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Review

Spontaneous Intracerebral Haemorrhage: A Practical Guide to Diagnosis and Management

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Abstract

We present a topical review intended to update on the pathophysiology, investigation and management of intracerebral haemorrhage (ICH). Recent advances in the field have refined our knowledge of managing ICH and, when implemented, can significantly improve patient outcomes. The aim of this article is to describe the common causes of acute ICH that might be encountered in accident and emergency departments or hyperacute stroke units, and to discuss investigation and management. Reading this article will give the reader confidence in establishing the diagnosis of ICH, starting the investigation pathway and instigating evidence-based acute management to significantly improve outcomes across the United Kingdom. We have included case vignettes with neuroradiology expert opinion on imaging of more commonly found aetiologies of intracerebral haemorrhage. Neurosurgical management is also discussed in addition to medical management, with the limits to knowledge elucidated.

Keywords: intracerebral haemorrhage; stroke; cerebral amyloid angiopathy; acute stroke

1. Introduction

In 2021, stroke ranked as the third leading cause of death worldwide, with intracerebral haemorrhage (ICH) responsible for nearly half of its burden in terms of mortality and disability-adjusted life years (DALYs) [1,2,3]. Despite a gradual decline in age-standardized stroke incidence, ICH remains disproportionately prevalent in regions with a low socio-demographic index (SDI), particularly in Southeast Asia, East Asia, and Oceania. Hypertension and increasing age are the most common risk factors [3,4,5] for both deep and lobar ICH, but do not explain these variations alone. Ischaemic stroke accounts for most strokes worldwide, but the equal prioritization of ICH alongside ischaemic stroke is essential to drive progress in public health prevention programmes, research and care delivery [5].

Mortality due to ICH remains significant despite an overall decrease in the number of deaths due to the condition, and in 2021 claimed 3.3 million lives worldwide [6]. As life expectancy is increasing in the UK, there is expected to be a substantial increase in the number of deaths due to ICH seen in those over 70 years old over the coming years [7].

Incidence of ICH varies widely between countries, being highest in China, Japan and Southern Greece [6,8], and when Asians migrate, this high incidence tends to travel with them [8]. In China, there seems to be a correlation with hypertension, and those living in Asian countries also have higher salt intake than Western Europe and the USA [8,9]. Rates of ICH in high-income Asian regions declined in 2021, probably due to improving lifestyles, lending credence to public health awareness campaigns as a way of significantly reducing incidence [6,9]. The higher numbers in Japan and Greece can be to some extent explained by longer life expectancies. There is also a high incidence of hypertension and vascular disease generally in Greece [8]. Of course, accurate numbers of ICH patients cannot be established in countries without national registries, and the whole continent of Africa remains unfathomable with regard to establishing incidence [8].

Spontaneous ICH is defined as a focal collection of blood within the brain parenchyma or ventricular system that is not due to trauma. A nomenclature originally coined by neurosurgeons in the 1930s who needed to be able to differentiate traumatic and non-traumatic ICH [10], this is still used today in national guidance and produces the least confusion [11]. The term ‘haemorrhagic stroke’ is used variably to refer to a range of different types of intracranial bleeding, including aneurysmal subarachnoid haemorrhage or secondary haemorrhage into an infarct and can cause confusion [12]. There is no single internationally used classification system for ICH, but it is usual to classify by location, e.g., lobar or non-lobar. Further classification using imaging markers is limited by the availability of magnetic resonance imaging (MRI) imaging, which can provide aetiological clues to help [12,13]. A list of causes of spontaneous (i.e., non-traumatic) ICH can be seen in Table 1 (Ref. [12,13,14]); the most common, accounting for about 80% of all cases, are the two common age-related cerebral

Table 1. Causes of spontaneous (i.e., non-traumatic) intracerebral haemorrhage [12,13,14].

Cerebral small vessel diseases
Cerebral amyloid angiopathy (CAA)
Arteriolosclerosis
Mixed (both CAA and arteriolosclerosis)
Macrovascular disease
Arteriovenous malformation (AVM)
Aneurysm
Cavernoma
Dural arteriovenous fistula
Vasculopathies
Infective endocarditis (mycotic aneurysms)
Cerebral infections, e.g., varicella zoster
Moyamoya disease
Primary CNS vasculitis
Reversible cerebral vasoconstriction syndrome (RCVS)
Bleeding diatheses (coagulopathies)*
Thrombocytopaenia
Haemophilias
Disseminated intravascular coagulation syndrome
Iatrogenic*
Oral anticoagulation
Thrombolysis
Other
Haemorrhagic infarction due to cerebral venous sinus thrombosis
Haemorrhagic transformation of an arterial infarct
Brain tumour (primary or metastatic)
Recreational drugs, especially sympathomimetic agents (e.g., amphetamine, cocaine)

*note that these rarely cause ICH alone, i.e., in the presence of structurally normal cerebral vessels.
Bold type is for clarity.

small vessel diseases: arteriolosclerosis (causes >90% of non-lobar and at least 50% lobar haemorrhages) and cerebral amyloid angiopathy [12]. Genetic conditions which are linked to a higher ICH risk have been identified and can be due to polymorphisms in several genes, e.g., APOE ε4. Rarer types of cerebral small vessel disease that can cause ICH in younger people include monogenetic conditions, e.g., CADASIL, hereditary forms of CAA, and collagen gene mutations (COL4A1/2). Other conditions affecting small vessels (e.g., vasculitis, autoimmune disease, post-radiation vasculopathy) account for only a very small proportion of ICH [13].

