Chapter 2

Intracerebral Hematoma

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Additional information is available at the end of the chapter

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Abstract

Intracerebral hematoma occurs in about 35/100,000 population and the incidence is likely increase over the next few decades as the population ages. The most common causes are hypertension and amyloid angiopathy. Bleeds due to these two causes are classified as primary while all other causes, such as AVM bleeds, coagulopathies, and so on, are classified as secondary. Primary tissue damage due to the intracerebral hematoma is followed by edema, neuronal damage, and secondary damage due to cellular breakdown. Basal ganglia are the most common site of intracerebral hemorrhage, accounting for nearly 50% of cases. CT scan, CT angiogram, DSA, and MRI are the investigations of choice. The initial management is medical, with control of blood pressure and antiedema measures forming the mainstay of treatment. Surgical option includes external ventricular drainage, endoscopic evacuation of hematoma, craniotomy and evacuation of hematoma, and decompressive craniectomy and is usually reserved for patients who deteriorate while on treatment.

Keywords: intracerebral hematoma, management, guidelines

1. Epidemiology

Intracerebral hemorrhage (ICH) or hemorrhage within the brain parenchyma is the second most common cause of all cases of sudden neurological deficits following strokes and has the highest mortality amongst all varieties of stroke [1]. A study in 1993 [2] showed that 1-year survival following an ICH was 38% and this had improved to only 52% by 2009 [3]. In-hospital mortality has remained stable at around 34% over the last three decades [4], all of which makes prevention a cornerstone of treatment of this devastating disease. The incidence of ICH in the United States is estimated to be about 24.6/100,000 of person years, ranging from 1.8 to 129 per 100,000 person years [5] and this is likely to go up over the next few decades as the percentage of the elderly in the population increases.

One of the major risk factors identified with ICH has been that of race, with Asian populations being affected almost twice as much as other races. Whites appear to have a lower incidence when compared to nonwhites. The second risk factor to be identified was age, with those over 85 years of age having a 10-fold increase in the incidence of ICH compared to younger patients. Women were found to have a 15% lower chance of hemorrhages, but this was not statistically significant [6]. The single most important modifiable risk factor associated with ICH has undeniably been hypertension. A meta-analysis of 11 case control studies found the risk of bleeding in hypertensives to be 3.5 times above that in normotensives [7] while a multicentric case control study put the risk at nine times above that in normotensives in patients who had a blood pressure of over 160/90 mm Hg [8]. Even increases within the normal range of blood pressure have been associated with a linear increase in the risk of ICH [9].

Increased alcohol intake in the 24 hours preceding the onset of bleed as well as during the week prior to the ictus have been identified as independent risk factors for ICH [10] and the location of the hematoma in these patients tended to be lobar [11]. While high cholesterol levels have been associated with increased risk for ischaemic stroke, low cholesterol has been associated with an increased risk of cerebral micro bleeds and ICH. However, the exact association of different lipid fractions with this risk has yet to be elucidated [12]. Recent studies have shown that apolipoprotein E (APOE) ε2 and ε4 are independent risk factors for lobar ICH, which stands to reason considering their association with amyloidosis [13]. Tobacco smoking has been consistently associated with occlusive diseases such as coronary and peripheral vascular disease, as well as ischaemic stroke and subarachnoid hemorrhage, but its association with ICH is tenuous at best [14].

2. Etiology

ICH has been divided into primary and secondary varieties based on the underlying pathology. When no underlying pathology such as vascular malformation or coagulopathy is detected, and the primary cause of the bleed is due to rupture of small vessels in the brain, usually secondary to chronic damage from long-standing hypertension or cerebral amyloid angiopathy, the bleed is considered primary (**Figures 1** and **2**). Nearly 80% of all intracerebral hematoma cases fall into this category [15].

Chronically elevated blood pressure causes smooth cell hyperplasia in the cerebral arteries, followed by death of smooth muscle cells. There is also an increase in the stiffness of arterial walls in these patients, and this loss of elasticity may predispose to arterial rupture with sudden elevations in blood pressure. Electron microscopic studies have shown that most ruptures occur near the bifurcation of arteries, where there is degeneration of tunica media. This may be why most of the bleeds that occur in hypertension are located in deeper parts of the brain [16, 17]. Cerebral amyloid angiopathy, in contrast, features amyloid deposition in the leptomeningeal and intraparenchymal cortical vessels, resulting in superficial or lobar hemorrhages. Patients with amyloidosis are more likely to be older (over 60 years of age) and have hematoma sizes greater than 30 cc while those with hypertension are younger and have hematoma volumes less

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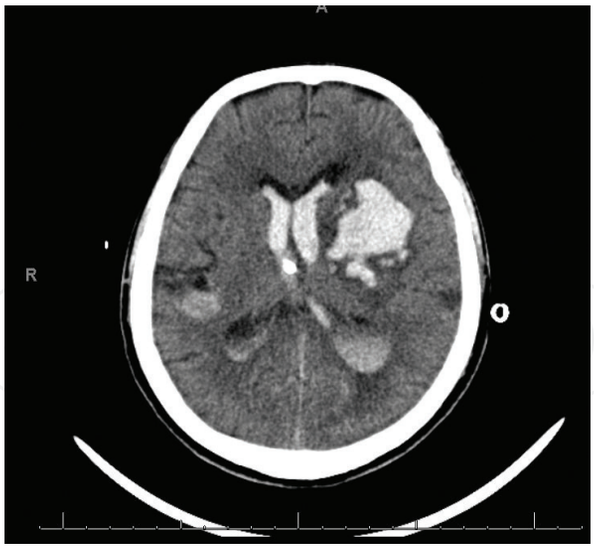

Figure 1. Hypertensive left ganglionic hematoma with IVH.

