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Computed tomography perfusion assessment of poor neurological outcome in comatose cardiac arrest patients (CANCCAP): a prospective study

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Abstract

Background Computed tomography perfusion (CTP) of the brain, are increasingly being employed for the assessment of critically ill patients admitted to intensive care units (ICU), including comatose cardiac arrest patients (CCAP). The purpose of our study was to validate the use of CTP in predicting in-hospital mortality in CCAPs.

Method This prospective cohort study enrolled newly admitted adult CCAP, with an out of hospital cardiac arrest (OHCA) and were scheduled for admission to the ICU for further management. Just before ICU admission, CCAP underwent a routine CT scan of the head and CTP of whole head. The treating physicians remained blinded to the CTP results and all patients received standard management. The CTP maps were evaluated to determine a binary outcome of non-survivable brain injury (NSBI), by two independent neuroradiologists, blinded to each other's assessment and to the clinical history of the patients.

Results A total of 91 patients were enrolled and 90 (Male-78; mean age-62 years) were included in the final analysis. One patient declined consent. Of these, 42 individuals (47%) had in-hospital mortality. Patients with in-hospital mortality were older; had higher levels of creatinine, blood urea nitrogen, blood CO_2 and lower pH, carbonate, and heart rate. In multivariate analysis, PCI was independently associated with reduction in-hospital mortality. CTP demonstrated exceptionally high specificity (100%; 95% CI 92–100%) and positive predictive value (100%; 95%CI 6.3–100%) for the prediction of NSBI. For CTP, Bennet's S-score showed excellent agreement between the two readers (s = 0.82–0.95).

Conclusion CTP was safe and demonstrated very high specificity and positive predictive value and may be used as an additional diagnostic tool for identifying patients at high risk of in-hospital mortality.

Keywords CT perfusion, Comatosed cardiac arrest patients, Cardiac arrest, In-hospital mortality, Neurological outcome

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Introduction

Approximately 85% of patients experience cardiac arrests outside of a hospital, a condition referred to as out of hospital cardiac arrest (OHCA). It is estimated that only 5% of Canadians suffering OHCA survive [1]. Patients who do survive OHCA often present at the hospital in a comatose state. Advances in resuscitation and post-resuscitation care including temperature management have led to improvements in both survival and neurological outcomes of cardiac arrest patients [2, 3]. Nevertheless the mortality rate remains over 50% among comatose cardiac arrest patients (CCAP) who survive to intensive care unit (ICU) admission [4]. Moreover, many survivors experience poor cognitive and neurologic function defined as a score of ≥ 4 on the modified Rankin scale (mRs), indicating a need for assistance with basic bodily functions or death [4, 5].

Many CCAP arriving at the hospital may already exhibit severe and potentially fatal brain injury. Prolonged cardiac arrest carries significantly graver consequences for the brain compared to the rest of the body [6]. Resuscitation, following cardiac arrest, is rarely successful beyond 20 min with hypoxic-ischemic brain injury a major contributor to morbidity and mortality [1, 7]. To assess the severity of brain injury, clinicians typically conduct serial comprehensive neurological examinations. However, these assessments have shown limited reliability predicting outcomes during the initial days following cardiac arrest [8]. Moreover, the use of sedatives and paralytics during temperature management further complicates clinical evaluations. Consequently, CCAP receive resource-intensive therapies, even thought up to half may have already sustained severe, non-survivable brain injury before hospital admission [4]. In the absence of a clear prognosis, these patients may undergo treatments that provide minimal benefit. Therefore, a pivotal question in the treatment of these patients arises: "Which CCAP will benefit the most from treatment?" [9].

Ancillary imaging tests, particularly computed tomography perfusion (CTP) of the brain, are increasingly being employed for the assessment of critically ill patients admitted to ICU, including CCAP [10–18]. Despite promising results from pilot studies regarding the utility of CTP in predicting poor neurological outcomes in CCAP, there has been a lack of prospective, well-powered studies to validate these findings [12]. The purpose of our study was to validate the early use of CTP in predicting in-hospital mortality in CCAPs.

Methods

This prospective cohort study was approved by our institutional research ethics board (REB number-HS23646 B2020:017) and was registered on ClinicalTrials.gov (clinical trial registration number: NCT04323020; registration date-March 17, 2020). The study protocol has been previously published [13].

Participants

Our study enrolled newly admitted CCAP, defined as adults aged 18 years or older, who experienced an OHCA and were scheduled for admission to the ICU for further management. Patients were excluded if deferred consent could not be obtained from either the patient or a substitute decision maker, or if consent was subsequently withdrawn. Deferred consent, approved by our research ethics board, was obtained within one week of hospital admission. Additionally, individuals with known pregnancy; documented contraindications to CT contrast agents (such as a history of allergy or anaphylactic reaction), or known chronic kidney disease, stage 4–5 (eGFR < 30 mL/min/1.73 m^2) were excluded from the study.