Acute interventions for ICH were previously limited by a lack of evidence; however, there is now emerging evidence for benefits from very early intensive blood pressure reduction to a target of 130–140 mmHg systolic [15]. However, this did worsen patients with ischaemic stroke, so there will be limited scope in implementing this in clinical practice, as differentiation of the two requires neuroimaging which will not be available to paramedics. This does, however, inform management in the emergency department where blood pressure management should be prioritised [16]. There is also increasing evidence for early intervention with minimally invasive surgical evacuation, which does improve outcomes in those with anterior basal ganglia and lobar haemorrhages [17]. This helps us to reconsider hesitancy in sending patients for neurosurgical intervention, which happened due to delays in accessing services and also a lack of clear evidence that this would improve the outcome. Understanding of multidisciplinary care and service provision has also evolved, with increasing evidence indicating that multidisciplinary care with ‘bundles’ which include monitoring on a hyperacute stroke unit, prompt anticoagulation reversal, rapid blood pressure lowering and access to high dependency beds when appropriate significantly reduces 30-day mortality [16,18].

2. Imaging and Diagnosis

Neuroimaging of the brain and cerebral blood vessels is key in investigating ICH as it not only confirms the diagnosis but also provides important clues as to aetiology, aiding classification and diagnosis [12,19]. Clinical history, examination including fundoscopy and symptomatology are also important in guiding diagnostic tests, management and investigations. The clinical onset of ICH is usually sudden but subsequently may be progressive as

haematoma expansion occurs [13]. The symptoms and signs of ICH are of focal neurological deficit, according to the anatomical location of haemorrhage, but may also include signs of raised intracranial pressure such as headache, confusion, vomiting, seizures and reduced level of consciousness [12].

Computed tomography (CT) is rapid, accessible and is ideal in use in the acute phase where there is a risk of a drop in consciousness compromising the airway or seizures which make MRI scanning unsafe until the patient is stabilised. CT will show the volume of the haematoma and signs of mass effect and rising intracranial pressure and can be repeated to monitor haematoma expansion which, when it occurs, is usually in the first six hours [19,20]. It is demonstrated by an increasing volume of blood on serial CT scans. Poor prognostic signs seen on initial CT are a large initial haematoma, infratentorial (posterior fossa or brainstem) origin of bleed and intraventricular haemorrhage [20]. In addition to diagnosis, acute CT can be useful for establishing aetiology, i.e., microvascular and macrovascular causes given in Table 1. Features suggesting cerebral amyloid angiopathy (CAA) are subarachnoid extension of bleed and finger-like projections; if both of these are present, then the probability of CAA is likely to be very high [21]. This can be helpful when, for example, the patient cannot tolerate an MRI scan or has incompatible implants. CT angiogram (CTA) or MR angiogram (MRA) are appropriate initial investigations to detect macrovascular bleeding sources including arteriovenous malformations or aneurysms [19,22]. CTA is usually the easier of the two to access and can be done in the acute phase with initial CT scanning. CTA may also demonstrate a 'spot sign', which is an independent predictor of haematoma growth, as it shows extravasated contrast within the bleed itself; this can be 'mimicked' by calcification [23]. The gold standard for establishing a macrovascular cause of ICH is intra-arterial digital subtraction angiography (IADSA) [24]. This is usually done after an interval, as it requires a skilled neuroradiologist to perform and also carries procedural risk which the patient needs to be consented for. Diagnostic accuracy studies comparing acute CTA with IADSA suggest that diagnostic accuracy is modest, with CTA having a sensitivity of 74% and specificity of 91% for detecting vascular anomaly [24]. This means that IADSA may be necessary even with normal CTA imaging to detect vascular abnormalities which were not visible on CTA. Review of the case with imaging can be helpful in establishing the need for IADSA, and this is usually done in a multidisciplinary forum with clinicians and neuroradiologists. Considering factors such as older age, deep location and hypertension with markers of end-organ damage, e.g., renal failure and hypertensive retinal changes, can clarify hypertension as a major risk factor and remove the need for further imaging [16]. Recent data clearly show that the presence of features of cerebral small vessel disease on an admission CT scan predicts a low yield of a macrovascular cause, so this can also be helpful when decision-making about the need for IADSA [25] to definitively rule out a macrovascular cause of ICH. Decision-making can be aided further by the use of imaging-based scoring systems. The DIAGRAM score uses information from CT and CT angiography to predict the yield from IADSA and is regularly used in clinical practice [25]. The MRI-based MACRO score can also be used to estimate the probability of a macrovascular cause; it is usual practice where MRI is available to request this after the acute phase, when CT and CTA are preferred [26]. The use of the MACRO score is therefore limited by MRI availability. MRI findings in ICH which help establish aetiology can be seen in Table 2 (Ref. [19,22,27]). Being able to establish aetiology is important as it is linked to prognosis with a far higher rate of recurrent lobar haemorrhage associated with cerebral amyloid angiopathy; one meta-analysis reported a recurrence rate of 7.4% for CAA-related ICH compared with 1.1% for ICH of any other cause [28], while a recent cohort study found a comparable recurrence rates of 8.5% for CAA-related ICH and 0.6% for ICH due to arteriolosclerosis [29]. Information about ICH recurrence rates can help to inform conversations with patients, families and other clinicians about prognosis.