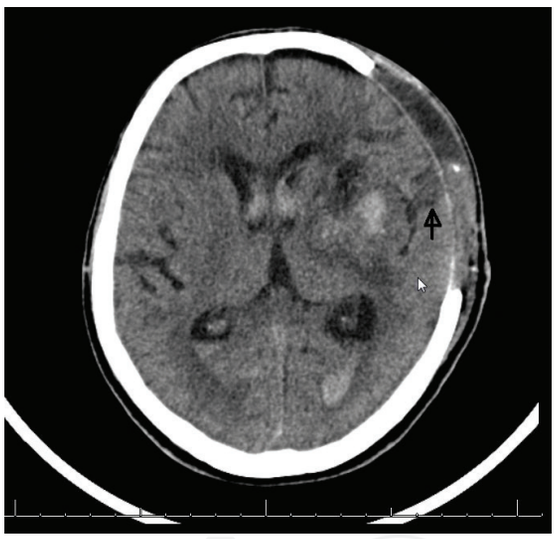

Figure 2. Same patient as in **Figure 1**, after surgical evacuation of hematoma following neurological deterioration. The black arrow shows widening of Sylvian fissure, which was split to approach the hematoma from its most superficial area. The bone flap was not replaced due to severe brain swelling.

than 30 cc [18]. A history of recurrent episodes of intracranial bleeds is likely to favor a diagnosis of cerebral amyloid angiopathy. It should, however, be borne in mind that these characteristics are nonspecific and need to be confirmed by histopathology if possible [19].

Bleeding from vascular malformations such as arteriovenous malformation (**Figure 3**), cavernous angiomas, and dural arteriovenous (AV) fistulae, hemorrhagic conversion of an ischemic stroke, bleeding from intraparenchymal tumors, ICH occurring in patients with bleeding diathesis and in those taking anticoagulants (**Figure 4**) all come into the category of secondary ICH [20]. Tumor bleeds typically occur in metastases, such as melanoma, choriocarcinoma, renal carcinoma, or thyroid carcinoma, and from high grade gliomas [21]. Aneurysmal bleeds

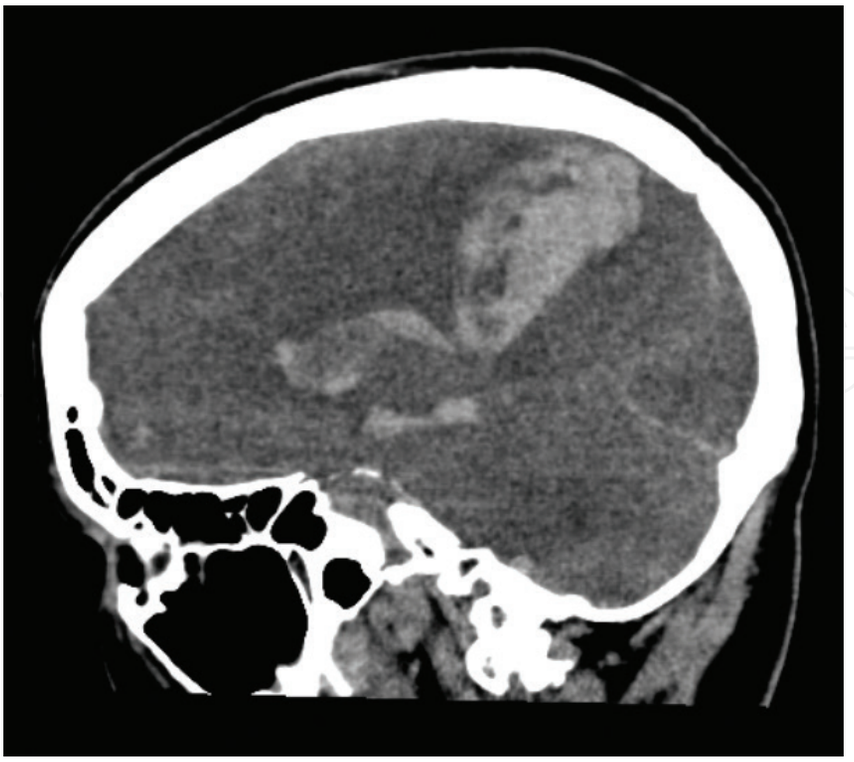

Figure 3. Large lobar hematoma with IVH due to a vascular malformation.

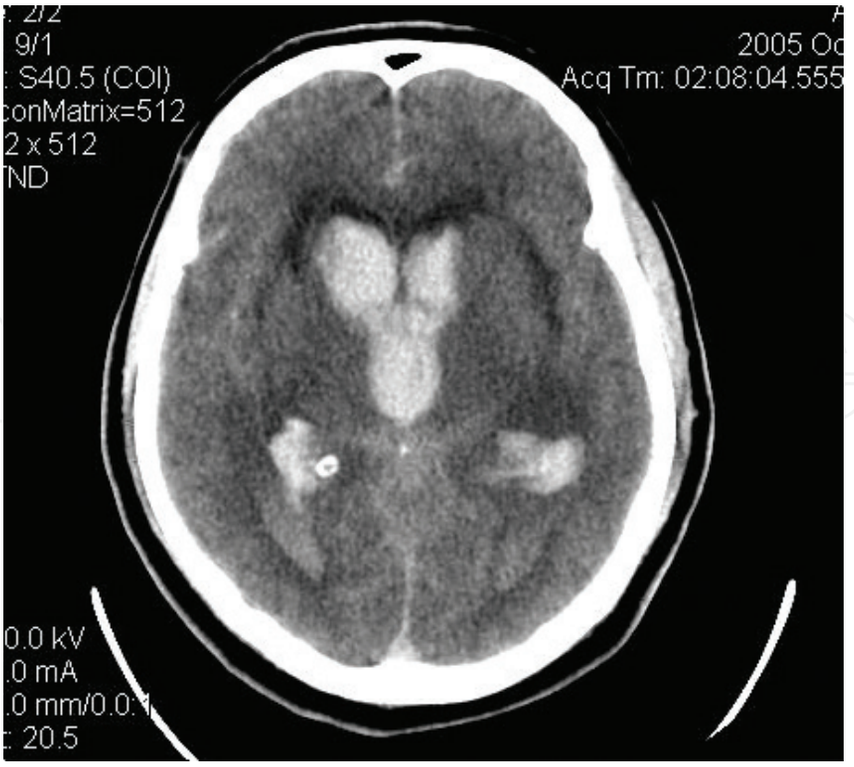

Figure 4. Isolated IVH in an anticoagulated patient.

