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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 resulting from bleeding within the brain 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 (ENLS) protocol because intervention within the first critical hour may improve outcome, and it is helpful to have a protocol to drive care quickly and efficiently.

Keywords: Hypertensive hemorrhage · Secondary brain injury · Coagulopathy · Protocol

Introduction

Intracerebral hemorrhage (ICH) results from direct bleeding into the brain. 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.

ICH remains without an approved acute treatment of proven benefit, 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. Rapid and accurate diagnosis using neuroimaging
2. Concise clinical assessment regarding ICH characteristics and patient condition
3. Targeted assessment for potential early interventions including:
   a. Control of elevated blood pressure
   b. Correction of coagulopathy
   c. Need for early surgical intervention
4. Anticipation of specific patient care needs such as:
   a. Risk for early clinical deterioration and hematoma expansion
   b. Need for intracranial pressure (ICP) or other neuromonitoring
   c. Patient disposition from emergency department (ED)
   d. Specific treatment aspects related to underlying ICH cause

The ENLS-suggested algorithm for the initial management of ICH is shown in Fig. 1. Suggested items to

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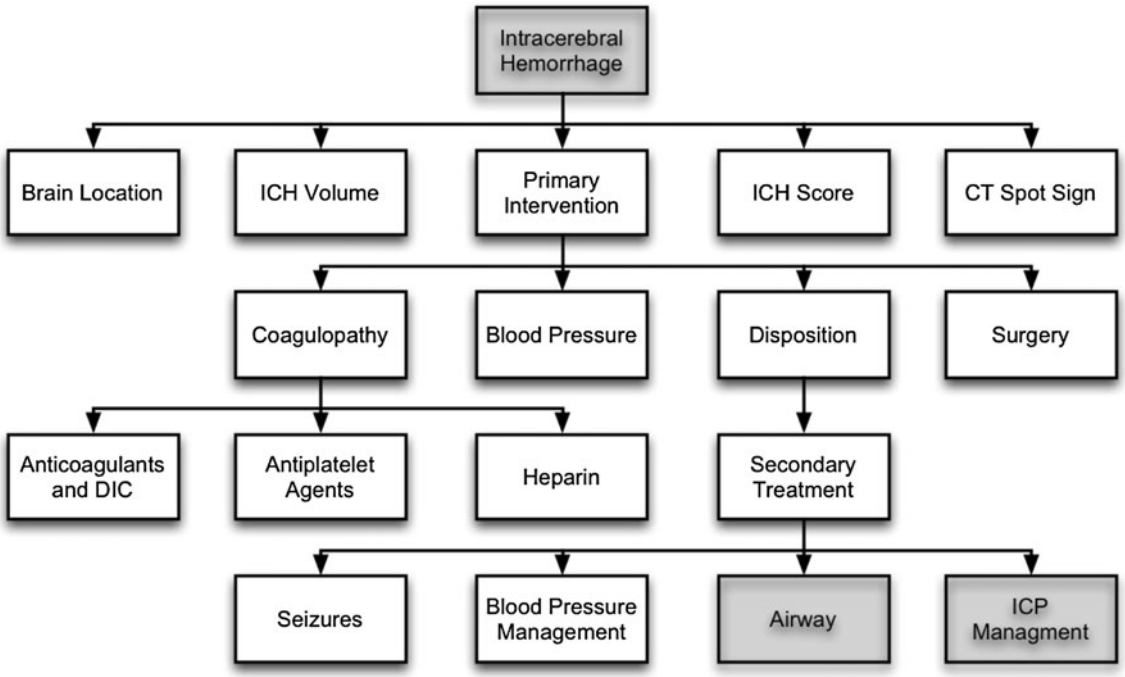

Fig. 1 ENLS intracerebral hemorrhage protocol

complete within the first hour of evaluating a patient with ICH are shown in Table 1.