3. Emergency Management

Hyperacute stroke units (HASUs) are 'hubs' which typically serve populations of about 1–2 million people and are fed into by surrounding hospital emergency departments and general practices. HASUs have stroke-trained teams who provide multidisciplinary care to patients with acute stroke [30]. They are also usually co-located with neurosurgical centres. It is usual practice for patients with ICH to be stabilised within emergency departments and immediately referred and transferred to a HASU for ongoing care [30].

3.1 Initial Assessment and Stabilisation

The patient diagnosed with ICH is evaluated as any other acutely unwell patient using an ABCDE approach, working through a systematised evaluation of airway, breathing, circulation, disability and exposure to check for injury [31]. Special attention should be paid to the airway, which can be compromised in patients with a reduced level of consciousness. In addition, seizures are relatively common at the onset of ICH and may be the presenting feature [32]. Standard protocols are used for symptomatic early seizure management as there is no favoured anti-epileptic drug in acute stroke. Treatment of any symptomatic seizure is recommended, but not prophylactic antiseizure medication [32]. ITU support may be necessary in those with ongoing seizures. After stabilising and initiating monitoring in the emergency department, early brain imaging with non-contrast CT head is recommended to confirm the diagnosis and look for signs of raised intracranial pressure [18].

Table 2. MRI imaging with specific sequences which can be used to diagnose aetiological factors which are causative of ICH [19,22,27].

Imaging findings	Imaging sequence	Disease process detected
White Matter Hyperintensities	Fluid Attenuated Inversion Recovery (FLAIR)	Found in all forms of cerebral small vessel disease including arteriosclerosis and CAA
Perivascular Spaces	T2 weighted	Marker of small vessel disease and part of diagnostic criteria for CAA when seen in the centrum semiovale
Cerebral microbleeds	Susceptibility Weighted Imaging (SWI), T2*weighted	Cerebral small vessel diseases: can be found in both arteriosclerosis or CAA. Also found in rarer conditions, e.g., endocarditis
Subcortical infarction	T1 weighted, FLAIR	An imaging marker of small vessel disease
Cortical Superficial Siderosis	SWI, T2*	Highly specific for CAA
Venous filling defect	MR Venogram (MRV)	Venous occlusion
Arteriovenous anomalies	MR Angiogram (MRA)	AVM, aneurysm, cavernoma, dural arteriovenous fistula
Vessel wall inflammation	'Black-blood' vessel wall imaging MRA	Medium-vessel cerebral vasculitis.
Mass lesion (e.g., cerebral tumour)	MRI with contrast	Solid tumour or infiltrative process - can be primary brain tumour or secondary metastasis

"*" is the name of the imaging sequence T2 'star'.

A flow chart of management of ICH in the emergency department can be seen in Fig. 1 (Ref. [33]).

3.2 Reversal of Anticoagulation

Urgent reversal of anticoagulation to reduce haematoma expansion if they are on the patient's medication list should be undertaken. For warfarin-associated ICH, prothrombin complex concentrate (PCC) and vitamin K should be given as quickly as possible to inhibit its action; protocols can vary between hospitals and usually require consultation with haematology to be issued by the laboratory. The PCC dosing regimen is dependent on both weight and INR value and stroke units may have produced their own local guideline, e.g., UCLH London-based HASU, which is published in the literature [34,35]. For ICH associated with direct oral anticoagulants (apixaban, rivaroxaban and edoxaban), andexanet alfa initially had some positive trial data [36] but has more recently been withdrawn from the market due to concerns about thromboembolic complications. Commonly, fresh frozen plasma is advised to patients who are on direct oral anticoagulants (DOACs) and have ICH via haematology consultation, but there is no evidence that this is effective in improving outcomes. Antiplatelets have no reversal agent but should be stopped; there is no evidence that platelet transfusions improve outcomes [18].

3.3 Blood Pressure Treatment

Rapid blood pressure (BP) management is recommended if the blood pressure is above 150 mmHg systolic, as control in the acute phase is associated with reduced death and disability at 3 months [15,37,38]. Recent evidence from an individual patient data meta-analysis of 11,312 participants from the INTERACT trials found that intensive blood pressure lowering significantly improved the chances of functional recovery, with more patients achieving good outcomes of mRS 0–3 [39]. Intravenous routes of antihypertensive delivery can be used, or a nasogastric tube. The emphasis is on early control (within three hours), with the biggest treatment benefits in those with a mildly raised BP of 150 mmHg; patients with very high values of 200 mmHg or higher gain no benefit or may even have worse outcomes. Intensive lowering of the blood pressure (e.g., to less than 110 mmHg in the ATACH 2 trial) in the acute phase appears to have a negative effect overall, with possible harm to those with moderate to severe chronic kidney disease and those with known cardiac disease [37,39,40]. Once lowering is commenced, monitoring is then required to ensure it remains stable and does not rebound. The aim is for gradual lowering and avoiding precipitous drops. By close monitoring, it should be safe to lower the blood pressure in all cohorts of patients, for example dialysis patients. Fast-acting intravenous antihypertensives with short half-lives are most effective and safe in this context—usually intravenous labetalol, but in case of contraindication such as asthma or bradycardia, then hydralazine or nicardipine can be used as an alternative [40]. If BP is resistant to lowering, then a transfer to a high dependency or intensive care unit and more intensive treatment with arterial line monitoring can be necessary [13]. At the same time as intravenous treatment, oral agents should be introduced early to prevent rebound increases in blood pressure and to avoid prolonged intravenous treatment. In those with mildly raised blood pressure (130–150 mmHg systolic) or late presenters, continuing usual anti-hypertensive medications may be sufficient [40].