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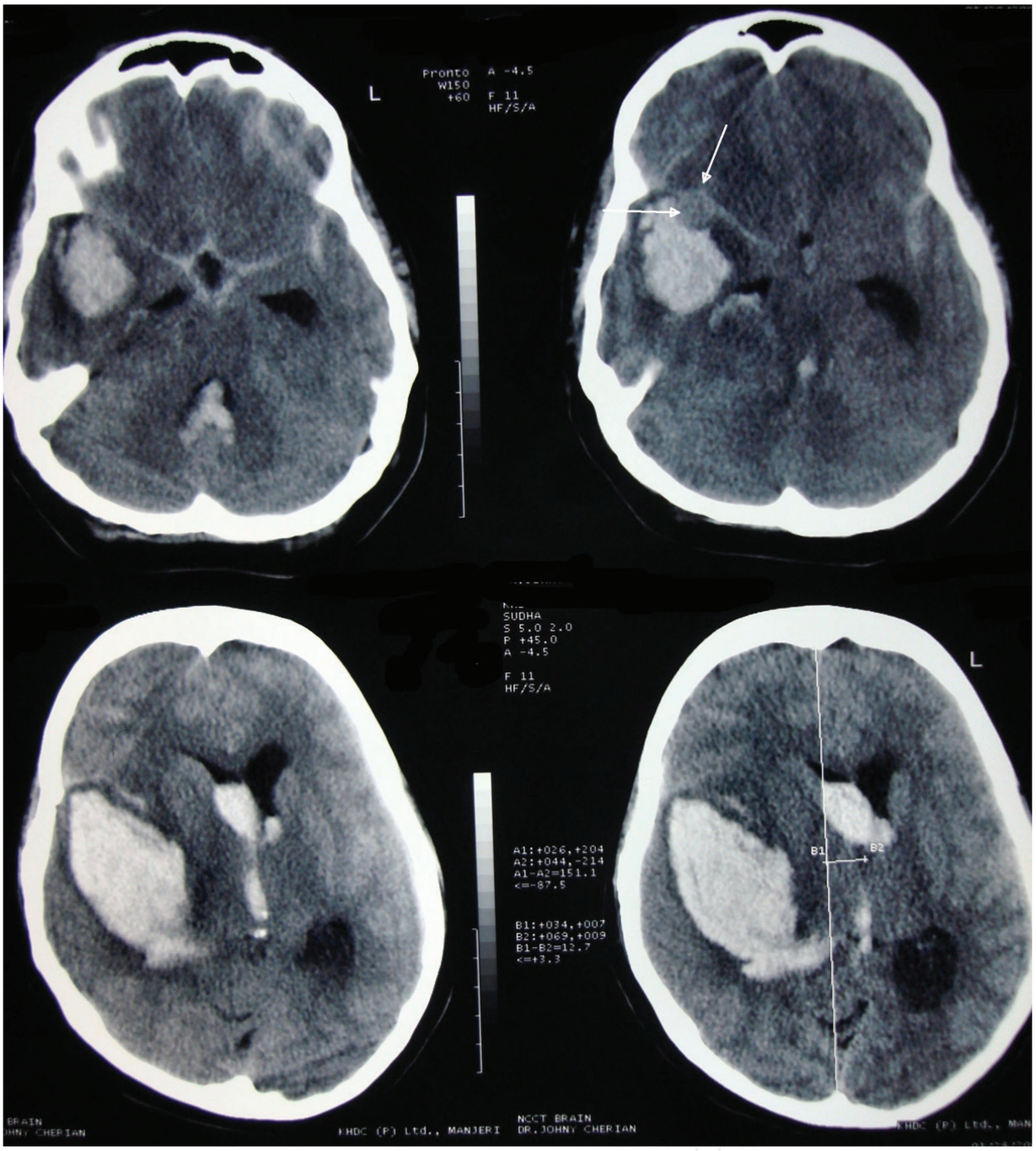

Figure 5. This patient had a bleed which looked like a ganglionic hematoma with IVH, but closer observation showed a partially thrombosed MCA aneurysm (white arrows, top right).

(**Figure 5**) may sometimes present with an intraparenchymal component and an ICH may occasionally be the result of a sinus thrombosis [22]. Recently, patients with hepatitis C infection have been identified as having a higher risk of ICH compared to a control group without HCV infection. The risk was higher in younger patients and increased with increasing severity of the viral infection [23]. Drug abuse, especially cocaine use, has been found to cause an increased incidence of subcortical hemorrhages with intraventricular extension and these

patients have a poorer prognosis when compared to nondrug users with spontaneous ICH [24]. Patients or oral warfarin have an 8- to 14-fold increase in the risk of ICH compared to the normal population, but the risk appears to be lower in those being treated with newer anticoagulants such as dabigatran [25, 26].

