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Clinical paper

Bedside monitoring of hypoxic ischemic brain injury using low-field, portable brain magnetic resonance imaging after cardiac arrest



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Abstract

Background: Assessment of brain injury severity is critically important after survival from cardiac arrest (CA). Recent advances in low-field MRI technology have permitted the acquisition of clinically useful bedside brain imaging. Our objective was to deploy a novel approach for evaluating brain injury after CA in critically ill patients at high risk for adverse neurological outcome.

Methods: This retrospective, single center study involved review of all consecutive portable MRIs performed as part of clinical care for CA patients between September 2020 and January 2022. Portable MR images were retrospectively reviewed by a blinded board-certified neuroradiologist (S.P.). Fluid-inversion recovery (FLAIR) signal intensities were measured in select regions of interest.

Results: We performed 22 low-field MRI examinations in 19 patients resuscitated from CA (68.4% male, mean [standard deviation] age, 51.8 [13.1] years). Twelve patients (63.2%) had findings consistent with HIBI on conventional neuroimaging radiology report. Low-field MRI detected findings consistent with HIBI in all of these patients. Low-field MRI was acquired at a median (interquartile range) of 78 (40–136) hours post-arrest. Quantitatively, we measured FLAIR signal intensity in three regions of interest, which were higher amongst patients with confirmed HIBI. Low-field MRI was completed in all patients without disruption of intensive care unit equipment monitoring and no safety events occurred.

Conclusion: In a critically ill CA population in whom MR imaging is often not feasible, low-field MRI can be deployed at the bedside to identify HIBI. Low-field MRI provides an opportunity to evaluate the time-dependent nature of MRI findings in CA survivors.

Keywords: Neuroimaging, Hypoxic-ischemic encephalopathy, MRI, Cardiac arrest

Introduction

Cardiac arrest (CA) is a major cause of morbidity and mortality, with almost 600,000 cases annually in the United States.¹ Only 1 in 10 out-of-hospital CA and 1 in 4 in-hospital CA patients survive to hospital discharge.² Over two-thirds of deaths in successfully resuscitated CA patients are related to hypoxic ischemic brain injury (HIBI).^{3–5} Accurate outcome prediction is critically important, as withdrawal of life sustaining therapy (WSLT) most often occurs due to

perceived poor neurologic outcome.^{6,7} Neuroimaging is a valuable prognostication tool in cardiac arrest.⁸ Magnetic resonance imaging (MRI) is more sensitive than computed tomography (CT) at detecting subtle abnormalities related to HIBI and has a higher sensitivity than CT for predicting poor neurologic outcome.^{9,10}

Despite the high value clinicians place on MRI for neurologic prognostication after CA, it remains underutilized.¹¹ Conventional MRI systems operate at high magnetic field strength, which requires a strict access-controlled environment, expensive infrastructure, technicians, and transport out of the intensive care unit (ICU). In

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addition, there are risks of transporting critically ill patients to imaging suites, including compromise of venous or arterial access, endotracheal tube displacement, hypoxia, hypotension, increased intracranial pressure, and recurrent cardiac arrest.^{12–17} Identified factors associated with clinical complications during intrahospital transport include the use of sedatives, vasopressors, and transport times exceeding 36 minutes.¹⁸ Up to 20% of patient transports are aborted, occurring most commonly in those who require transport to MRI.¹⁹ Patients resuscitated from CA are at high risk for complications secondary to transport, which often excludes or delays MRI. Delay to MRI is associated with increased length of hospital stay and total hospitalization cost.²⁰ Furthermore, MRI is an established methodology for quantification of cerebral edema and neuronal death.²¹ Limited MRI use in this patient population precludes the development of intermediate endpoints that are essential for identifying candidate neuroprotective treatments.

Advances in MRI technology have led to the development of low magnetic field image acquisition with portable MRI scanners.¹² Portable, bed-side MRI is safe and feasible to perform in critically ill patients and can identify pathology similar to that observed on conventional MRI.^{22,23} We report the first use of low-field (64mT) MRI to evaluate HIBI in CA survivors. Our primary objective was to identify whether portable MRI could detect HIBI observed on conventional neuroimaging. Given the global nature of injury in HIBI, we measured fluid-attenuated inversion recovery (FLAIR) signal intensity to provide an objective measure of HIBI. Our secondary objective was to evaluate the safety of portable MRI in this critically ill population. We hypothesized that portable MRI would detect widespread HIBI and no safety events would occur during portable MRI.

Methods

Study setting and participants

This study was performed at Yale New Haven Hospital in New Haven, Connecticut from September 2020 through January 2022 and was approved by the Yale University institutional review board (2000028136). On August 11, 2020 the Hyperfine™ (Guilford, CT) portable MRI device received US Food and Drug Administration clearance. Informed consent was waived, and images were acquired as part of clinical care.

