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REVIEW ARTICLE

Emergency Neurological Life Support: Intracerebral Hemorrhage

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Abstract Intracerebral hemorrhage (ICH) is a subset of stroke due to bleeding within the parenchyma of the brain. It is potentially lethal, and survival depends on ensuring an adequate airway, reversal of coagulopathy, and proper diagnosis. ICH was chosen as an Emergency Neurological Life Support protocol because intervention within the first critical hour may improve outcome, and it is critical to have site-specific protocols to drive care quickly and efficiently.

Keywords Intracerebral hemorrhage · Coagulopathy · ICH Score

Introduction

Intracerebral hemorrhage (ICH) results from direct bleeding into the brain. In the US, ICH accounts for 10–15 % of all strokes, but it carries a disproportionately high risk of death or long-term disability. It is considered an acute neurological emergency because of the potential to treat or mitigate injury, and the risk of ongoing secondary brain injury.

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The availability of treatments proven to benefit ICH patients has lagged behind that of ischemic stroke and aneurysmal subarachnoid, and this has resulted in variability in care that ranges from aggressive treatment to a nihilistic approach. Guidelines exist for the management of ICH, and the purpose of this ENLS protocol is to emphasize initial management, with the goal of optimizing recovery. Acknowledging that there is variability in the strength of evidence for treatment recommendations for certain interventions, aggressive initial care of the ICH patient is recommended, in accordance with existing guidelines [1, 2].

Management of the ICH patient during the initial "golden hour" emphasizes the following aspects:

(1) Stabilization and reassessment of the patient's airway, breathing, and circulation (ABCs).
(2) Rapid and accurate diagnosis using neuroimaging.
(3) Concise clinical assessment regarding ICH characteristics and patient condition.
(4) Targeted assessment for potential early interventions including:
    (a) Control of elevated blood pressure.
    (b) Correction of coagulopathy.
    (c) Need for early surgical intervention.
(5) Anticipation of specific patient care needs such as:
    (a) Specific treatment aspects related to underlying ICH cause.
    (b) Risk for early clinical deterioration and hematoma expansion.
    (c) Need for intracranial pressure (ICP) or other neuromonitoring.
    (d) Patient disposition from emergency department (ED).

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The ENLS suggested algorithm for the initial management of ICH is shown in Fig. 1. Suggested items to complete within the first hour of evaluating a patient with ICH are shown in Table 1.

Table 1 Intracerebral hemorrhage checklist for the first hour

Checklist
☐ Complete blood count with platelets, PT, PTT, INR
☐ Head imaging results: hematoma size, location
☐ Glasgow Coma Scale (GCS) score
☐ Calculate ICH Score
Interventions
☐ Coagulopathy reversal
☐ Blood pressure lowering
☐ Surgical hematoma evacuation (if indicated)
☐ Airway/ventilation management

Diagnosis

ICH may result from a variety of underlying etiologies. Rupture of a small arteriole due to chronic hypertension accounts for approximately 60 % of cases. Other common causes include cerebral amyloid angiopathy, coagulopathy due to treatment with antithrombotic medications, sympathomimetic drugs such as cocaine, and underlying vascular anomalies such as arteriovenous malformations (AVMs) or cavernous malformations. Less common causes include cerebral vasculitis, Moya–Moya syndrome, and rupture of a saccular or mycotic aneurysm. Secondary hemorrhagic transformation of an arterial or venous infarct may also occur.

Most patients with acute ICH develop the sudden onset of a focal neurological abnormality. Without neuroimaging, the ICH neurologic syndrome often cannot be reliably distinguished from an acute ischemic stroke. Headache, progressive neurologic signs and symptoms, acute severe hypertension, and decreased level of consciousness occur more frequently in ICH than in ischemic stroke.

The initial prehospital and ED resuscitation is similar across stroke subtypes, with rapid neuroimaging being essential to diagnosis. Because treatments for ICH and acute ischemic stroke are different, ICH-specific interventions are not provided until the diagnosis is made. Thus, prehospital care focuses on management of the ABCs and rapid transport to a designated stroke receiving hospital. Non-contrast computed tomography (CT) is the most commonly used modality given that it can be done quickly, can be used for critically ill patients, and has a very high sensitivity and specificity for acute parenchymal hemorrhage. Magnetic resonance imaging (MRI) may have a similar sensitivity to identify ICH, but logistics related to availability and the clinical condition of the patient limits its use as a primary modality [3, 4].