3. Pathophysiology

Intracerebral hemorrhage results in primary damage due to the injury to neural tissues. This is followed by secondary damage resulting from increased intracranial pressure as well as the presence of intraparenchymal blood. The secondary damage occurs through several pathways that run concurrently, eventually leading to the loss of the blood brain barrier and severe cerebral edema resulting in extensive cellular lysis. As in traumatic brain injury, early removal of the hematoma and cellular debris, either by surgical removal of the clot or by the action of the inflammatory cells such as microglia and macrophages, help to reduce the extent of secondary damage [27, 28]. Animal studies have shown that perihematoma edema can be divided into three phases: immediate (up to 24 h after hemorrhage), intermediate (from 24 h to 5 days), and late (beyond 5 days) [29]. The immediate edema results from osmotically active proteins accumulating in the extravascular compartment and can be seen on histological studies, but not on imaging. Intracerebral hemorrhage results in primary damage due to the injury to neural tissues. This is followed by secondary damage resulting from increased intracranial pressure as well as the presence of intraparenchymal blood. The secondary damage occurs through several pathways that run concurrently, eventually leading to the loss of the blood brain barrier and severe cerebral edema resulting in extensive cellular lysis. As in traumatic brain injury, early removal of the hematoma and cellular debris, either by surgical removal of the clot or by the action of the inflammatory cells such as microglia and macrophages, help to reduce the extent of secondary damage [30]. Red cell destruction due to activation of the clotting cascade releases thrombin which again causes disruption of the blood brain barrier, failure of the sodium pump, and increase in the edema [31, 32]. This intermediate edema can be visualized radiologically. The late cerebral edema results from oxidative damage due free radical release which is a result of cellular destruction and hemoglobin breakdown.

Several additional pathways for cellular damage following intracerebral hemorrhage have been advocated, such as apoptosis or programmed cell death associated with the expression of nuclear factor-kB in neuronal nuclei [33] and breakdown of extravasated heme into bilirubin and bilirubin oxidation products, which activate microglia. The microglia then activate leucocyte adhesion molecules on endothelial walls, leading to an influx of leucocytes into the brain. The activated microglia produce cytokines which along with the leucocytes mediate further cell injury [34].

The commonest site for an intracerebral hematoma is the basal ganglia, with the putamen being the preferred location. Nearly 50% of all ICH cases involve in the basal ganglia, followed by the thalamus, pons, cerebral white matter, and brainstem. The source of bleeding is usually a Charcot-Bouchard micro-aneurysm, arising from either the lenticulostriate arteries in case of putaminal bleeds or thalamoperforators in case of the thalamic bleeds. The paramedian branches of the basilar artery are the source for basilar and cerebellar hemorrhages [35].

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4. Presentation

The clinical presentation of intracerebral hemorrhage depends on the location of the bleed and its size. Smaller bleeds in non-eloquent areas may present with headache, nausea, and vomiting. Larger hemorrhages into the frontal lobe may be associated with contralateral hemiparesis, mainly involving the upper limbs, and aphasia if the dominant lobe is involved. Parietal lobe involvement causes contralateral hemisensory impairment with mild hemiparesis and cognitive impairment. Occipital hematomas may present with pain in the ipsilateral eye and contralateral homonymous hemianopia. Dominant temporal lobe involvement causes Wernicke's aphasia which may be associated with poor auditory comprehension, while nondominant temporal lobe hemorrhage may be asymptomatic unless it is large enough to cause symptoms due to the mass effect. Putaminal hemorrhages may present with a range of symptomatology, from minimal pure motor deficits on the contralateral side to severe sensorimotor impairment, aphasia, neglect, gaze deviation, and impaired level of consciousness. These patients have a gradual deterioration in clinical symptoms from the time of onset with a 30-day mortality as high as 50%, but only a small percentage has headache as a presenting symptom [36]. In contrast, nearly 30% of patients with a thalamic bleed present with headache. They have hemisensory disturbances that are out of proportion to the minimal weakness. Significant weakness may be found when the hematoma extends into the internal capsule and associated eye signs indicate involvement of the midbrain.

Tanaka et al. [37] found that when compared to putaminal hemorrhages, thalamic hemorrhages were associated with a more pronounced reduction in cerebral blood flow (CBF)