Imaging

Just prior to admission to ICU, CCAP underwent a routine non-contrast CT scan of the brain and CTP of whole brain. All patients received the standard institutional care, with the exception of the additional CTP scan. The CTP images were acquired following a standardized stroke imaging protocol ensuring whole brain coverage [12, 14, 16, 19]. The CTP results were not disclosed to the treating physicians and patient care proceeded according to local practice.

The CTP analysis was performed in the imaging core lab using a semiautomatic deconvolution algorithm implemented on a vendor-neutral software package (Oleasphere 32). Qualitative assessment of CTP primarily followed standard clinical practice. For this assessment, non-survivable brain injury was characterized by a concurrent decrease in cerebral blood flow (CBF) and cerebral blood volume (CBV) within the brainstem (Fig. 1 and Fig. 2). Perfusion maps for CBF and CBV were evaluated to determine a binary outcome of 'non-survivable' or 'survivable', in accordance with previously published methodologies [2, 20]. For quantitative analysis, non-survivable brain injury was defined as CBF < 10 mL/100 g/min and CBV < 2 mL/100 g within the brainstem. Two independent neuroradiologists, blinded to each other's assessment and to the clinical history of the patients, evaluated the perfusion maps. In case of disagreements, consensus was reached through discussion to achieve a final analysis. This consensus decision reflects the practical challenges encountered in real-life scenarios.

Non-contrast CT head and CT angiogram (CTA) images derived from the source images of CTP were also subjected to analysis. The non-contrast CT head was