Interpreting the ICH CT Scan: Location, Volume, and Spot Sign

ICH tends to occur in characteristic locations, with hypertensive ICH most frequently located in the basal ganglia, thalamus, pons (brainstem), and cerebellum. ICH due to cerebral amyloid angiopathy or AVM tends to have

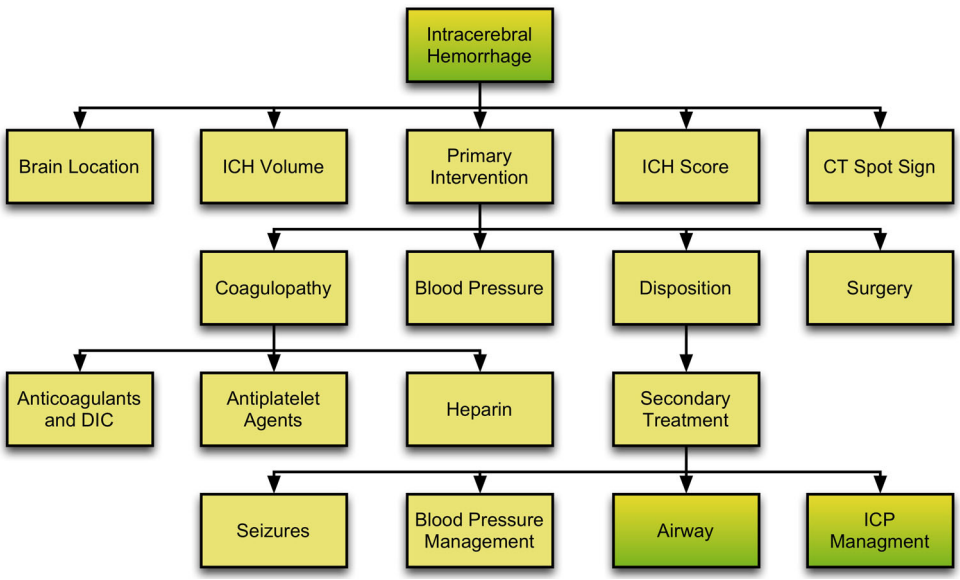

Fig. 1 ENLS intracerebral hemorrhage protocol

a lobar location. The origin of the hematoma is usually evident from the initial CT scan, and its location influences outcome and treatment (Fig. 2). The presence of intraventricular hemorrhage (IVH) also has an impact on outcome and the risk for hydrocephalus.

While ICH location is important, ICH hematoma volume is a stronger predictor of patient outcome. The ability to calculate hematoma volume quickly from the initial CT scan is an advantage in directing communication and treatment decisions. Automated CT software algorithms can be used to calculate hematoma volume. However, the manual ABC/2 formula, which approximates the volume of an ellipsoid, is simple and reasonably accurate compared to computerized methods [5].

When using the ABC/2 method for calculating volume, the axial CT image is selected with the largest cross-sectional area of hemorrhage. Measure the largest hemorrhage diameter (Fig. 3a). Next, perpendicular to this line, measure the largest hemorrhage diameter on the same image (Fig. 3b). Then, multiply the total number of CT slices with hemorrhage by the slice thickness to obtain (c). For (c), if the hematoma area on a slice is approximately 25–75 % of the hematoma area on the reference slice used to determine (a), then this slice is considered half a hemorrhage slice, and if the area is less than 25 % of the reference slice, the slice is not considered a hemorrhage slice [5]. Alternately, (c) can be assessed by measuring the largest diameter, superior to inferior, that is seen on coronal or sagittal images. Multiply (a) times (b) times (c), then divide by 2 in order to obtain the hematoma volume. Figure 3 demonstrates an example.

Many ICH patients experience hematoma growth after initial presentation, and the ability to anticipate expansion is desirable, as expansion is associated with worse clinical outcome [6]. Several retrospective reports have suggested that the use of intravenous (IV) contrast administration during the initial CT scan may identify extravasation into the hematoma and that this "spot sign" (contrast within the hematoma) is predictive of hematoma growth (Fig. 4) [7–9].

Thus, the use of a "stroke CT" that includes non-contrast CT as well as CT angiography (and possibly CT perfusion and post-contrast images) may be considered in patients with acute ICH in order to detect a "spot sign," as well as to reveal an underlying vascular anomaly. Ongoing studies are seeking to use the "spot sign" as a way to identify those at risk for hemorrhage expansion and to determine if hemostatic agents may benefit these specific high risk patients.