Diagnosis

ICH may result from a variety of underlying etiologies. Rupture of a small arteriole because of chronic hypertension accounts for approximately 60 % of cases. Other common causes include cerebral amyloid angiopathy, coagulopathy because of 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 berry 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 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. 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 limit 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 because of cerebral amyloid angiopathy or AVM tends to have a lobar location. The origin of the hematoma is usually evident from the initial CT scan, and its location determines outcome and treatment (Fig. 2). The presence of intraventricular hemorrhage (IVH) also has an impact on outcome and the risk for hydrocephalus.

In addition to location, ICH hematoma volume is the strongest predictor of patient outcome. The ability to

Table 1 Intracerebral hemorrhage checklist for the first hour

Intracerebral hemorrhage checklist for the first hour
☐ Blood pressure
☐ PT, PTT, Platelets, INR
☐ Head CT: size of hemorrahge
☐ GCS
☐ ICH Score

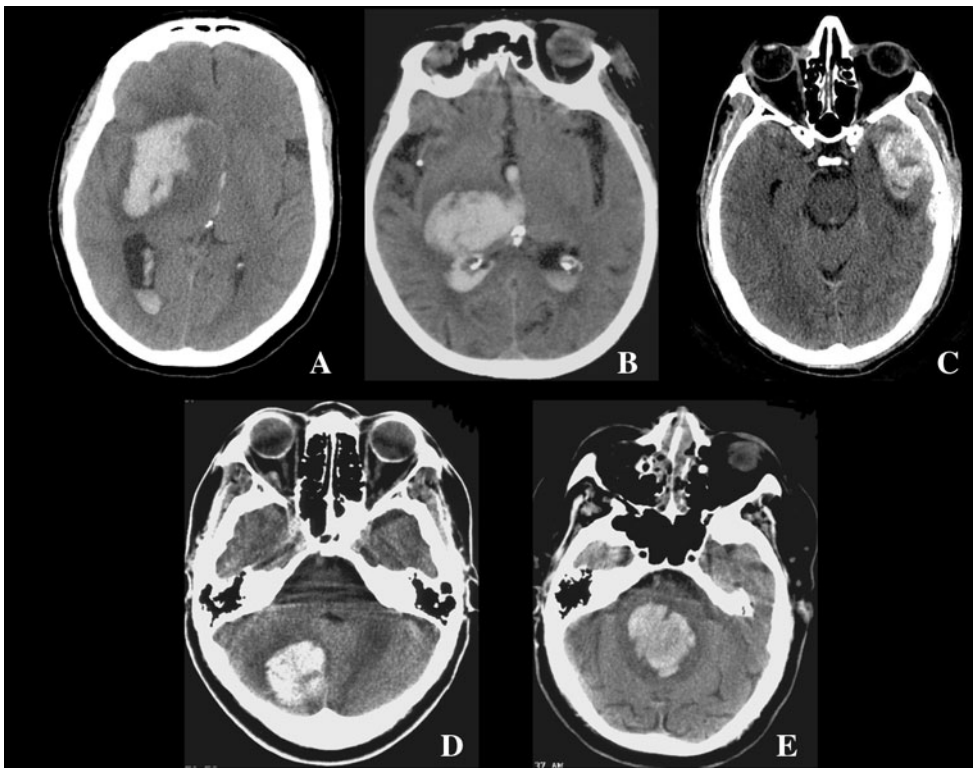

Fig. 2 Typical locations for intracerebral hemorrhage (ICH). ICH because of chronic hypertension is usually because of rupture of small penetrating arterioles and typically occurs in the basal ganglia (**a**), thalamus (**b**), cerebellum (**d**), and pons (**e**). ICH from cerebral amyloid angiopathy 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

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 (A). Next, perpendicular to this line, measure the largest hemorrhage diameter on the same image (B). 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 <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 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 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 at risk patients.