3.4 Surgical Intervention

In the acute phase, raised intracranial pressure is caused by the space-occupying effect of the acute

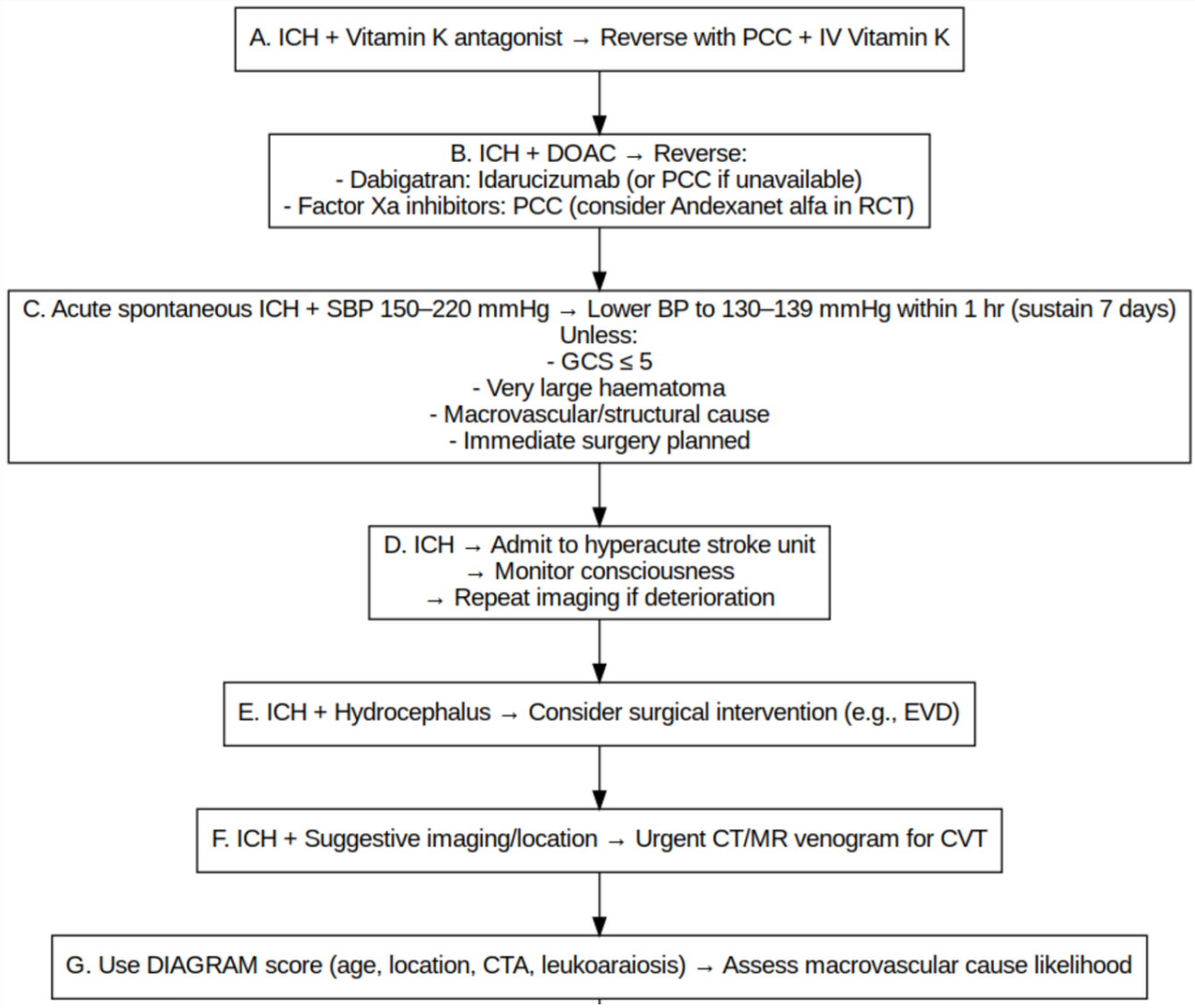

Fig. 1. Emergency management of acute ICH in the emergency department. Adapted from the National Clinical Guideline for Stroke (2023) [33]. ICH, intracerebral haemorrhage; PCC, prothrombin complex concentrate; DOAC, direct oral anticoagulant; RCT, randomised controlled trial; SBP, systolic blood pressure; BP, blood pressure; GCS, Glasgow coma scale; EVD, external ventricular drain; CT, computed tomography; MR, magnetic resonance; CVT, cerebral venous thrombus; CTA, CT angiogram.

haematoma, with an increased risk in those with haematoma expansion and significant peri-haematomal oedema [41]. Surgical management of ICH can include intracranial pressure (ICP) monitoring, e.g., with an ICP bolt, external ventricular drainage (for hydrocephalus) or more invasively with haematoma evacuation and decompressive craniectomy to reduce ICP. Earlier trials have never shown a clear treatment benefit with surgery, so conservative management has always been preferred where possible, with surgery only in cases where it was thought coning was probable [41,42]. However, surgical techniques have been developed, and a more recent meta-analysis of minimally invasive techniques for haematoma evacuation was weakly positive [43]. Following this, the SWITCH followed by ENRICH trials had a more strongly positive outcome, providing hope [17,44]. The combined effect size was greater than blood pressure lowering in the acute phase, which is the current standard of care. It should be noted that this was for patients with supratentorial ICH only who were operated on with minimal delay. It seems we are early on in our development of minimally invasive surgery as an intervention, as a recent Cochrane meta-analysis of all available evidence has reinforced uncertainty, showing overall weakly positive evidence in favour of intervention [45]. European guidelines have been updated to advise that minimally invasive surgery within the first 24 hours of bleed onset can have benefit and reinforce a message to discuss cases with neurosurgeons [32]. In addition to the patient's physiological reserve factors which influence decision-making about neurosurgical intervention are location and volume of ICH, level of consciousness and timing since onset of bleeding. Good outcomes are associated with supratentorial location,