Initial Patient Assessment and Primary Intervention: ABCs and the ICH Score

As with all emergency medical care, initial assessment of the ABCs is critical. Until the diagnosis of ICH is made from neuroimaging, overall airway and hemodynamic

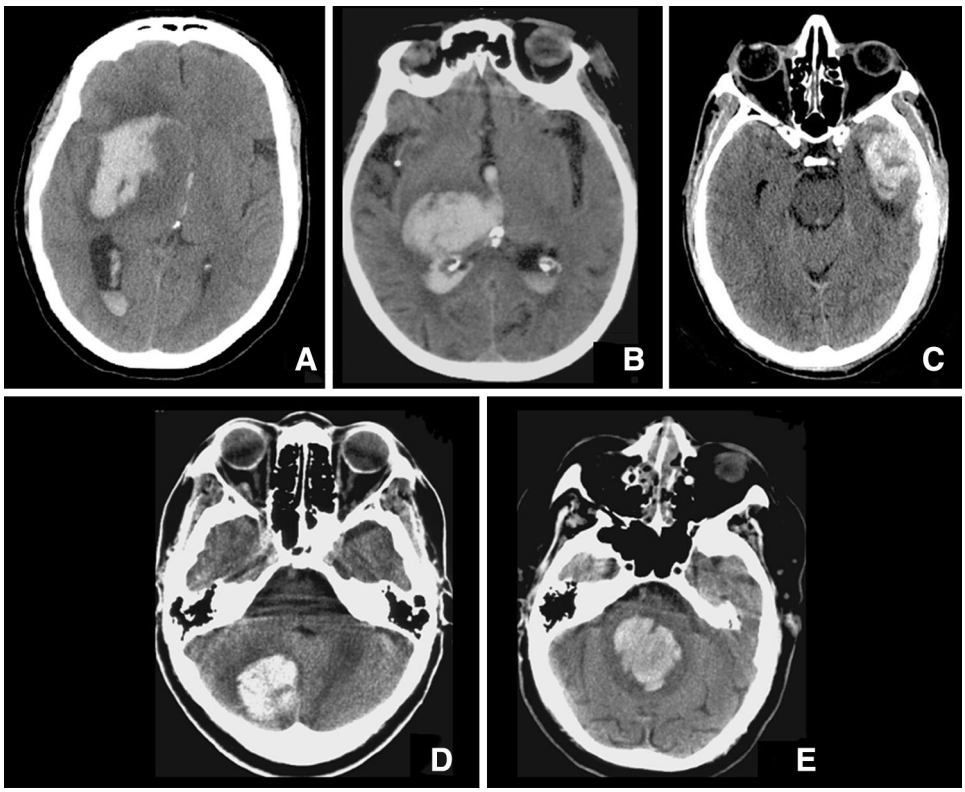

Fig. 2 Typical locations for intracerebral hemorrhage (ICH). ICH due to chronic hypertension is usually due to rupture of small penetrating arterioles and typically occurs in the basal ganglia (**a**), thalamus (**b**), cerebellum (**d**), and pons (**e**). ICH from cerebral amyloid angiopathy and sympathomimetic drugs of abuse such as cocaine or methamphetamine often occurs in lobar regions such as the temporal lobe (**c**). Supratentorial ICH would be considered as basal ganglia, thalamic, or lobar (**a**–**c**), whereas ICH originating in the cerebellum or pons would be considered infratentorial (**d**–**e**). **a**, **b**, and **e** also demonstrate IVH

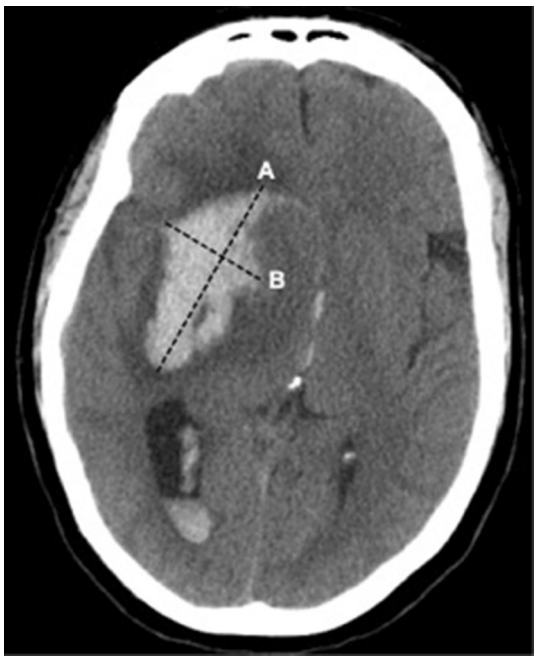

Fig. 3 ABC/2 method for estimating ICH hematoma volume [5]. Right basal ganglia intracerebral hemorrhage. The axial CT image with the largest cross sectional area of hemorrhage is selected. In this example, the largest diameter **a** is 6 cm, the largest diameter perpendicular to (**a**) on the same image **b** is 3 cm, and hemorrhage is seen on 6 slices of 0.5 cm (5 mm) thickness for a (**c**) of 3 cm (not shown). Thus, the hematoma volume is $(6 \times 3 \times 3)/2 = 27$ cc. Note that for (**c**), if the hematoma area on a slice is approximately 25–75 % of the hematoma area on the reference slice used to determine (**a**), then this slice is considered half a hemorrhage slice, and if the area is <25 % of the reference slice, the slice is not considered a hemorrhage slice