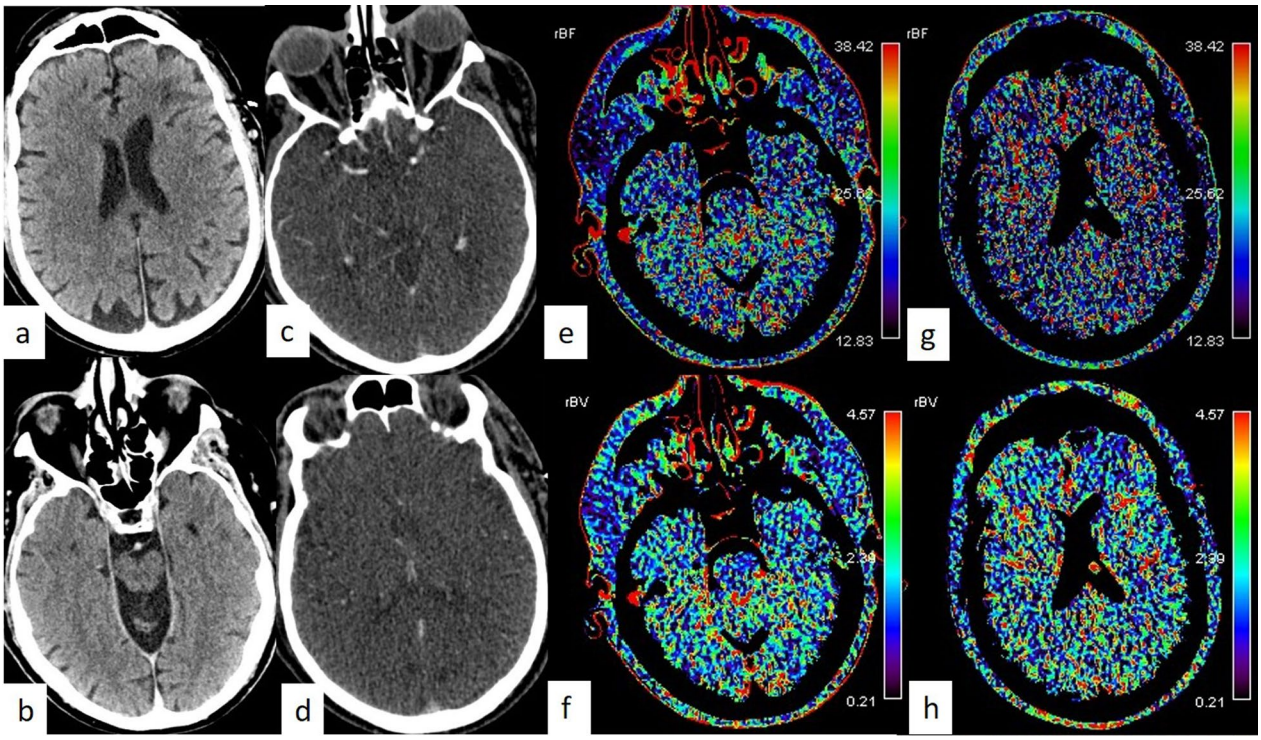

Fig. 1 Non-contrast CT images for a comatose cardiac arrest patient in his 70 s demonstrated preserved grey-white matter differentiation (**a** and **b**). CT angiography revealed preserved filling of the distal branches of anterior, middle, and posterior cerebral arteries (**c** and **d**) as well as opacification of the internal cerebral veins (**d**). Coloured perfusion maps indicate severely reduced or absent cerebral blood flow (**e** and **g**) and cerebral blood volume (**f** and **h**) in the brainstem, consistent with CT perfusion features of non-survival brain injury

examined to document the presence of diffuse ischemic changes or any other incidental findings. CTA images were evaluated using 4-, 7- and 10- point scale to assess possibility of non-survivable brain injury [21].

Outcome

Clinical, imaging, and laboratory data from CCAP were collected throughout their hospitalization using the RED-Cap platform. Primary outcome was a binary outcome of in-hospital mortality. The proportion of participants exhibiting CTP features of non-survivable brain injury upon presentation was recorded. Functional outcome was recorded using the modified Rankin Score (mRs) at hospital discharge and at 6 months post-discharge for those who survived their index hospital stay.

The enrolment process for our study was significantly delayed by the COVID-19 pandemic, which resulted in limited research activity within our hospital, mirroring the situation worldwide. To evaluate the impact of this delay on our recruited patient population, we compiled denominator data encompassing all CCAP transferred to our hospital during the study period. This population was then compared with the recruited patient cohort to identify any significant differences in terms of basic demographics.

Sample size calculation and statistical analyses

The sample size was determined based on findings from a pilot study of CTP [12], which demonstrated a specificity and positive predictive value of 100% along with a sensitivity of 37.5% and a negative predictive value of 28.6% for death at hospital discharge. Utilizing Buderer's formula [22], a sample size of 75 CCAPs was calculated assuming a prevalence of poor clinical outcome of at least 50%, to validate the use of CTP features of non-survivable brain injury against the clinical outcome of death. This sample size was anticipated to achieve a sensitivity and specificity of 97.5%, a confidence interval of 5% (± 2.5% around the point estimate). To accommodate for potential drop-out rate of up to 20% (inclusive of technical issues with CTP acquisition, protocol violation, consent withdrawals, or new contraindications for CTP), the sample size was increased to a total of 90 CCAPs.

Diagnostic validity analysis was conducted by calculating sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). The reference standard used was clinical outcome on mRS at