We have an established clinical protocol for portable MRI in patients resuscitated from CA; inclusion criteria are age > 18 years, admission to an ICU after CA, and weight < 400 lbs.; exclusion criteria include the presence of electrical or ferromagnetic implants, electroencephalography monitoring with MRI incompatible leads, haemodynamic support with extracorporeal membrane oxygenation or an intra-aortic balloon pump, esophageal probe temperature monitoring, pregnancy, and inability to tolerate supine positioning for 30 minutes. The primary temperature sensing probe for targeted temperature management (TTM) at our institution is a bladder-sensing foley catheter, which is MRI-compatible. Given the low-field strength of portable MRI, life-saving ferromagnetic equipment did not need to be removed or shielded.

We retrospectively evaluated all consecutive portable MRIs performed as part of clinical care for CA patients. A low-field MRI scanner and operator were available during daytime hours. Portable MRI was interpreted by the neurointensivist involved in the patients' clinical care. Conventional neuroimaging was performed in all patients to confirm the findings of portable MRI. All portable MR images were

retrospectively reviewed by a blinded board-certified neuroradiologist (S.P.) for the presence or absence of HIBI and evolution in a subset of patients with more than one portable MRI.

Imaging parameters

All patients were scanned using a 64mT portable MRI scanner (Hyperfine™, Guilford, CT, USA) which was controlled using an electronic tablet (iPad Pro third generation). Throughout the course of this study five software version were used. Imaging parameters for each sequence, across software versions (RC8.1.0/RC8.1.1/RC8.2.0/RC8.3.0/8.3.0) (supplemental digital content). The low-field MRI operator was present throughout the duration of the scan and systematically documented any adverse events leading to early termination.

Region of interest selection

Low field MRI intrinsically has lower signal-to-noise ratio (SNR) compared to conventional MRI, degrading the quality of the images.¹² The diffusion-weighted imaging (DWI) sequence is most affected, rendering the T2/FLAIR sequence with superior sensitivity compared to DWI on low-field MRI.²⁴ To provide an objective measure of HIBI, we selected three regions of interest (ROI) and measured FLAIR signal intensity. The caudate, putamen, and thalamus were selected as ROIs given the frequency of involvement in HIBI and the ease in identifying these structures on low-field MRI. ROIs were drawn for the right and left caudate, putamen and thalamus using Analysis of Functional NeuroImages ((AFNI) 2020.1.01). Each ROI was drawn on T1 for better structural reference and then was adjusted on the FLAIR sequence to account for patient motion between sequences. In cases without a T1 sequence, ROIs were directly drawn on the FLAIR sequence. A single slice was chosen to represent each structure.

Statistical analysis

The mean FLAIR signal intensity for each individual ROI was calculated. For group calculations based on ROI, left and right regions were combined. Patients were divided in two groups, HIBI present versus absent, based on conventional neuroimaging report- regardless of severity. The data was analyzed in Matlab 2020b. A Kolmogorov-Smirnov test confirmed non-normative distribution of the data; therefore, a Kruskal-Wallis test was performed for each ROI to determine statistical differences between groups.

Results

Characteristics of the cardiac arrest cohort imaged with portable MRI

We obtained 22 portable MRI examinations for 19 patients with CA (13 male [68.4%]; mean [SD] age, 51.8 [13.1] years) (Table 1). All but one patient (patient 18) presented with out-of-hospital CA. All patients except one (patient 8), who progressed to brain death within 26 hours of resuscitation, were treated with TTM. Examinations were acquired at a median (interquartile range) of 78 (40–136) hours post-arrest. Fourteen (73.7%) patients had a conventional MRI performed within a median (interquartile range) of 105.5 (77–144) hours post-arrest. In ten (71.4%) patients, portable MRI preceded conventional MRI by median (interquartile range) 33 (4.5–58) hours. Three patients (Patient 2, 3, and 8) had a portable MRI performed within six hours of return of spontaneous circulation. Delay to conventional

Table 1 – Demographic, arrest related details, and outcomes of the cohort.