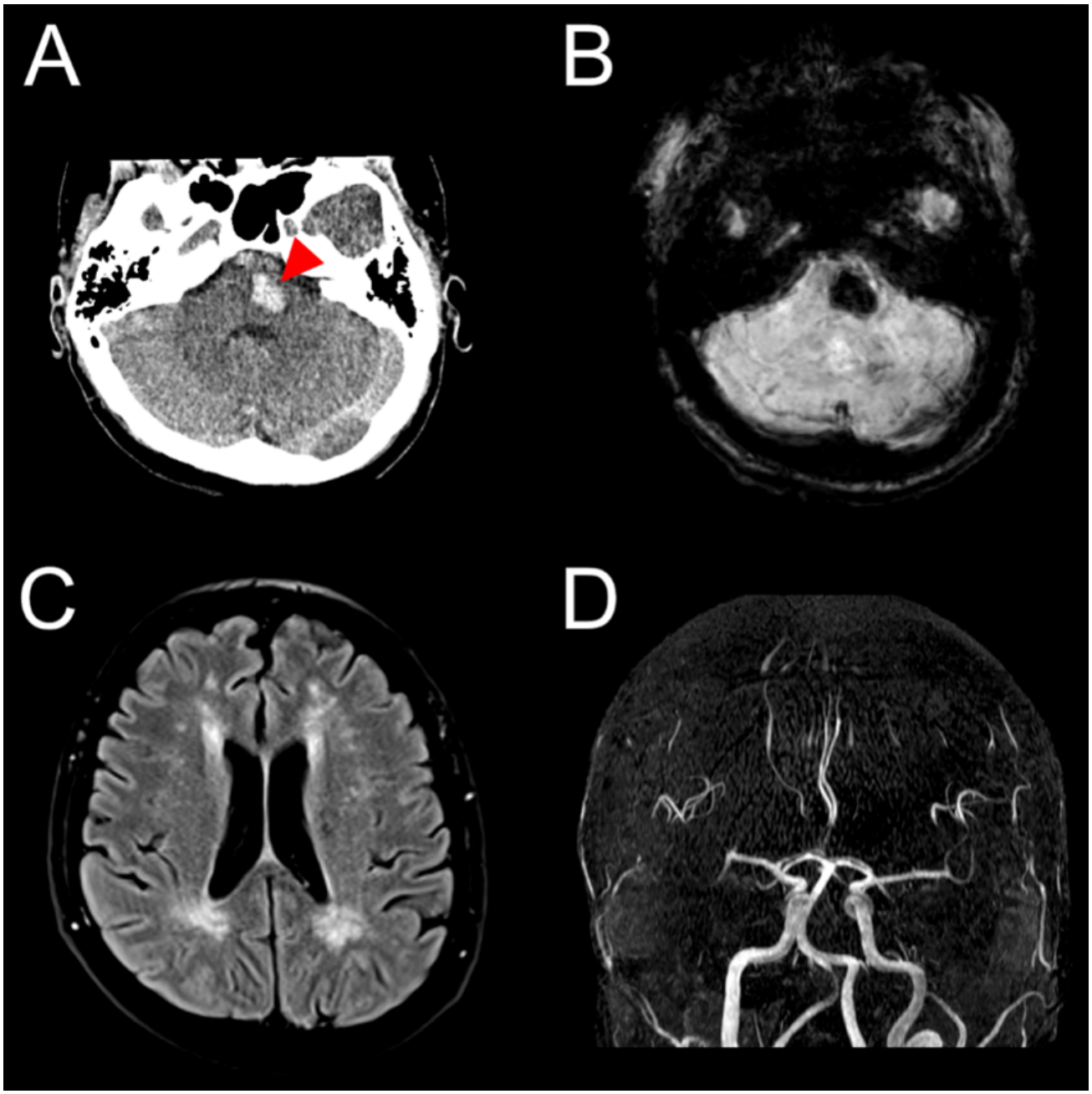

Fig. 2. Acute CT, MRI and MRA sequences of patient in Case 1. An acute haematoma within the pons was shown on CT (A, red arrow) and susceptibility weighted imaging (B). Fluid-attenuated inversion recovery imaging showed confluent white matter hyperintensity that was consistent with a severe burden of small vessel disease (C). Tortuosity of the vertebrobasilar system on time-of-flight angiography indicated dolichoectasia (D).

quick intervention, and lack of intraventricular blood, although there is still overall significant post-surgical neurodisability in this cohort of patients [46].

3.5 Seizures

These are usually treated in the same way as seizures of any other cause, with initial control with short-acting benzodiazepines and loading doses of intravenous anti epileptics with maintenance therapy [32,47]. There is no evidence for prophylactic anti-seizure medications [32]. Aspiration pneumonia is another complication of acute ICH, either due to airway compromise caused by reduced level of consciousness, seizures or a loss of swallow function [48]. It is managed in accordance with local antibiotic protocols and respiratory support as required. This persists through the acute and subacute phase as a common complication of ICH.