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

As with all emergency medical care, initial assessment of airway, breathing, and circulation is critical. Until the diagnosis of ICH is made from neuroimaging, overall airway and hemodynamic 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 protocol (Fig. 1), it is concurrent with the initial evaluation. In general, if an ICH patient is comatose, rapid sequence intubation (RSI)

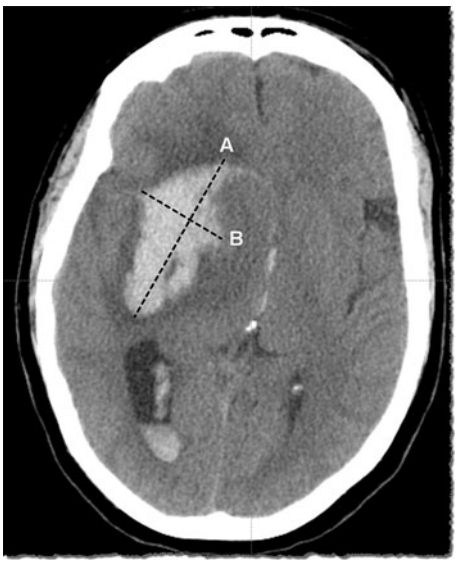

Fig. 3 ABC/2 method for estimating ICH hematoma volume [5]. Right basal ganglia intracerebral hemorrhage. The axial CT image with the largest cross sectional area of hemorrhage is selected. In this example, the largest diameter (**a**) is 6 cm, the largest diameter perpendicular to **a** on the same image (**b**) is 3 cm, and hemorrhage is seen on six 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

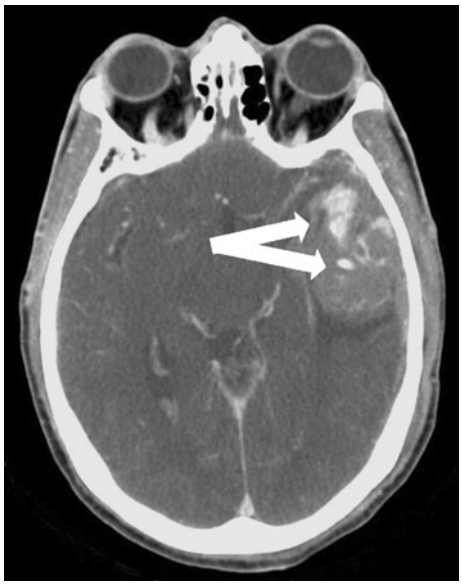

Fig. 4 Contrast extravasation ("spot sign") in acute ICH. In this post-contrast image obtained after administration of IV contrast during a stroke CT (non-contrast study, CT angiogram, CT perfusion study), contrast extravasation is present in this acute left temporal lobe ICH. This is commonly referred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

should be undertaken, with a goal of normoventilation (see the *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, 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 (30-day mortality rates with ICH Scores of 1, 2, 3, and 4 were 13, 26, 72, and 97 %, respectively). However, the ICH score is best used as a communication tool among providers and with patients or family members regarding a patient's condition. 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

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

recommended to avoid the potential for a self-fulfilling prophecy of poor outcome in the context of early care limitations [1, 15, 16].

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 because of 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 additional monitoring of ICP or use of continuous electroencephalography (cEEG).

Hematoma expansion is common in patients with acute ICH, and this is associated with worse outcomes [6, 17]. Although 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 expansion of the hematoma because of increased bleeding or to elevated ICP from worsening edema, data from clinical studies are conflicted regarding the impact of acutely elevated blood pressure and the value of 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 modestly lowering extremely elevated blood pressures [1, 2]. Two pilot randomized clinical trials, INTERACT and ATACH, have suggested that acutely lowering systolic blood pressure to $<$140 mmHg is safe [23, 24]. INTERACT2 and ATACH2 are ongoing phase III randomized clinical trials that are assessing the efficacy of mild therapeutic blood pressure reduction on clinical outcome and hematoma expansion. The precise value to which blood pressure should be lowered is unclear.

None of the current guidelines recommend allowing blood pressure to remain extremely elevated without treatment [1, 2]. Pending the results of ongoing clinical trials, the use of blood pressure targets from the American Heart Association/American Stroke Association Guidelines for the Management of Intracerebral Hemorrhage is recommended [1]. These guidelines suggest reducing the blood pressure to $<$160/90 mmHg or a mean arterial pressure (MAP) $<$110 mmHg. In patients with potential for elevated ICP, a cerebral perfusion pressure (CPP) of $\geq$60 mmHg should be maintained.