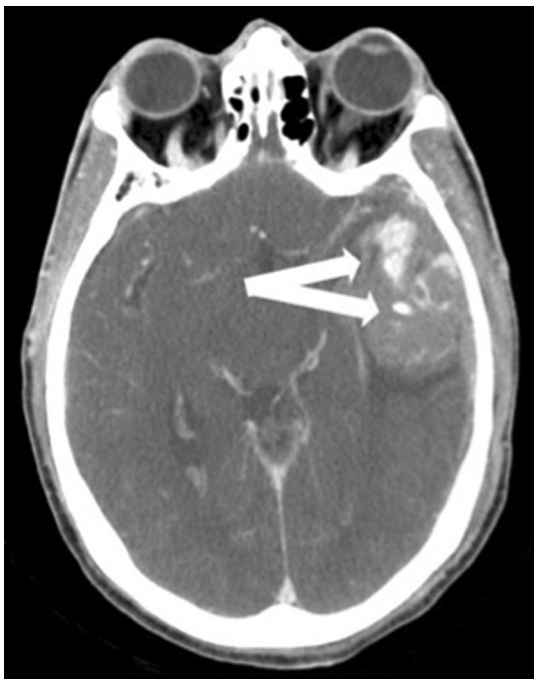

Fig. 4 Contrast extravasation ("Spot Sign") in acute ICH. In this post-contrast image obtained after administration of IV contrast during a "code stroke" CT (non-contrast study, CT angiogram, CT perfusion study), contrast extravasation is present in this acute *left temporal lobe* ICH. This finding is commonly referred to as a "spot sign" (*arrows*) and is associated with increased risk of hematoma expansion

management proceeds in a common pathway with other stroke subtypes. However, immediately following the ICH diagnosis, disease-specific treatment can be instituted.

Because many ICH patients are obtunded or comatose, airway management (specifically the need for intubation for airway protection) should be considered throughout the early treatment course. Thus, while "Airway" is listed under secondary treatment in the ENLS ICH protocol (Fig. 1), it is concurrent with the initial evaluation. In general, if an ICH patient is comatose, rapid sequence intubation (RSI) should be undertaken, with a goal of normoventilation (see the ENLS *Airway, Ventilation, and Sedation protocol)*.

An initial clinical assessment of the patient's condition and stroke severity is essential to rapid treatment planning and communication among providers. While performance of a complete, detailed neurological examination is ideal, much information can be gleaned from a quick assessment using existing clinical grading scales. The ICH Score is the most commonly used validated clinical grading scale for patients with ICH, combining elements related to patient demographics, clinical condition, and neuroimaging findings that are readily available at the time of hospital admission [10, 11]. Several other useful clinical grading scales are also available [12–14].

Components of the ICH Score include age, initial Glasgow Coma Scale (GCS) score, ICH hematoma volume, ICH hematoma location (supratentorial or infratentorial), and presence of IVH. Table 2 demonstrates the components of the ICH Score, with the full score being the sum of points given for each component. Each point increase in the ICH Score is associated with an increased risk of mortality and a decreased likelihood of good functional outcome. The ICH Score is best used as a communication tool among providers and with patients or family members regarding a patient's condition rather than as a tool to precisely prognosticate outcome. While it is tempting to utilize clinical grading scales to triage severely impaired patients toward less-aggressive intervention, this approach is not recommended. Rather, in general, initial aggressive therapy is recommended in order to avoid the potential for a self-fulfilling prophecy of poor outcome in the context of early care limitations [1, 15, 16].

Table 2 The ICH Score [10]

Component	ICH Score points
Glasgow Coma Scale	
3–4	2
5–12	1
13–15	0
ICH volume (cc)	
≥30	1
<30	0
Presence of IVH	
Yes	1
No	0
Infratentorial origin of ICH	
Yes	1
No	0
Age (years)	
≥80	1
<80	0
Total ICH Score	0–6

Primary Intervention: Blood Pressure, Coagulopathy, and Surgery

Following the diagnosis of ICH, immediate consideration should be given to the need for (a) acute control of elevated blood pressure, (b) correction of coagulopathy due to medications or underlying medical conditions, and (c) the need for urgent surgical hematoma evacuation. These are common themes that should form part of the initial ICH evaluation and treatment plan. Decisions regarding these interventions will influence the succeeding aspects of ICH care, such as disposition from the ED, planning for repeat imaging, and need for ICP monitoring or continuous electroencephalography (cEEG).

Hematoma expansion is common in patients with acute ICH, and this is associated with worsened outcomes [6, 17]. Though the pathophysiology that leads to hematoma expansion is incompletely understood, it tends to occur early (within a few hours of onset) and coagulopathy increases the frequency of its occurrence and its extent [18]. However, hematoma expansion is common even in patients without coagulopathy or who are not receiving antithrombotic medications. Thus, intervention to address treatable aspects should not be delayed pending patient disposition.