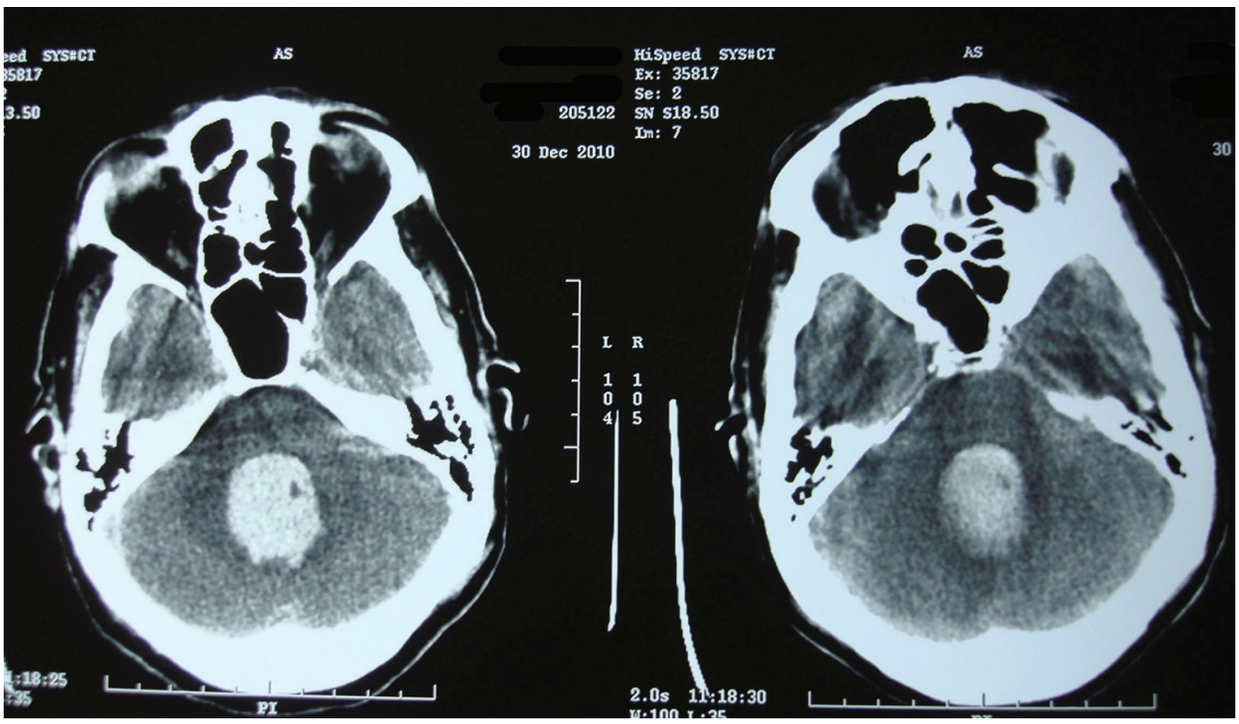

Figure 6. Preoperative images of a vermian hematoma with intraventricular extension in a patient with hemophilia.

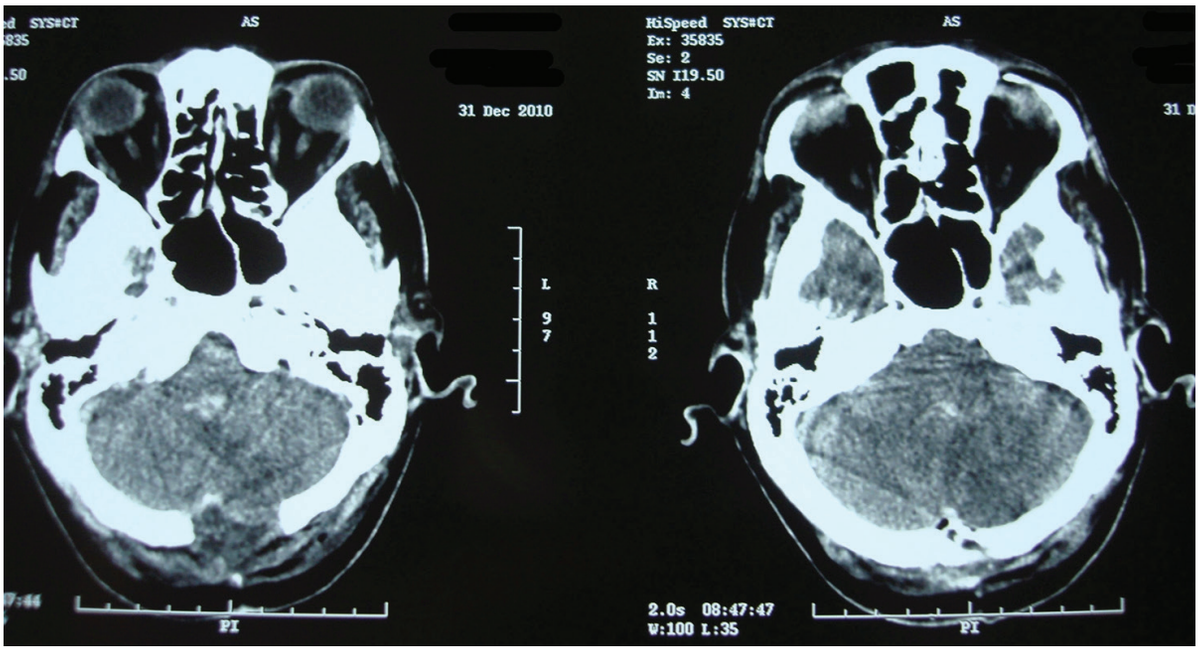

Figure 7. Postoperative images of a vermian hematoma with intraventricular extension in a patient with hemophilia.

bilaterally, even though their hematoma volumes were much smaller. They hypothesized that the reduction of CBF may be secondary to metabolic depression due to transneuronal or functional depression ("diaschisis"). The metabolic depression in thalamic hemorrhages is more extensive and persistent than in putaminal hemorrhages, which probably accounts for the reduced CBF and worse outcome in thalamic hemorrhages. While several studies have quoted a size of 3.3 cm as a determinant for survival in patients with thalamic hemorrhages [38–40] in a prospective trial that included 100 patients there were three survivors with hematomas larger than 3.3 cm who were able to lead independent lives [41]. However, sizes of the hematoma and intraventricular extension were two major factors that were found to correlate with the eventual outcome.

Cerebellar ICH may present with limb ataxia, ipsilateral gaze palsy, cranial nerve deficits such as an abducens or peripheral facial palsy, and nonspecific symptoms like headache and vomiting. In a series of 56 patients, Ott et al. found that nearly three-fourth of the patients had one of the first three signs described [42]. As in other types of ICH, intraventricular extension, initial GCS, and older age were associated with a poor prognosis in patients with cerebellar ICH (**Figures 6** and **7**) [43].

5. Evaluation

A plain computerized tomography (CT) scan of the brain is the standard investigation performed in all suspected intracranial pathologies which present as an emergency. In cases of ICH, CT scan will usually reveal a hyperdense lesion within the brain parenchyma, with

possible intraventricular or subarachnoid components. The latter typically occurs with anterior communicating or middle cerebral artery aneurysms.