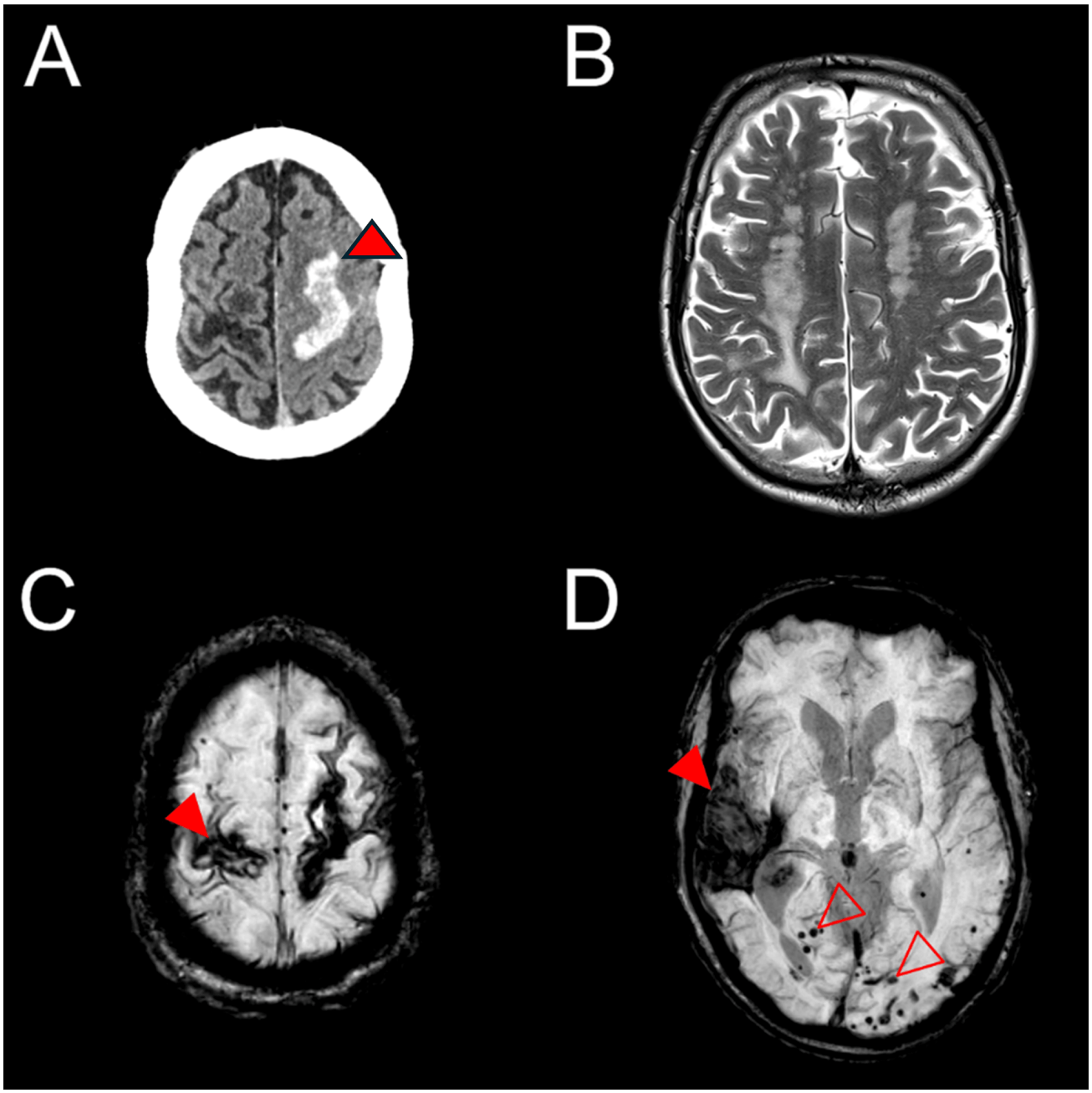

Fig. 3. Acute CT and MRI imaging of patient in Case 2. CT showed an acute lobar haematoma in the left superior frontal gyrus (A, red arrow). T2-weighted imaging showed confluent white matter hyperintensity that was consistent with a severe burden of small vessel disease (B). Susceptibility weighted imaging showed, in addition to the recent haematoma in the left frontal lobe, an old intragyral haematoma in the right frontal lobe (C, red arrow) and right temporal lobe (D, red arrow) as well as many lobar microhaemorrhages (D, open arrows).

4. Secondary Prevention

Secondary prevention is focused on managing risk factors for further ICH and managing any iatrogenic factors such as anticoagulation or antiplatelets which would have been stopped at the time of ICH.