Blood Pressure

Elevated blood pressure is extremely common in patients with acute ICH. While it seems intuitive that elevated blood pressure may predispose to hematoma expansion due to increased bleeding or to elevated ICP from worsening edema, clinical studies have had conflicting results regarding the impact of acutely elevated blood pressure and the value of acutely lowering the blood pressure [19, 20]. There has been a concern that acutely lowering blood pressure could lead to ischemic brain injury in the peri-hematoma region, but this risk has not been supported by recent studies [21, 22].

While blood pressure management has remained controversial, current approaches favor rapid lowering of moderately elevated blood pressures [1, 2]. Two pilot randomized clinical trials, INTERACT and ATACH, suggested that acutely lowering systolic blood pressure to below 140 mmHg is safe [23, 24]. INTERACT2 was a phase III clinical trial of acute blood pressure lowering in ICH patients presenting with a systolic blood pressure between 150 and 220 mmHg [25]. Patients were randomized to two different blood pressure thresholds: a standard threshold of <180 mmHg and an intensive threshold of less than 140 mmHg. Patients in the intensive arm had modestly better outcomes with about 4 % fewer patients having death or severe disability (defined as a modified Rankin Scale score of 3–6). Interestingly, there was no difference in hematoma expansion between groups.

None of the current guidelines recommend allowing blood pressure to remain extremely elevated without treatment [1, 2]. The current American Heart Association/American Stroke Association Guidelines for the Management of ICH and the guidelines from the European Stroke Organization recommend a target blood pressure of less than 140 mmHg in patients like those studied in INTERACT2 [1, 2]. Acute lowering of blood pressure is reasonable in patients presenting with more extreme levels of hypertension, but less is known about the specific safety and efficacy of treatment [1].

Basic principles of blood pressure lowering in ICH are that management should be initiated immediately and a titratable agent should be used to ensure that the target value is reached quickly and with minimal potential for overshoot. IV beta-blockers and calcium-channel blockers are the most commonly used medications for this indication in the ED and the intensive care unit (ICU).

Labetalol is rapid acting, has mixed alpha and beta adrenergic antagonism, and is commonly used in the ED in an initial IV bolus dose of 5–20 mg. Nicardipine is a calcium channel blocker of the dihydropyridine family that is more selective for vascular smooth muscle. A common initial nicardipine dose of 5 mg/h as continuous IV infusion is often used, with titration up every 5–15 min as needed, up to a maximum of 15 mg/h. Clevidipine is another calcium-channel blocker that acts even more rapidly than nicardipine. If possible, nitroprusside should be avoided due to its potential for cerebral vasodilation,

disturbed cerebral autoregulation, and elevated ICP. ICU admission is recommended, due to the close monitoring and frequent medication changes required to lower blood pressure.

Coagulopathy: Anticoagulants, Antiplatelet Agents, and Heparin

The use of antithrombotic medications for prevention and treatment of ischemic stroke, cardiovascular disease, and systemic venous thromboembolism is common and is increasing as the population ages. Antithrombotic medications are a risk factor for the occurrence of ICH, as well as for hematoma expansion if an ICH occurs. Given the range of antithrombotic medications, including warfarin, heparin, antiplatelet agents such as aspirin and clopidogrel, and newer agents such as dabigatran, rivaroxaban, apixaban, and edoxaban, the specific risks and interventions to reverse coagulopathy vary. Additionally, coagulopathies may be due to underlying medical conditions, such as liver disease or hematologic malignances.

The second focus in ICH is on treatment of coagulopathy. Reversal of coagulopathy is discussed in the ENLS Pharmacotherapy manuscript with dosing recommendations, but will also be reviewed here in the specific context of ICH. As part of the initial evaluation of the ICH patient, a medical history and medication list should be obtained from the patient, family, prehospital providers, or medical record; specifically the use of antithrombotic medication and, if possible, when the last dose was taken should be noted. Urgent laboratory tests should include a complete blood count (CBC) with platelet count, PT, an international normalized ratio (INR), and a partial thromboplastin time (PTT). A general principle is that any ICH occurring in a patient on antithrombotic medications should be considered life-threatening due to the risk of hematoma expansion. Interventions to treat coagulopathy are based on this history and laboratory information more than on size or location of the hematoma or ICH Score.

Patients taking warfarin and whose INR is >1.4 should receive agents to normalize the INR. Options to reverse warfarin therapy include the administration of fresh frozen plasma (FFP), vitamin K, prothrombin complex concentrates (PCC), and the hemostatic agent recombinant factor VIIa (rFVIIa). The most important principle is to reduce the INR as soon as possible, ideally within minutes.