The volume of the hematoma can be calculated by the modified ellipsoid volume calculation where the anteroposterior (A), right to left (B), and cranio-caudal lengths of the clot (C) are measured. The last (that is, C) is calculated by counting the number of slices in which the clot is seen and multiplying it by the slice thickness. The formula (A×B×C)/2 can then be used to calculate the clot volume [44]. On multivariate analysis in several studies, initial Glasgow Coma Scale score, hematoma volume, and an infratentorial location of hemorrhage were found to correlate strongly with the outcome of intracerebral hematoma. Univariate analysis had also implicated higher initial and 48-h maximum glucose concentrations, and higher percentage of ICH expansion as being significantly associated with poor functional outcome at hospital discharge. In anticoagulated patients, the initial INR or the time to INR correction did not affect the outcome [45].

Magnetic resonance imaging (MRI) is rarely the investigation of choice in the acute scenario due to various reasons. A complete study requires much more time than a CT scan and the spontaneous movements of a partially obtunded patient may cause imaging artefacts and significantly prolong the imaging time. If the patient worsens clinically while undergoing imaging, it may be difficult to access him immediately. Initially, it was felt that small amounts of hyperacute blood were difficult to visualize in many imaging sequences, but a study by Linfante et al. showed that MRI scans were capable of demonstrating a hyperacute bleed within 2 h of symptom onset [46]. An advantage of MRI is the ability of gradient echo sequences to distinguish hemorrhages of varying ages, which is extremely useful in amyloid angiopathy and cavernous angiomas.

While initial investigators believed that the progressive neurological deterioration that occurred in the hours following an intracerebral hemorrhage were due to the mass effect of the hematoma, radiological studies have shown that early hematoma growth occurs in up to 38% of all patients with ICH [47]. This has led to efforts to identify the factors that promote rehemorrhage, so as to identify the patients at risk of deterioration, as well as to develop management strategies to prevent a rebleed. The major risk factors for a rebleed include uncontrolled hypertension with a systolic blood pressure more than 195 mm Hg, a previous infarction at the site of the hemorrhage, alcoholism which predisposes to liver disease and which in turn leads to coagulopathies, anticoagulant use, high white cell count, and hyperthermia [48]. Contrast extravasation on CT angiography was found to correlate well with the risk of rehemorrhage [49]. The presence of tiny enhancing foci, known as the "spot sign" on axial images of a 3D CT angiogram has been reported to be associated with a higher risk of hematoma expansion [50]. This correlation was found to be true especially when the Hounsfield units (HU) of these foci were in the range of 192.12 ± 45.97 while patients with spot signs having a lower HU of 151.10 ± 25 did not suffer from a hematoma expansion [51]. The surgical trial in intracerebral hemorrhage (STICH trial) introduced the concept of a prognostication score for ICH, which was calculated using the equation (10 × admission GCS) – age (years) – (0.64 × clot volume (ml)) [52].

If there is a suspicion of a vascular anomaly as the main cause of the hemorrhage, and if the CT angiogram is noncontributory, a catheter angiogram (cerebral DSA) should be considered.

6. Medical management

The optimum treatment for intracerebral hemorrhage is an area of ongoing research and changing guidelines (**Table 1**). While the STICH trial went some way in answering some of the questions regarding the role of surgery, it also raised a number of issues which are yet to be resolved [53]. The initial, prehospital and emergency room management for patients with ICH is the same as those for patients with ischemic stroke and was elucidated in the guidelines published by the American Heart Association (AHA) and the American Stroke Association in 2013. A severity score should be calculated as soon as possible, so as to enable prognostication (**Tables 2** and **3**). If facilities for stroke care are not available in the hospital, the patient should be transferred to a tertiary care center as early as possible.

No.	Problem	Recommendation	Class of evidence
1	Initial presentation in emergency	Perform baseline ICH severity score	I
2	Differentiate between ischemic and hemorrhagic stroke	CT or MRI scan	I
3	Possibility of hematoma expansion	Contrast CT and CT angiogram to be considered	IIb
4	Vascular anomaly	CT or MR angiograms (arterial/venous studies) and DSA to be considered	IIa
5	Management of coagulation defects	Correct coagulopathy or thrombocytopenia with appropriate therapy	I
6	Patients on vitamin K antagonists (VKA)	Stop VKA, give intravenous vitamin K	I
7		Fresh frozen plasma (FFP) or prothrombin complex concentrates (PCC)	IIb
8	Patients on heparin	Protamine sulphate	IIb
9	Patients on newer anticoagulants	PCC, factor VIII inhibitor bypassing activity (FEIBA) or rFVIIa, hemodialysis and activated charcoal if drug was ingested within 2 h	IIb
10	Prevention of deep venous thrombosis (DVT)	Intermittent pneumatic compression	I
		Low molecular weight heparin to be considered after 1–4 days once cessation of hematoma expansion is documented	IIb
11	Established DVT	IVC filter or systemic anticoagulation	IIa
12	Hypertension (systolic BP 150–220 mm Hg) without contraindication for acute lowering of BP	Lower SBP to 140 mm Hg	Ia
	Hypertension (SBP > 220 mm Hg)	Aggressive reduction of SBP	IIb

No.	Problem	Recommendation	Class of evidence
13	High blood sugar	Control adequately, avoiding hypoglycemia	I
14	Fever	Control adequately	IIb
15	Seizures	Clinical seizures and abnormal EEG in patients with altered mental status should be treated	I
		Continuous EEG monitoring in patients with altered mental status that is out of proportion to extent of brain injury	IIa
16	Concurrent cardiac events	ECG and cardiac enzymes to be checked	IIa
17	Hydrocephalus	Ventricular drainage	IIa
18	GCS less than or equal to 8, presence of IVH	ICP monitoring, maintain CPP at 50–70 mm Hg	IIb
19	IVH	Intraventricular rtPA-efficacy uncertain	IIb
20	Cerebellar hematoma, neurological deterioration	Surgical evacuation	I
21	Supratentorial ICH, neurological deterioration	Surgical evacuation	IIb
		Decompressive craniectomy	IIb
		Minimally invasive clot evacuation-efficacy uncertain	IIb
22	Aspiration pneumonia	Screen for dysphagia before starting oral feeds	I

Table 1. Evidence-based recommendations on management.