Patient	Age	Sex	Rhythm	Witnessed	Bystander CPR	Aetiology of arrest	Initial FOUR score	mRS at discharge
1	39	M	Non-Shockable	No	No	Substance abuse (cocaine)	5	5
2	35	F	Non-Shockable	Yes	Yes	Substance abuse (opiate)	0	6
3	64	M	Shockable	Yes	No	Cardiac	5	2
4	66	F	Shockable	No	No	Cardiac	3	6
5	71	F	Non-Shockable	No	No	Substance abuse (opiate)	0	6
6	52	M	Non-Shockable	No	No	Substance abuse (opiate)	0	6
7	47	F	Non-Shockable	Yes	Yes	Respiratory	2	5
8	44	M	Non-Shockable	No	No	Substance abuse (opiate)	0	6
9	57	M	Non-Shockable	No	No	Substance abuse (alcohol)	1	6
10	23	M	Non-Shockable	No	No	Hanging	3	3
11	34	F	Non-Shockable	No	No	Substance abuse (opiate)	0	6
12	36	F	Non-Shockable	No	No	Drowning	3	5
13	57	M	Non-Shockable	No	No	Cardiac	5	6
14	64	M	Non-Shockable	No	No	Respiratory	5	6
15	63	M	Shockable	Yes	Yes	Cardiac	3	1
16	57	M	Non-Shockable	No	No	Substance abuse (alcohol)	1	6
17	66	M	Shockable	No	No	Cardiac	4	3
18	51	M	Non-Shockable	Yes	Yes	Respiratory	10	4
19	58	M	Non-Shockable	No	No	Substance Abuse (opiate)	3	6

CPR: cardiopulmonary resuscitation.

FOUR: Full-Outline of Unresponsiveness.

mRS: modified Rankin Scale.

UNK: unknown because patient remains hospitalized at the time of this manuscript.

MRI (≥ 48 hours from order) occurred in 6 (42.9%) patients, due to nursing concerns regarding travel, need to complete TTM therapy prior to MRI, or radiology scheduling. Eight (42.1%) patients had portable MRI performed during TTM and there were no interruptions to TTM treatment. Conventional MRI was not obtained in five patients due to progression to brain death [4 (80%)] or placement of an implantable cardioverter-defibrillator [1 (20.0%), placed after portable MRI performed]. Eighteen (94.7%) patients remained on the ventilator at the time of portable MRI, with a mean [standard deviation] fraction of inspired oxygen of 54 [26.7]% and positive end expiratory pressure of 5.7 [1.58]. Eight (42.1%) patients were on one or more vasopressors and 13 (68.4%) patients were on two or more continuous infusions (predominantly sedatives) at the time of portable MRI. There were no adverse events or complications associated with portable MRI scanning. No patients were disconnected from ICU monitoring equipment, including cooling gel pads for TTM and infusion pumps, and no examinations were aborted early.

In four patients (28.6%), portable MRI was performed 75 (27–312) hours after conventional MRI. Portable MRI was performed five days after conventional MRI in patient 4 who had prolonged myoclonic status epilepticus but normal conventional MRI on post-arrest day 4. Portable MRI confirmed no evidence of HIBI. Portable MRI was performed in patient 5 one day after conventional MRI due to an exam change (newly fixed and dilated pupils). Portable MRI did not show evidence of herniation, however, family opted for transition to comfort measures. In patient 7, portable MRI was performed 21 days after conventional MRI when neurology was re-consulted due to persistently poor neurologic examination. No new findings were identified. In patient 10, portable MRI was performed one day after conventional MRI due to the development of paroxysmal sympathetic hyperactivity. No new findings were identified. Three patients (2, 3, 10) were imaged at two sequential time-points (post-arrest days 0 and 2, 0 and 6, and 3 and 9, respectively). Portable MRI

was repeated for loss of brainstem reflexes in patient 2, development of altered mental status after extubation in patient 3, and to evaluate worsening paroxysmal sympathetic hyperactivity in patient 10. Repeat portable MRI in patient 2 and 10 showed progression of HIBI (Fig. 1) while no new structural cause was found in patient 3 with altered mental status, later diagnosed with delirium.

Imaging parameters

Of the 22 total scans, all 22 completed FLAIR, 21 completed DWI/ Apparent Diffusion Coefficient (ADC), and 20 completed both T1 and T2 imaging sequences. The total acquisition time for all sequences ranged from 29 min and 16 seconds to 31 minutes and 22 seconds.

Imaging findings

Twelve patients (63.2%) had evidence of HIBI by conventional MRI neuroradiology reports. All were identified as having HIBI on portable MRI. HIBI was over-called on portable MRI in two (28.6%) patients without evidence of HIBI on conventional MRI neuroradiology report (patient 5 and 14). These two patients had portable MRI performed within 24 hours of conventional MRI, but had prominent periventricular and subcortical FLAIR signal abnormalities on conventional MRI, suggestive of microvascular ischemic disease. DWI abnormalities were apparent in patients with widespread HIBI (Fig. 2), however, FLAIR signal hyperintensity was superior for visualization of global and focal ischaemia (Fig. 3). Portable MRI detected obstructive hydrocephalus in one patient (patient 19) who also had widespread HIBI. In this patient, hydrocephalus was likely the result of HIBI in the cerebellum (Fig. 4). FLAIR signal intensity was higher in the caudate (mean 11067.3 SD [2612.2] vs 14495.0[4666.9]), putamen (10341.7[2413.6] vs 14970.6[5011.0]), and thalamus (9699.1[2483.6] vs 12753.8[3683.8]) amongst patients reported to have HIBI on the conventional MRI report ($p < 0.05$, Fig. 5).