While FFP is widely used for reversing the effect of warfarin, it may not be optimal in particular medical conditions. FFP contains factors I (fibrinogen), II, V, VII, IX, X, XI, XIII, and antithrombin. Fairly large volumes of FFP (10–15 ml/kg) are often required for full reversal of anticoagulation, and this places patients at risk for volume overload and pulmonary edema [26]. FFP, like other blood products, also carries a risk for transfusion related events and requires thawing after cross-matching by a blood bank.

PCCs contain factors II, IX, X (and varying amounts of VII, depending on the specific preparation) with much higher concentrations of clotting factors in smaller amounts of volume than FFP. PCCs can correct the INR within minutes, faster than FFP, and with fewer cardiopulmonary complications [27]. However, in a study comparing PCC and FFP, there was no difference in hematoma growth in patients whose INR was corrected within 2 h [28]. This suggests that the timing of coagulopathy reversal, not the specific agent, makes the greatest impact.

Table 5 of the ENLS Pharmacotherapy manuscript details the PCC dosing based on current INR, and the dosing of FFP if PCC is not available. Current guidelines [1, 29] recommend the use of vitamin K 5–10 mg administered intravenously by slow IV infusion, in conjunction with another more rapidly acting agent (e.g., FFP, PCC), as it typically takes hours after vitamin K administration for reversal of warfarin-induced coagulopathy, but it has a more long-lasting effect than PCC or FFP [30].

While rFVIIa also quickly reverses an elevated INR, this may reflect a specific effect on the INR laboratory test and a clinically important coagulopathy may remain. The rFVIIa has been shown to decrease hematoma growth in non-coagulopathic ICH patients, but this did not translate into improved clinical outcome [32]. Thus, rFVIIa is not recommended for use in ICH patients with or without warfarin-related coagulopathy; however, it is occasionally used in patients with coagulopathy related to liver failure.

Studies vary regarding the impact of concurrent antiplatelet therapy on hematoma expansion and outcome for patients presenting with ICH, though increased risk of hematoma growth while on these agents is suggested [33–36]. There is heterogeneity in clinical practice, ranging from the empiric use of platelet transfusions, to determining the need for transfusion by laboratory tests for platelet function, to complete the avoidance of platelet treatment. Pending definitive data, transfusion of platelets for patients on acetylsalicylic acid (ASA), clopidogrel, or other antiplatelet agents, as well as adding desmopressin (DDAVP, which promotes the release of von Willebrand factor) for patients on clopidogrel, may be considered.

Newer anticoagulants, such as direct thrombin inhibitors (e.g., dabigatran) or direct Xa inhibitors (e.g., rivaroxaban, apixaban, and edoxaban), currently do not have specific reversal agents available, and experience with ICH in patients taking these medications is limited. There is some suggestion that PCCs may have limited effectiveness in reversing the effect of rivaroxaban and apixaban, but not of dabigatran [37]. The use of rFVIIa or FEIBA (factor VIII inhibitor bypassing activity) in ICH patients on dabigatran

may have potential [38, 39]. It should be noted that additional laboratory tests, such as endogenous thrombin potential and thrombin clotting time, may have some value in assessing the activity of these newer agents. Activated charcoal can be used if the most recent dose was within about 2 h and hemodialysis could be considered in patients on dabigatran [40]. Vitamin K is of no value and FFP is of unclear utility. Specific antidotes for these medications are in development [41].

Unfractionated heparin (UFH) is used for many medical conditions, including acute coronary syndromes, pulmonary embolism, and endovascular surgery, as well as for maintaining the patency of indwelling catheters. Heparin binds to and activates antithrombin III, thus inactivating thrombin and favoring thrombolysis. The reversal agent for UFH is protamine sulfate, administered 1 mg for every 100 U of UFH IV received in the prior 2 h, with a maximum dose of 50 mg [42]. Protamine sulfate binds to and inactivates heparin, allowing it to be broken down by the reticuloendothelial system. Given the short half-life of UFH, reversal is likely unnecessary if the last dose was received greater than 4 h prior to ICH onset. Protamine sulfate can also be used in an attempt to reverse the effect of low-molecular weight heparin that was given within the prior 8 h. However, this reversal may be incomplete.

Surgical Hematoma Evacuation

Though most patients with acute ICH do not require surgery for removal of the hematoma, it is worthwhile to address the option of surgery immediately after ICH diagnosis, since the theoretical benefits of surgery include prevention of brain herniation, improvement in elevated ICP, and removal of blood and blood degradation products that may produce cytotoxic secondary brain injury.