Component	Value	Points
Glasgow Coma Scale (total score)	3–4	2
	5–12	1
	13–15	0
Intracerebral hematoma volume	≥30 cm^3	1
	<30 cm^3	0
Intraventricular hemorrhage	Present	1
	Absent	0
Origin of intracerebral hematoma	Infratentorial	1
	Supratentorial	0
Age	≥80 years	1
	<80 years	0

Table 2. ICH severity score [89].

Total ICH score (points)	Mortality rate (%)
0	0
1	13
2	26
3	72
4	97
5–6	100

Table 3. The total ICH score obtained by adding all the component scores from **Table 1** can be used to prognosticate the mortality as given below.

The patient has to be admitted into a dedicated stroke unit or an intensive care unit, and invasive (arterial) blood pressure monitoring should be instituted as soon as possible for control of hypertension. The INTERACT2 trial showed a significantly better outcome for patients whose systolic blood pressure (SBP) was less than 140 mm Hg compared against a group where the SBP was less than 180 mm Hg [54]. Therefore, an SBP of 140 mm Hg should be targeted for all patients admitted with ICH. If multiple, long-term, intravenous access is anticipated, a multilumen central venous line can be inserted if the coagulation profile is normal.

Patients with a low Glasgow Coma Scale score who are not candidates for early surgery may need intracranial pressure monitoring. Using an intraventricular catheter for the same has the dual advantage of monitoring pressure and allowing drainage of CSF, for countering acute rise in intracranial pressure and in cases of obstructive hydrocephalus due to intraventricular hemorrhage. The intraventricular blood may frequently block the catheter, which can be overcome by using a thrombolytic agent such as 1 mg of tissue plasminogen activator (tPA) given through the catheter, following which the catheter is clamped for 30 min. This can be repeated every 8 hours until the third and the fourth ventricles are cleared of blood on CT or until a maximum cumulative dose of 20 mg rtPA is reached [55]. Ventricular catheters, however, are associated with a higher risk of parenchymal bleeds and infections, and this has to be borne in mind while choosing the type of ICP monitoring. In patients with coagulation disorders, the coagulation should be corrected and if the patient is on antiplatelet drugs, platelet transfusion administered prior to catheter insertion [56].

As the medical management of ICH aims primarily to reduce the intracranial pressure, the tenets of management have been borrowed from the experience gained in treating traumatic brain injury. Mannitol has been the mainstay in the management of raised ICP for a long time, but problems such as rebound phenomenon have led to the increasing use of hypertonic saline (23.4%) for the same purpose [57]. Both the drugs can be used in patients with ICH but the latter may be more effective [58].

Though initial trials with recombinant activated factor VII (rFVIIa) showed promise in limiting the hematoma size following early administration of the drug to patients with intracerebral hemorrhage, this was not borne out in phase three trials [59, 60] Furthermore, the use of rFVIIa has been associated with an increased incidence of thromboembolic events compared

to placebo (7% vs. 2%) and as such the medication is not recommended in noncoagulopathic patients (**Table 4**) [56].

Therapy	Class of evidence
rFVIIa for VKA reversal	III
Prophylactic anticonvulsants	III
Intraventricular drainage in patients with cerebellar hematoma, brainstem compression	III
Early surgery for clot evacuation in a stable patient	IIb

Table 4. Therapies not indicated in ICH.

A vast majority of patients with ICH have fever during the postictal period and the incidence is higher in those with intraventricular hemorrhage. The duration of fever correlates inversely with the patient outcome. Furthermore, cooling the body has been reported to reduce the perihematoma edema, and hence body temperature needs to be controlled with medicines or external cooling after an ICH [61–63].

Almost all patients admitted in the ICU after an intracranial bleed have stress induced hyperglycemia. This may result in loss of control of blood sugars in a diabetic, or high blood sugars in a nondiabetic, both of which are associated with a poor outcome in patients with supratentorial ICH [64]. A study suggested improved clinical outcomes with tight control of blood sugar to the range of 80–110 mg%, but this was found to cause occasional hypoglycemia resulting in increased mortality [65, 66]. As such, no specific target is recommended for blood sugar control in these patients and the broad recommendation that both hypo- and hyperglycemia need to be avoided can probably be met by trying to maintain the blood sugar levels in the range of 120–150 mg%, at least till new evidence is available.

Up to 16% of patients have seizures after an ICH and the incidence is higher in those with lobar bleeds, probably due to the cortical involvement in this cohort of patients. While the incidence of seizures in these patients can be reduced by use of prophylactic anticonvulsants, their use has not been associated with any change in long-term clinical outcome or mortality [67–69]. Though some studies had linked the use of antiseizure drugs, particularly phenytoin, to increased death and disability, probably due to their sedative and cardiovascular side effects, a recent study found no such correlation [70–72]. A study of sodium valproate showed no difference in the rate of new onset seizures in patients given either a placebo (22.2%) or the drug (19.5%) [73]. Therefore, use of prophylactic anticonvulsants is not recommended at present.

The co-occurrence of myocardial infarction and ischemic stroke has been well documented, and Sandhu et al. found that 15% of patients admitted to the ICU who had an elevated troponin I level in the first 24 h, and that is contributed to an increased mortality [74]. A large meta-analysis of stroke patients put the annual risk of MI at 2.2% for these patients [75]. A

preventive protocol including cardiac enzymes, ECG, and echocardiogram should be in place for the management of patients with ICH.

Many patients with intracerebral hemorrhage have an associated coagulopathy or platelet dysfunction, either due to an underlying disease such as factor deficiency or due to the use of anticoagulant/antiplatelet medications. The presence of such a coagulopathy must be identified and corrective measures taken as soon as the patient is admitted, in order to prevent hematoma expansion. The detailed management of patients on oral anticoagulants can be obtained from the guidelines published by the American College of Chest Physicians [76].