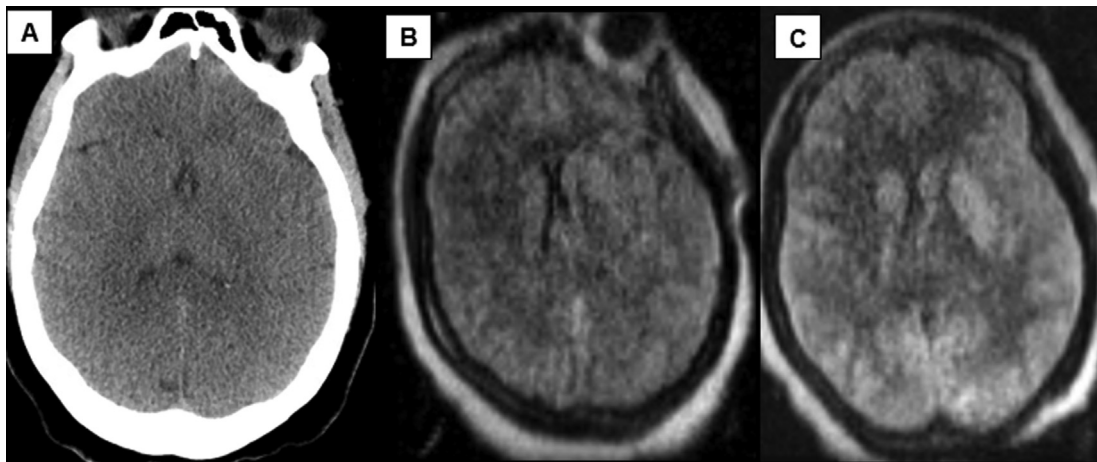


Fig. 1 – Sequential neuroimaging in patient 2, a 35-year-old F who presented after a witnessed non-shockable rhythm cardiac arrest secondary to drug overdose. Initial HCT (A) was performed 5 hours after CA and showed diffuse loss of gray-white differentiation and cerebral edema, portable MRI (FLAIR sequence) was obtained at 4 hours (B) and 53 hours (C) after CA. Over the course of 49 hours, FLAIR hyperintensities became apparent in the bilateral deep and cortical regions. This patient was declared brain-dead 74-hours after CA.