After decades of ambiguity, the effects of surgical evacuation were addressed in the Surgical Trial in Intracerebral Hemorrhage (STICH) that found early surgical evacuation of a supratentorial ICH was not harmful, but there was no difference in long-term mortality or functional outcome [43]. Because the subgroup of patients in STICH with lobar ICH within 1 cm of the cortical surface may have benefited from surgical evacuation, the STICH II clinical trial was undertaken for this group of patients [44]. However, STICH II did not demonstrate a significant benefit to early hematoma evacuation in these patients either. Minimally invasive techniques, including endoscopic hematoma aspiration or instillation of a thrombolytic such as urokinase or recombinant tissue plasminogen activator into the hematoma with aspiration of contents, are also being studied [45–47]. At present, routine removal of supratentorial hematoma cannot be endorsed, but it is still undertaken as a life-saving measure in selected patients.

In contrast, several case series suggest that patients with cerebellar ICH >3 cm in diameter or with compression of the brain stem or hydrocephalus may benefit from surgical hematoma evacuation [48, 49]. There has not been a randomized trial of cerebellar hematoma evacuation analogous to STICH, but it is not clear there is equipoise to justify such a trial.

Current American Heart Association ICH guidelines recommend that patients with cerebellar hemorrhage who are deteriorating neurologically or have brainstem compression should undergo surgical removal of the hemorrhage as soon as possible. Initial treatment of these patients with ventricular drainage alone rather than surgical evacuation is not recommended [1]. Supratentorial hematoma evacuation or decompressive hemicraniectomy might be considered as a life-saving measure in deteriorating patients. Correction of coagulopathy is critical in patients undergoing surgical hematoma evacuation.

Secondary Intervention: Hospital Admission, ICP Management, and Seizures

Ideally, patients with acute ICH should be admitted to an ICU based on the need for close monitoring of neurological and hemodynamic condition and the risk for early deterioration from hematoma expansion, cerebral edema, hydrocephalus, or airway compromise. Admission to a neurological ICU has been associated with improved outcomes compared with admission to a non-neurological ICU [50]. Acknowledging that certain patients will require transfer between hospitals for neurological intensive care management, neurosurgical intervention, or neurointerventional capabilities, all aspects of ICH primary intervention can and should take place without delay in the initial presenting hospital.

Specifically, correction of coagulopathy with appropriate agents, blood pressure control, and treatment of acute seizures should be initiated in the ED of the presenting hospital and not deferred until after transfer. It is critical that the above-discussed aspects of acute ICH evaluation and treatment are initiated at the time of original diagnosis and that transitions in care are smooth from ED to ICU (or operating room, interventional radiology, or comprehensive stroke center). The use of standardized ICH checklists (Table 3) is encouraged.

While this ENLS ICH protocol is principally concerned with the initial evaluation and treatment period, it is important to anticipate the health care needs of the following 24–72 h as part of care planning. The first 24 h are critical for blood pressure management, identification of seizures, ICP management, and maintaining a secure airway. Avoidance of

Table 3 Standardized ICH Algorithm

Prehospital care
☐ ABCs
☐ Determine time of onset and circumstances
☐ Perform prehospital stroke screen
☐ Finger stick for glucose
☐ Brief medical history and medication list
☐ Triage to stroke center
☐ Perform prehospital notification of pending stroke patient
ED care
☐ Emergent triage to high acuity area
☐ Perform primary assessment—ABCs
☐ Perform focused neurologic exam (GCS, NIHSS)
☐ Obtain baseline screening labs (CBC and platelet count, electrolytes, PT/INR and PTT, glucose)
☐ Obtain cerebrovascular imaging as soon as possible (non-con CT, stroke CT/CTA/CTP, or MRI)
☐ Obtain brief medical history and medication list
After confirmation of ICH
☐ Reassess ABCs (consider intubation if comatose)
☐ Initiate blood pressure intervention (target SBP <140 mmHg)
☐ Quantify ICH volume (ABC/2 calculation)
☐ Perform ICH Score (0–6)
☐ Begin correction of anticoagulation as required
☐ Correction of antiplatelet agents as required
☐ Consult neurosurgery for potential hematoma evacuation or ICP monitor placement
☐ Admit to (neuro) ICU (may require transfer to another hospital)
In-hospital setting
☐ Continue to reassess ABCs
☐ Continue neurologic reassessment
☐ ICP monitor and/or ventriculostomy for treatment of elevated ICP or hydrocephalus
☐ Continue management of blood pressure
☐ Place arterial blood pressure catheter as needed
☐ Place central venous catheter as needed
☐ Urine toxicology screen (if not already done)
☐ Foley catheter (needed for most ICH patients early)
☐ Feeding tube (goal to begin feeding within first day)
☐ DVT prophylaxis with sequential compression devices (consider heparin/LWMH within the first 3 days)
☐ Recheck PT/INR and PTT (or specific labs for other oral anticoagulants) if patient was coagulopathic and receiving reversal agents
☐ No anticonvulsant prophylaxis; treat clinical seizures; continuous EEG if level of consciousness impaired out of proportion to ICH or IVH
☐ Consider need for repeat head CT
☐ Consider need for catheter cerebral angiography

fever, hyperglycemia/hypoglycemia, and hypoxia are also important, as these may impact outcomes [1, 51, 52]. In addition, patients with ICH are at increased risk for the development of deep venous thrombosis (DVT); current guidelines recommend use of intermittent pneumatic compression devices at hospital admission, as well as initiation of prophylaxis-dose unfractionated or low-molecular weight heparin within 1–4 days following onset (assuming cessation of bleeding) [1].