Intraventricular hemorrhage associated with ICH has been consistently associated with a worsening of the eventual patient outcome. While an external ventricular drain is useful in draining the blood and treating the obstructive hydrocephalus produced due to obstruction of the ventricular pathway by blood clots, this treatment is not very effective in practice due to the propensity of blood clots to also block the catheter. The use of local fibrinolytic agents was promoted to overcome this problem. The CLEAR-IVH (clot lysis: evaluating accelerated resolution of IVH) trial [77] showed that there was a significant reduction in the incidence of catheter blockage and duration of catheter insertion in patients given intraventricular rtPA. They had a lower incidence of permanent CSF diversion procedures, but also suffered a higher incidence of rehemorrhage [78, 79]. An alternative to the use of intraventricular catheters is the endoscopic clearance of the intraventricular hemorrhage. While studies have not shown any improvement in mortality or neurological outcome, patients who underwent endoscopic clot evacuation have been reported to have a lower requirement of permanent CSF diversion procedures [80].

7. Surgical management

The exact role that surgery plays in the management of ICH remains shrouded in controversy, mainly due to the multitude of factors and the heterogeneity of patients that present with ICH. The largest studies that looked at the benefit of early surgery for patients with supratentorial ICH did not show any benefit compared to medical management, but detailed analysis showed that two subgroups of patients had a better prognosis with surgery. The first were those with lobar hemorrhages within 1 cm of the cortical surface and the second were those who were assigned a poor prognosis at the time of presentation, using a formula devised for the study.

The ideal surgical procedure is also open to question, with the reported procedures including craniotomy and evacuation of the hematoma, decompressive craniectomy, minimally invasive clot evacuation with the use of rtPA (MISTIE II), stereotactic aspiration of the clot and needle aspiration of the basal ganglionic hematoma [81–84].

There is more clarity in cases of infratentorial hematoma, with most surgeons in agreement that pontine hemorrhages are best managed conservatively (**Figure 8**). This is due to the difficulty in surgically accessing the brainstem, the morbidity associated with surgery on the brain stem and the high mortality associated with these hemorrhages. However, it has been noted

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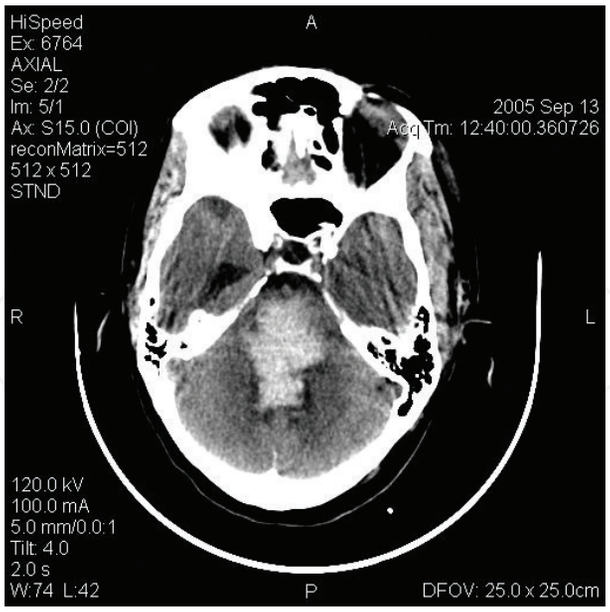

Figure 8. Large hypertensive pontine hemorrhage destroying the entire brainstem.

that patients with bleeding cavernous malformations have a much better outcome than those in whom the cause of hemorrhage is uncontrolled hypertension, and this subgroup may merit surgery [85]. On the other hand, most patients with cerebellar hemorrhages warrant early surgery, especially when the clot volume is more than 3 cm. This is due to the direct compression of brainstem caused by even small infratentorial hemorrhages, due to the small volume of the posterior fossa and the propensity of these bleeds to cause obstructive hydrocephalus. Insertion of external ventricular drain to treat the hydrocephalus may seem like a logical move, but patients thus treated have had a worse prognosis due to the incidence of reverse transtentorial herniation following decompression of the ventricles [86, 87].

While performing a craniotomy for evacuation of ICH, a few points have to be kept in mind [88].

1. Position the patient so that a vertical track from the surface will lead to the hematoma. This will reduce the chances of the surgeon becoming disoriented and missing the hematoma.
2. In putaminal hemorrhages, the shortest track will often involve dissecting the Sylvian and performing the corticectomy in the insula (see **Figure 2**).
3. In deep-seated hematomas, use intraoperative ultrasound or neuronavigation to find the shortest track to the clot to avoid excessive damage to normal brain.
4. When there is a large hematoma abutting the Sylvian fissure, a preoperative angiography (CTA or DSA) should be done to exclude a middle cerebral artery aneurysm (see **Figure 5**).
5. After obtaining hemostasis, increase the blood pressure by 20–30 mm Hg above the baseline and ensure there is no bleeding, so as to reduce the risk of rebleed.
6. In most cases, the bone flap can be safely replaced as the brain will be lax once the hematoma is evacuated.

8. Conclusion

The goal of treatment in patients with ICH is very similar to that in traumatic brain injury, namely, to prevent further damage. This can be achieved by preventing the expansion of the hematoma, reducing ischemic and hypoxic damage, removal of the hematoma, and management of hydrocephalus when appropriate and effective rehabilitation. Primary prevention by controlling hypertension should be a goal of national healthcare programs. Many of the strategies used in the management of these patients is in a state of constant flux since studies on the subject wind up raising more questions than they answer. It is up to the physicians to constantly update themselves on the best management protocols for these patients, and to devote some time to clinical research that will shed more light on this devastating disease.

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