Consideration may need to be given to restarting antiplatelets or anticoagulation after ICH in circumstances where they are necessary, e.g., metallic heart valve, atrial fibrillation or significant vasculopathy in cerebral, myocardial or lower limb vascular beds. An early (within 7 days) restart of anticoagulation in the case of patients with metal heart valves is preferred, and this can be with low molecular weight heparin or intravenous heparin [49]. The patient then needs to have level of consciousness and neurological signs monitored for signs of rebleed for at least some days. In patients in atrial fibrillation, restart of oral anticoagulation needs to be carefully considered, and if benefit

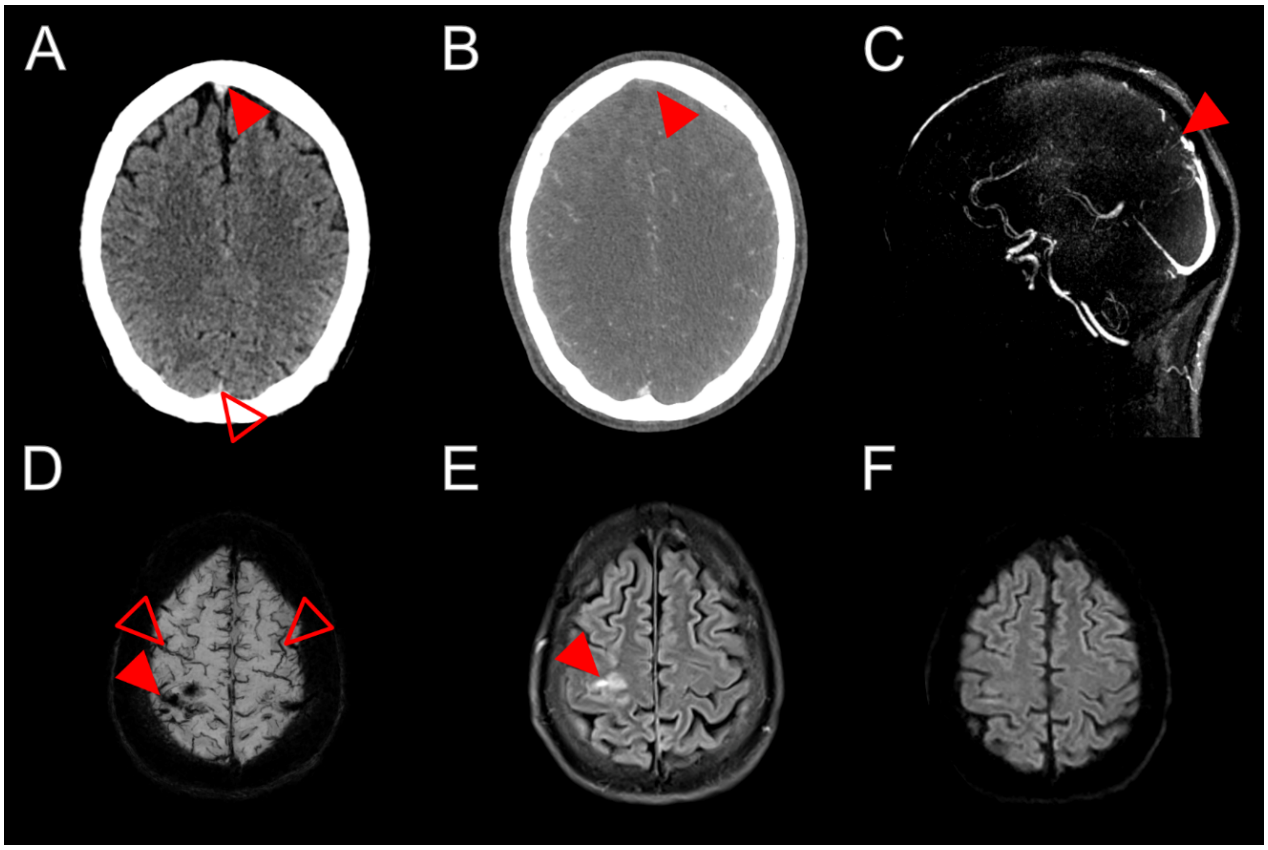

Fig. 4. Acute CT, MRV and MRI imaging of patient in Case 3. The anterior superior sagittal sinus was denser on CT anteriorly (A, red arrow) than posteriorly (open arrow) due to acute thrombus. This was confirmed on CT and MR venography, where there was no contrast seen (B, red arrow) or flow-related signal (C, red arrow) within the anterior superior sagittal sinus. Susceptibility weighted imaging showed blooming artefact within cortical veins (red arrow) due to thrombus (red arrow) and venous congestion (open arrows) (D). The right-sided cortical vein thrombus was associated with Fluid Attenuated Inversion Recovery (FLAIR) hyperintensity within the precentral gyrus due to oedema (E, red arrow). Diffusion weighted imaging showed only subtle hyperintensity but no evidence of venous infarction (F).

outweighs risk then DOAC is the preference with significantly reduced mortality when compared to warfarin but with similar rates of ischaemic stroke and recurrent ICH [50,51]. Risk stratification tools to evaluate the risk-benefit of anticoagulation can be somewhat helpful to establish the risk of systemic bleeding, but as they do not take into account the nuanced features influencing decision-making, such as imaging markers of higher risk of rebleed, e.g., cortical siderosis or aetiology of ICH [50]. Decision-making is usually by a stroke physician in a multidisciplinary forum, as case experience, haematology and radiology input is vital in decision-making. Patients who have capacity should also be informed about the risk-benefit of restarting anticoagulation, so they may make an informed choice, with a shared decision-making approach always preferred. Those who lack capacity have decisions made for them in their best interests, consultation with a next of kin may be appropriate.

Restart of antiplatelets in those with significant ischaemia-related disease in their vascular, myocardial or vascular arterial tree is also considered to be safe based on the findings of a randomised controlled trial which confirmed benefits outweighed the risk of rebleed [52].