The incidence and impact of elevated ICP in ICH has received limited study, but it is undoubtedly a factor in management [53–56]. Patients with IVH are at risk for hydrocephalus and elevated ICP. Current guidelines for ICP monitoring in ICH follow the approach in severe traumatic

brain injury, with ICP monitoring recommended in patients with GCS $\leq$ 8, large hematomas with mass effect suggestive of elevated ICP, or hydrocephalus. As a goal, an ICP $<$20 mmHg should be maintained, with a minimal CPP of 60 mmHg, adjusted based on an individual patient's cerebral autoregulation status [1]. Ventricular catheters are beneficial in their ability to both measure ICP and drain cerebrospinal fluid (CSF); therefore, they should be used in patients with hydrocephalus. In contrast, intraparenchymal fiberoptic monitors have a lower risk of hemorrhage and infection, but cannot be used to drain CSF. Correction of coagulopathy prior to ICP monitor insertion is desirable.

While seizures may occur in ICH patients, their incidence and impact on outcome have varied across studies [57, 58]. In a single study, prophylactic anticonvulsants reduced seizure occurrence in lobar ICH [58]. However, two more recent studies found worse functional outcomes in patients routinely given prophylactic anticonvulsants (primarily phenytoin) [59, 60]. While comatose ICH patients may have a high risk (approximately 20 %) of non-convulsive seizures, the impact of prophylactic anticonvulsants on their occurrence is also unclear [61, 62]. Current guidelines do not recommend routine use of prophylactic anticonvulsants [1], though some practitioners still use a short course in patients with lobar ICH and those undergoing surgical hematoma evacuation. Clinical seizures should be treated, and continuous EEG monitoring should be performed in patients with inadequately explained decreased level of consciousness.

Pediatric Considerations

Since chronic hypertension and chronic anticoagulation therapy are less common in children, ICH is seen with much less frequency in pediatric patients. However, children may present with life threatening ICH due to arteriovenous malformations, other intracranial vascular anomalies, sickle cell disease, or cerebral venous thrombosis.

While $<$2 % of cerebral aneurysms are found in pediatric patients, as many as 24 % of children with intracranial aneurysms may have ICH at the time of their initial presentation [63]. For children with significant ICH, the same emergent care principles described earlier in these chapters apply regarding the need for establishment of the airway, provision of adequate oxygenation, and management of blood pressure (see pediatric section in Airway, Ventilation and Sedation Chapter). Hypotension is defined as systolic blood pressure (SBP) below the 5th percentile for age (SBP 5th percentile = 70 mmHg + age in years $\times$ 2). Careful attention should also be given to the detection and treatment of seizures, which may be present in as many as 21 % of children with intracranial aneurysms [64], and treatment of ICH in need of emergent surgical evacuation. In children with SAH, admission to a center with expertise in diagnosing and treating vasospasm is indicated, as cerebral vasospasm may be seen in as many as 67 % of children with SAH [65]. The diagnosis of cerebral vasospasm is particularly challenging in children given that cerebral blood flow velocity is age and gender dependent [66, 67]. When clinically indicated, cerebral angiography has similar complication rates compared to those reported in adults, even in children younger than 3 years of age [68].

There are no established parameters for treatment of hypertension in children with ICH, but a SBP threshold $<$140 mmHg is reasonable in older children. Nicardipine is well tolerated and the recommended initial dose is 0.5 mcg/(kg min), titrated by 0.5 mcg/(kg min) every 15 min to a maximum of 5 mcg/(kg min). In older children (adult weight) the initial dose is 2.5 mg/h, with titration by 2.5 mg/h every 15 min up to a maximum of 15 mg/h. Esmolol is a reasonable alternative and generally well tolerated. Finally, while anticoagulation therapy is less common in children, pediatric patients with ICH may present with coagulation abnormalities that require careful evaluation and treatment to prevent hematoma expansion and facilitate surgical therapy.

Communication

When communicating to an accepting or referring physician about a patient with ICH, consider including the key elements listed in Table 4.

Table 4 Intracerebral hemorrhage communication regarding assessment and referral

Communication
☐ Age
☐ Hematoma volume and location
☐ GCS
☐ ICH Score
☐ Hydrocephalus present?
☐ Blood pressure
☐ Coagulation parameters and reversal treatment
☐ Plan for surgery

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