle and a subsequent hydrocephalus (Greenberg 2010).

7.3. Rehabilitation

It is likely that patients will need specialist rehabilitation. The extent of this will be dependent on their symptoms and how poorly they have been. This is specific to what is available at their local hospital or what the specialist team thinks will benefit the patients the most. This includes medical input from a specialist in rehabilitation, occupational therapy, physiotherapy, language and speech therapy, and dietetic evaluation of nutrition and psychology. It may include the following:

- Specialist inpatient rehabilitation;
- Referral back to the hospital the patient came from for the specialist stroke rehabilitation.

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