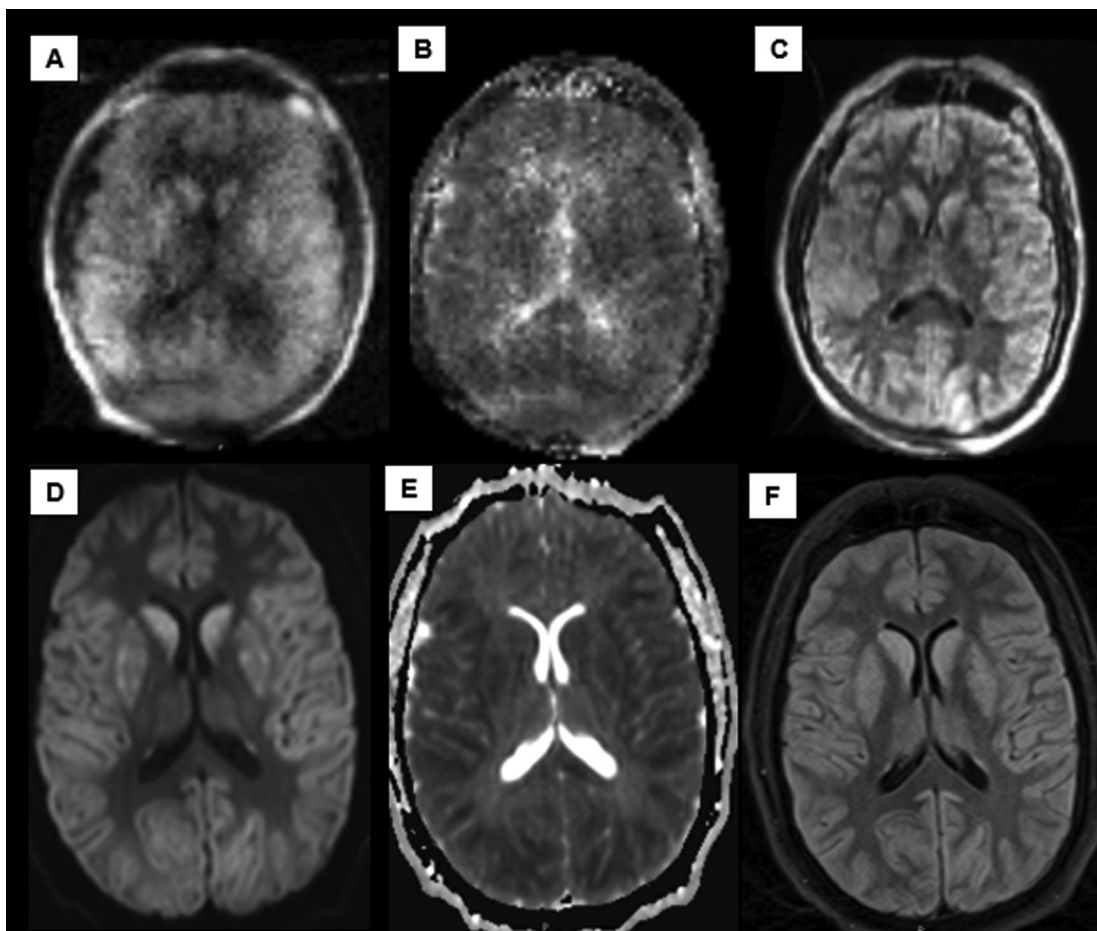


Fig. 2 – Comparison of DWI (A, D), ADC (B, E), and FLAIR (C, F) sequences in patient 6, a 52-year-old M who presented after an unwitnessed non-shockable rhythm cardiac arrest secondary to drug overdose. Portable MRI (A-C) was obtained 49 hours after CA, conventional MRI (D-F) was obtained 79 hours after CA.