Basic principles of blood pressure lowering in ICH are that management should be immediately initiated 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 bolus dose of 5–20 mg. Nicardipine is a calcium channel blocker of the dihydropyridine family that is more selective for coronary and cerebral vascular beds. A common initial nicardipine dose of 5 mg/h is often used, with titration up every 15 min as needed. Clevidipine is another calcium channel blocker that acts even more rapidly than nicardipine. If possible, nitroprusside should be avoided because of its potential for cerebral vasodilation, disturbed cerebral autoregulation, and elevated ICP. ICU admission is recommended, because of the close monitoring and frequent medication changes required to safely 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 and rivaroxaban, the specific risks and interventions to reverse coagulopathy vary. Additionally, coagulopathies may be because of underlying medical conditions, such as liver disease or hematologic malignances.

The second focus in ICH is on treatment of coagulopathy. As a 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, 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 because of 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 clinical scores.

Patients taking warfarin and whose INR is >1.4 should receive agents to normalize the INR. Options 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 normalize 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 other 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 [25]. 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. Several studies have shown that PCCs can correct the INR within minutes. However, in a study comparing PCC and FFP, there was no difference in hematoma growth in patients whose INR was corrected within 2 h [26]. This suggests that the timing of coagulopathy reversal, not the specific agent, makes the greatest impact.

Table 3 describes INR-based dosing for PCC (or FFP if PCC is not available). Some centers use a fixed dose of 25–50 international units (IU)/kg of PCC as an alternative to calculating an INR-based dose. Current guidelines [27] recommend the use of vitamin K 5–10 mg administered intravenously by slow push, 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 [28].

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. rFVIIa has been shown to decrease hematoma growth in non-coagulopathic ICH patients, but this did not translate into improved clinical outcome [30]. 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.

Table 3 Calculating volume of plasma or IU of PCC for warfarin reversal [29]

1. Decide on target INR. Consider goal INR <1.5.
2. Convert INR to % functional prothrombin complex (PC) expressed as % of normal plasma:
   - INR $>5 = 5$ %
   - INR 4.0–4.9 = 10 %
   - INR 2.6–3.2 = 15 %
   - INR 2.2–2.5 = 20 %
   - INR 1.9–2.1 = 25 %
   - INR 1.7–1.8 = 30 %
   - INR 1.4–1.6 = 40 %
   - INR 1.0 = 100 %
3. Calculate dose:
   (Target in % PC − Current level in % PC) × kg = mL of FFP or IU of PCC needed

Example. Patient with pulmonary embolism 3 months ago now has warfarin-associated ICH: present INR = 7.5, target INR = 1.4, body weight = 80 kg: (40 − 5) × 80 = 2,800. Therefore, necessary dose is 2,800 mL of FFP or 2,800 IU of PCC

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 [31–35]. 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 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 desomopressin (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), 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 but not of dabigatran [36]. The use of rFVIIa in ICH patients on dabigatran has theoretical potential [37]. 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.

Unfractionated heparin 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 heparin is protamine sulfate, administered 1 mg for every 100 units of heparin received in the prior 2 h, with a maximum dose of 50 mg [38]. Protamine sulfate binds to and inactivates heparin, allowing it to be broken down by the reticuloendothelial system. Given the short half-life of heparin, reversal is likely unnecessary if the last dose was received 4 h prior to ICH onset. Protamine sulfate can also be used in the same dose 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

Although 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, because the theoretical benefits of surgery include prevention of brain herniation, improvement in elevated ICP, and removal of blood that may produce cytoxic 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 [39]. Patients with lobar ICH within 1 cm of the cortical surface may have benefited from surgical evacuation, and this group is currently being studied in STICH II. 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 [40–42]. At the moment, 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 [43, 44]. 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 AHA 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]. Patients with lobar ICH with hematoma volume >30 cc and within 1 cm of the cortical surface or those with significant life-threatening mass effect may also be considered for surgery. Correction of coagulopathy is critical in patients undergoing surgical hematoma evacuation.