Reduced recurrent rate of ICH is seen in those with CAA and/or hypertension-related disease with blood pressure reduction in line with standardised guidelines for the general population to a value of <130/80 mmHg [53]. Sustained reduction is the aim and no agent is considered superior in risk reduction. A recently published trial showed that a ‘polypill’ of three low-dose agents was effective, making this a useful option in patients who struggle with adherence [54]. This showed that long-term antihypertensive therapy led to a ~22% relative reduction in recurrent ICH with intensive BP lowering and demonstrated that achieving target systolic BP <130 mmHg was both feasible and clinically beneficial. Mean systolic BP in the intensive-treatment arm was reduced by approximately 10–12 mmHg; recurrent ICH occurred in 3.8% of participants receiving intensive therapy versus 4.9%. However, only around two-thirds of participants maintained BP within target ranges

over follow-up, suggesting that this benefit may be attenuated outside trial conditions, including low-resource settings, where access to reliable BP monitoring is limited, and clinic follow-up may be sporadic. Nevertheless, simplified antihypertensive regimens, community-based monitoring strategies, and pragmatic BP targets may help translate TRIDENT's findings into routine care globally.

5. Illustrative Cases

Below we have compiled imaging and cases to illustrate some of the points made in the main text.

5.1 Case 1

A 47-year-old man presented with right-sided weakness and ataxia. He also had horizontal diplopia and dysarthria. On examination, he had a right hemiparesis with loss of sensation to light touch and left cranial nerve 6 and 7 palsy. His blood pressure was uncontrolled, with a systolic of more than 200 mmHg. He was on no regular medication other than antidepressants. Immediate management consisted of controlling his BP to a systolic of approximately 140 mmHg with intravenous labetalol bolus and infusion. His CT and MRI imaging are shown in Fig. 2. Chronicity of his hypertension was shown by early retinal changes on fundoscopy of hypertension-related disease. The diagnosis of ICH caused by small vessel disease (arteriolosclerosis) was made. Long-term secondary prevention will be achieved by good BP control and addressing his lifestyle factors via diet and exercise.

5.2 Case 2

A 71-year-old man presented with expressive dysphasia and right-sided hemiparesis. He was on antihypertensive agents, and his family confirmed compliance and regular monitoring of his BP at home. He was consistently in the normotensive range. The CT and all subsequent imaging are shown in Fig. 3. Notes confirmed this was his third lobar ICH since his mid-60s. Multiple microhaemorrhages seen on MRI confirmed the diagnosis of probable cerebral amyloid angiopathy.

5.3 Case 3

A patient in his mid-50s with a history of polycythaemia rubra vera presented with a 2-day history of headache which did not improve with paracetamol. His imaging is shown in Fig. 4. Immediate anticoagulation was given with low molecular heparin at a treatment dose, and after therapeutic venesection the patient was then given a direct oral anticoagulant for lifelong anticoagulation.

6. Conclusion

Intracerebral hemorrhage remains a common neurological emergency, causing a heavy burden of death and disability worldwide. Acute management is focused on airway management and blood pressure control, and urgent reversal of oral anticoagulation when indicated. Initial CT imaging is usually the first step in establishing the diagnosis, followed by MRI with blood-sensitive sequences to look for a cause. Early complications other than loss of airway are commonly seizures and aspiration pneumonia, which require prompt management if they arise. Neurosurgical intervention via minimally invasive methods shows considerable promise but is currently not widely used; most cases are managed medically by emergency, medical and stroke teams. Prompt diagnosis and care of acute ICH (oral anticoagulation reversal, blood pressure control, referral to high dependency wards and neurosurgery as appropriate) can lead to better neurological outcomes and save lives.

Key Points

• ICH has a higher mortality than ischaemic stroke and causes significant disability worldwide.

• The most common underlying cause is cerebral arteriosclerosis, for which the strongest risk factor is uncontrolled hypertension.

• Rapid blood pressure lowering in the acute phase of ICH to a target of $\leq$140 mmHg systolic appears to help reduce mortality and reduce disability; very early treatment is likely to be most effective; lower than 130 mmHg may worsen outcomes.

• Reversal of oral anticoagulation can reduce haematoma expansion and may improve outcome.

• Minimally invasive neurosurgery shows promise to improve functional outcome after ICH, but further data from randomised trials are needed.

Availability of Data and Materials

Not applicable.

Author Contributions

SKG contributed to the conception and scope of the study, curation of included articles, image selection and obtaining patient consent, and manuscript writing and editing. DHM contributed to imaging analysis and obtaining patient consent, and contributed to reviewing and revising the final draft for important intellectual content. DJW contributed to the conception and scope of the study, curation of included articles, and manuscript writing and editing. All authors gave final approval of the version to be published. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki. All three patients have given informed consent for their imaging and clinical history to be used in a non-identifiable way.

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Conflicts of Interest

The authors declare no conflicts of interest. David Werring is an NIHR Senior Investigator and has received: grant funding from the Stroke Association, British Heart Foundation, Rosetrees Trust, Innovate UK, UCLH Biomedical Research Centre and National Brain Appeal Innovations fund; speaking honoraria from Bayer; speaking and chairing honoraria from AstraZeneca (Alexion) and NovoNordisk; and consultancy fees from Alnylam, Bayer and NovoNordisk. He was Chief Investigator for the PROHIBIT-ICH and OPTIMAS trials. He has participated in the data safety monitoring board for OXHARP, and the LACI-2, TICH-2, TICH-3, RESTART, MACE-ICH and PLINTH Trial Steering Committees.

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