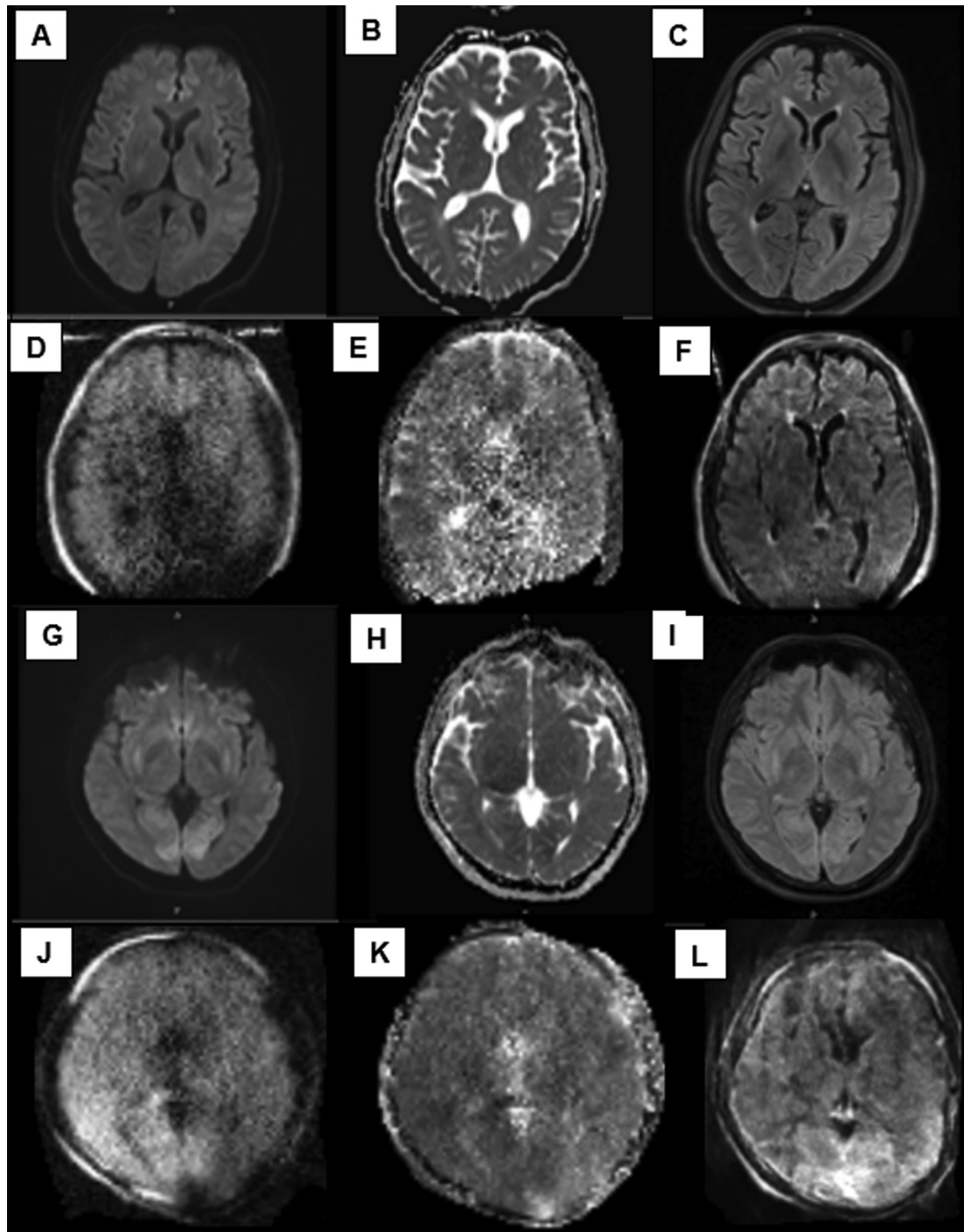


Fig. 3 – Comparison of conventional MRI [DWI (A, G), ADC (B, H), and FLAIR (C, I)] and portable MRI [DWI (D, J), ADC (E, K), and FLAIR (F, L)] in two patients. Patient 15 (A–F), a 63-year-old M who presented after a witnessed, shockable rhythm arrest with immediate bystander CPR secondary to myocardial infarction. No evidence of HIBI was seen on portable MRI (D–F), performed 96 hours after CA or on conventional MRI (A–C), performed 190 hours after CA. Patient 10 (G–L), a 23-year-old M who presented after an unwitnessed, non-shockable rhythm arrest secondary to suicide attempt by hanging. Focal findings consistent with HIBI in the bilateral occipital lobes were identified on portable MRI (J–L), performed 204 hours after CA and on conventional MRI (G–I), performed 225 hours after CA.

Discussion

We report the successful deployment of a novel and scalable bedside approach to evaluating brain injury after cardiac arrest. We

describe the imaging characteristics of HIBI on portable, low-field MRI. Neuroimaging is a highly valued prognostic tool after CA, however, there is a discrepancy between the perceived importance of brain MRI and the frequency of use.^{11,25} Large populations of

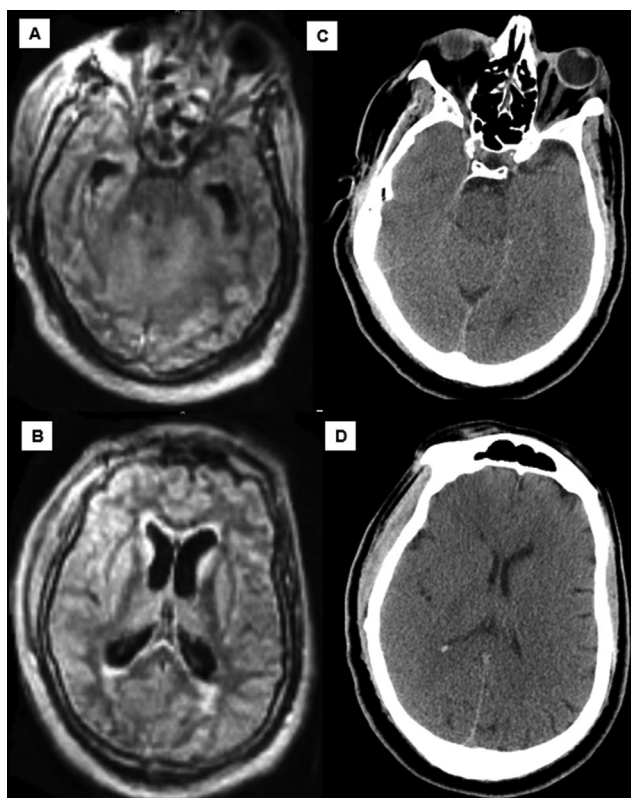


Fig. 4 – New onset hydrocephalus in patient 19, a 58-year-old M who presented after unwitnessed non-shockable rhythm cardiac arrest due to drug overdose. Initial HCT (C, D) was performed 2 hours after CA and showed diffuse loss of grey-white matter differentiation and small ventricles. Portable MRI (A, B) was performed 47 hours after CA and shows ventriculomegaly and diffuse FLAIR signal abnormalities consistent with HIBI in the bilateral cortex, deep structures, and cerebellum.

critically ill CA patients are precluded from conventional MRI due to hospital resources or the inability to safely travel.^{21,26} Critical events in ICU patients during transport are common, occurring at a rate of 1.6 events per patient transport.²⁷ Major critical events are related to haemodynamic instability, hypoxia, and other airway complications, all of which can contribute to secondary brain injury or pose life-threatening consequences given the high-risk for recurrent arrest in this population.^{27,28}

Conventional MRI is performed in 22–40% of the comatose post-CA population and timing is often delayed by many patient- and systems-related factors.^{29,30} Advances in low-magnetic field MRI have allowed for a paradigm shift, obtaining clinically useful neuroimaging at the bedside.^{23,31} We show that portable MRI can detect HIBI seen on conventional MRI. In our study, HIBI is most easily appreciated on the FLAIR sequence, which we hypothesize is due to the low SNR of portable DWI sequences.²⁴ Limitations in conventional signal processing and image reconstruction are being overcome by supervised and unsupervised machine learning approaches that facilitate improved SNR. For example, Automated Transform by Manifold Approximation (AUTOMAP) uses machine

learning and deep neural networks to find the best computational strategies to produce images with improved SNR.^{32,33} These ongoing advancements in low-field MRI technology are likely to improve image quality. Due to the improved quality of T2/FLAIR sequences for identifying HIBI on portable MRI, we chose to measure FLAIR signal intensity for a quantitative approach. FLAIR signal intensity was higher in patients with HIBI in the caudate, putamen, and thalamus and may provide a promising target to develop objective measures of brain injury using portable MRI.