Table 4 Standardized ICH checklists

Prehospital care
☐ ABCs
☐ Determine time of onset and circumstances
☐ Perform prehospital stroke screen
☐ 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, 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 (targets SBP <160 mmHg or MAP <110 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)
Inhospital 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 on day 3)
☐ Recheck INR and PTT 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

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 [45]. 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 the transition in care is smooth from ED to ICU (or operating room, interventional radiology, or

Table 5 Intracerebral hemorrhage communication regarding assessment and referral

- ☐ Age
- ☐ Relevant known medical history (e.g., hypertension, prior SAH or ICH, cancer, drug abuse)
- ☐ Relevant known medications (e.g., anticoagulants, antiplatelets)
- ☐ Brief history of new stroke event (if transferred from other facility, provide summary)
- ☐ Time of symptom onset (include witnessed or "found down" with last time known normal)
- ☐ Prehospital GCS and vital signs: SBP/DBP, HR, GCS
- ☐ Condition on arrival to ED: SBP/DBP, HR, GCS
- ☐ Head CT results:
  - Hematoma location and volume (cc)
  - Presence and degree of IVH
  - Contrast extravasation ("spot sign")
  - Midline shift (mm)
- ☐ Status of laboratory tests (pending, available): INR, platelet count, creatinine
- ☐ ICH Score (0–6)
- ☐ ED interventions performed: intubation (including paralytics/sedatives used), BP treatment, coagulopathy correction (PCC, FFP, platelet transfusion)
- ☐ Which consultants have been called (name):
  - Neurosurgery
  - Vascular Neurology
  - (Neuro) Critical Care
- ☐ Vascular access (e.g., peripheral IV, central line, arterial line)
- ☐ Current neurological status: GCS, NIHSS
- ☐ Current patient location
- ☐ Other relevant events in ED (e.g., neurological deterioration, presence and location of family)

Sample sign-off narrative

"I am signing out a 62 year man with known hypertension and atrial fibrillation who is presumed to be on warfarin. He was found at home this morning at 9 AM by his wife who last saw him normal at 7 AM. He was talking to EMS and had left-sided weakness, GCS in the field was 13, and BP was 170/100."

"On arrival to the ED here, he was the same, so we took labs and sent him for a head CT."

"CT completed at 10 AM showed a 20-mL right thalamic ICH with mild IVH, but no hydrocephalus. There is about 4 mm of right-to-left midline shift. CTA/CTP showed no AVM or aneurysm, but there is a positive spot sign."

"When he returned to the ED, he was sleepier, with a GCS of 10, and his left-sided weakness was worse. So he has an ICH Score of 2. His labs came back with an INR of 2.8."

"We intubated him using rocuronium and etomidate. PCC infusion of 2,250 IU (estimated weight 90 kg; dose of 25 IU/kg) is going in now. He also had 10 mg of IV vitamin K."

"Neurosurgery has been called, and they are on their way to see him. He is in ED resuscitation room 1, intubated and sedated now on propofol at 60 mcg/kg/min. His BP is 150/85 with no other treatment."

"They are ready to take him in Bed 2 in the neurocritical care unit in 5 min. Nursing is also calling report."

comprehensive stroke center). The use of standardized ICH checklists (Table 4) are encouraged.

While this 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 fever, hyperglycemia/hypoglycemia, and hypoxia are also important, as these may affect outcomes [1, 46, 47]. In addition, patients with ICH are at increased risk for the development of deep venous thrombosis (DVT); current guidelines recommend use of compression stockings and 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 [48–50]. 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; 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 before ICP monitor insertion is desirable.

While seizures may occur in ICH patients, their incidence and impact on outcome have varied across studies [51, 52]. In a single study, prophylactic anticonvulsants reduced seizure occurrence in lobar ICH [52]. However, two additional recent studies found worse functional outcomes in patients routinely given prophylactic anticonvulsants (primarily phenytoin) [53, 54]. 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 [55, 56]. Current guidelines do not recommend routine use of prophylactic anticonvulsants [1, 2], 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.

Communication

When communicating to an accepting or referring physician about a patient with ICH, consider including the key elements listed in Table 5.

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