Neuroimaging is time-sensitive after CA; DWI and T2 changes evolve, and guidelines recommend performing a brain MRI between two and six days after CA.^{34–36} After CA, the initial ischemic event is followed by secondary energy failure, resulting in ongoing brain ischaemia. Serial MRI in animal models of cerebral ischaemia found acute reductions in ADC during hypoperfusion, which partially or completely recovered following reperfusion.³⁷ Secondary ADC reductions, which are accompanied by T2 elevations and histological damage, are observed as more time elapses.³⁷ Increased T2 signal on MRI corresponds to histopathologic findings such as rarefaction of tissue, neuronal death, and macrophage infiltration throughout the severely injured parenchyma.³⁸ Furthermore, increased T2 signal after reperfusion is able to predict secondary deterioration in ADC values.³⁹ While the mechanisms responsible for both the time-sensitive and regional distribution of HIBI on MRI have not been fully characterized, the presence of vasogenic, ionic, and cytotoxic edema occurring at different timelines after HIBI are all likely to contribute.^{39,40} Early T2 MRI may help to predict reperfusion injury and reveal the timeline of secondary brain injury mechanisms.

Risks associated with transport out of the ICU makes conventional MRI impractical and unsafe in a subset of post-CA patients. For this reason, selection bias has influenced all studies evaluating MRIs prognostic performance.⁸ Advances in successful post-CA care have led to a growing population of comatose survivors. Accurate outcome prediction is critical to avoiding withdrawal of life sustaining therapy in patients with the potential to achieve good functional outcomes. We show that portable MRI is feasible and safe in critically ill patients immediately after resuscitation. Furthermore, serial MR imaging may help to elucidate the mechanisms involved in secondary deterioration and quantitative analyses using FLAIR intensity may help to provide objective thresholds.

The main limitations of this study are the single-center and retrospective nature, limited number of patients imaged, and lack of objective measures used to define HIBI. Our results are subject to selection bias, as this is a new technology and physicians may have chosen to proceed with portable MRI in only a subset of eligible patients. Prospective multicenter studies designed to validate the findings of HIBI on portable MRI, compared to conventional neuroimaging, are critical.

Conclusions

Low-field MRI provides clinically useful images without impeding critical care management and can successfully identify patients with HIBI. Future studies to assess the evolution of MRI findings after CA, at fixed time points, can provide important insights into the temporal nature of HIBI and pathophysiologic mechanisms involved in secondary brain injury and neurodeterioration.

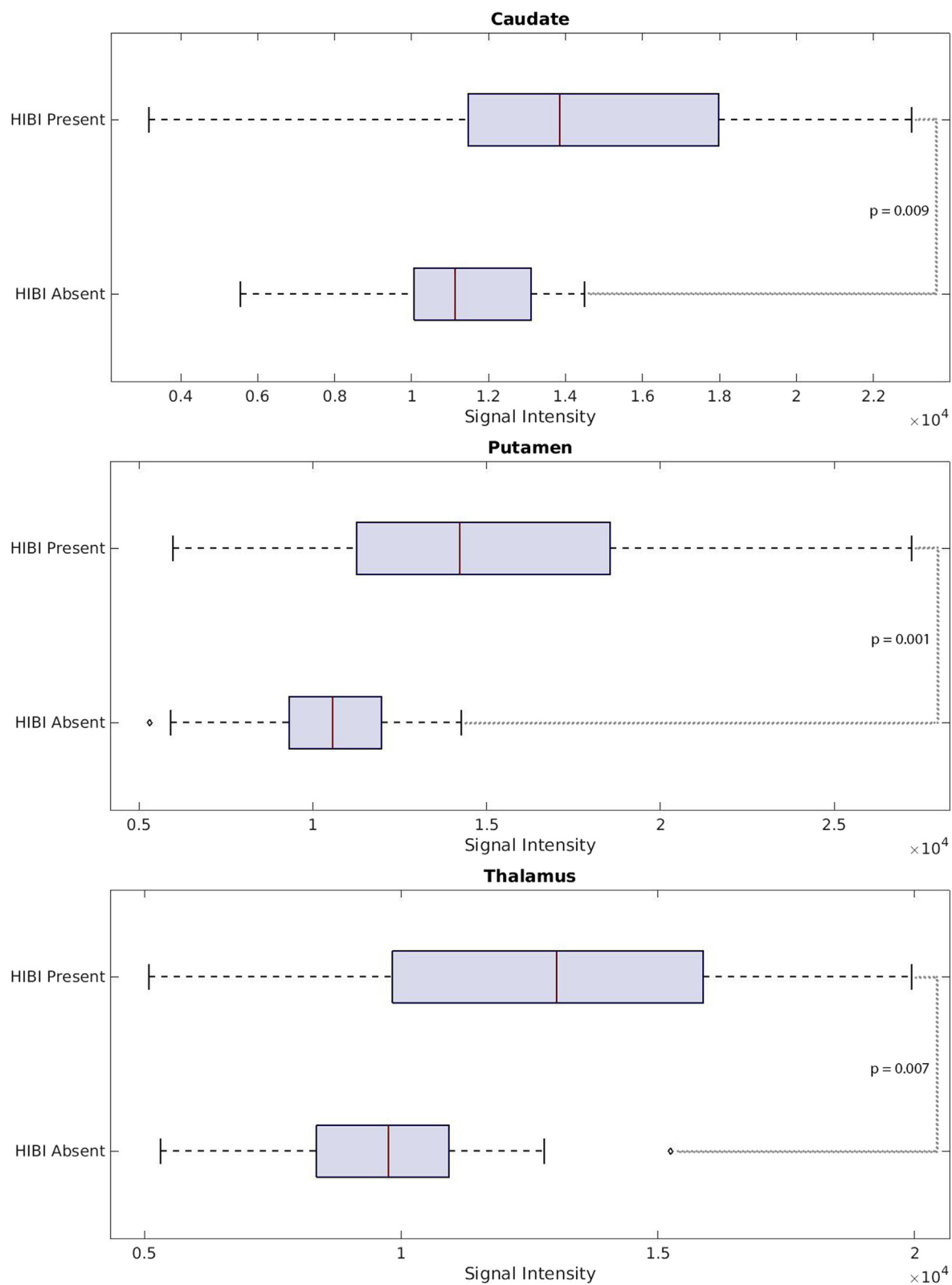


Fig. 5 – Box plot for FLAIR signal hyperintensity values on portable MRI in patients with or without HIBI on conventional neuroimaging.

CRediT authorship contribution statement

Rachel Beekman: Conceptualization, Methodology, Investigation, Project administration. **Anna Crawford:** Methodology, Investigation, Writing – review & editing. **Mercy H. Mazurek:** Investigation, Writing – review & editing. **Anjali M. Prabhat:** Investigation, Writing – review & editing. **Isha R. Chavva:** Investigation, Writing – review & editing. **Nethra Parasuram:** Investigation, Writing – review & editing. **Noah Kim:** Investigation, Writing – review & editing. **Jennifer A. Kim:** Investigation, Writing – review & editing. **Nils Petersen:** Investigation, Writing – review & editing. **Adam de Havenon:** Investigation, Writing – review & editing. **Akhil Khosla:** Investigation, Writing – review & editing. **Shyoko Honiden:** Investigation, Writing – review & editing. **P. Elliott Miller:** Investigation, Writing – review & editing. **Charles Wira:** Investigation, Writing – review & editing. **James Daley:** Investigation, Writing – review & editing. **Syedmehtdi Payabvash:** Methodology, Writing – review & editing. **David M. Greer:** Writing – review & editing. **Emily J. Gilmore:** Conceptualization, Investigation, Methodology, Writing – review & editing. **W. Taylor Kimberly:** Writing – review & editing. **Kevin N. Sheth:** Conceptualization, Methodology, Investigation, Writing – review & editing, Supervision, Project administration, Funding acquisition.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Dr. Rachel Beekman reports no disclosures. Anna Crawford reports no disclosures. Mercy H. Mazurek reports no disclosures. Anjali M. Prabhat reports no disclosures. Isha R. Chavva reports no disclosures. Nethra Parasuram reports no disclosures. Noah Kim reports no disclosures. Dr. Jennifer A. Kim reports no disclosures. Dr. Nils Petersen reports no disclosures. Dr. Adam de Havenon reports no disclosures. Dr. Akhil Khosla reports no disclosures. Dr. Shyoko Honiden reports no disclosures. Dr. P. Elliott Miller reports no disclosures. Dr. Charles Wira reports no disclosures. Dr. James Daley reports no disclosures. Dr. Syedmehtdi Payabvash reports no disclosures. Dr. David M. Greer reports no disclosures. Dr. Emily J. Gilmore reports no disclosures. Dr. W. Taylor Kimberly receives grants from the NIH and AHA to support his work with portable MRI. Dr. Kevin N. Sheth receives support from the Collaborative Science Award (American Heart Association), National Institutes of Health Supplement Grant (U01NS106513-S1), and Hyperfine Research, Inc. research grant for his work with portable MRI.

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Appendix A. Supplementary material

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