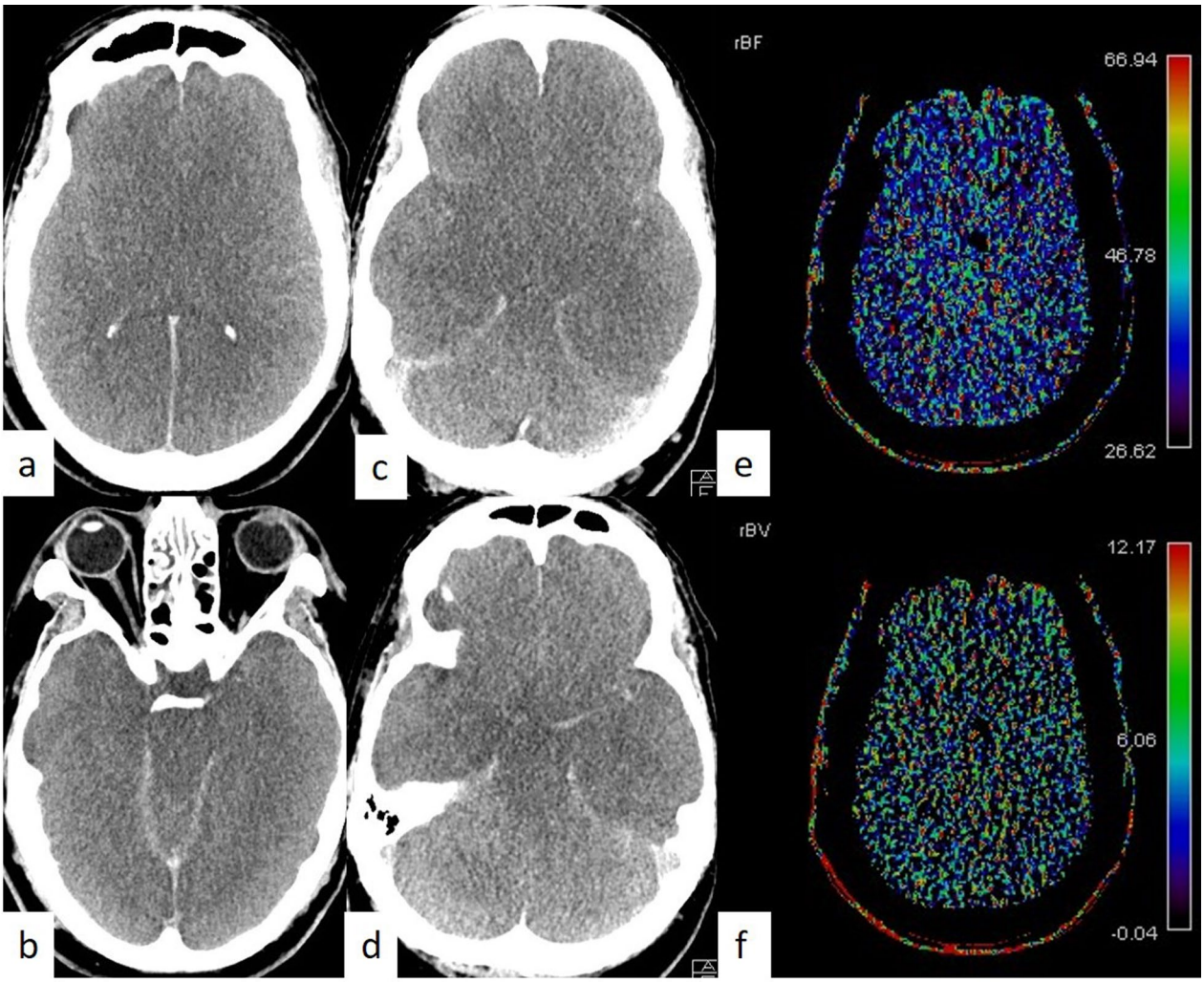

Fig. 2 Non-contrast CT images of a comatose cardiac arrest patient in his 30 s showing diffuse loss of grey-white matter differentiation (**a** and **b**). CT angiogram showed only minimal filling of the proximal M1 branches of middle cerebral arteries (**c** and **d**), with no visible filling of the cortical branches of middle meningeal artery or any branches of anterior or posterior cerebral arteries. Coloured perfusion maps reveal markedly reduced or absent cerebral blood flow (**e**) and cerebral blood volume (**f**) consistent with CT perfusion findings of non-survival brain injury

hospital discharge. Sensitivity was defined as the ability of CTP to correctly classify an individual as deceased, while specificity represented the ability of CTP to correctly classify an individual as not deceased. PPV was calculated as the percentage of patients exhibiting CTP features of non-survivable brain injury who ultimately passed away by the end of their hospital stay. NPV was determined as the percentage of patients without CTP features of non-survivable brain injury on CTP who survived until the end of their hospital stay. To facilitate comparison between CTP quantitative values and clinical evaluation, the area under the receiver operating characteristic (ROC) curve was computed. Similar validity analysis was conducted for CT Angiogram. Inter-observer agreement between the two neuroradiologists were assessed to evaluate the reliability of CTP.

Results

Between July 2021 to June 2023, a total of 91 patients were enrolled and 90 (Male-78; mean age-62 years) were included in the final analysis (Table 1). One patient declined consent and was consequently not included in the study cohort. Among our patient cohort, 42 individuals (47%) did not survive their hospital index stay. Following hospital discharge, with a median follow-up 227.5 days (range: 13–477 days), 6 (6.7%) additional patients died.

Table 1 Demographic and clinical characteristics of patients enrolled in the CANCCAP study

	Total N = 90	**Death N = 42**	**Survival N = 48**	***P*-value**
Age mean (SD)	62.2 (13.9)	66.7 (12.4)	58.3 (14.1)	0.003
Female n (%)	12 (100)	5 (41.7)	7/47 (58.3)	0.876
Race n (%)				
Asian	7 (100)	5 (71.4)	2 (28.6)	0.414
Black or African American	1 (100)	0 (0)	1 (100)	
Caucasian	73 (100)	32 (43.8)	41 (56.2)	
First nations	4 (100)	3 (75.0)	1 (25.0)	
Unknown	1 (100)	0 (0)	1 (100)	
Other	4 (100)	2 (50.0)	2 (50.0)	
Injury to CT scan				
Median (IQR1-3) (minutes)	259.5 (209.0–403.3)	260.5 (204.5–364.5)	258.0 (212.8–535.0)	0.365
Length of hospital stay, median (days)	8	5	14	< 0.001
Length of ICU stay, median (days)	5	5	5	0.437
Cause of cardiac arrest n (%)				
Arrhythmia	90 (100)	42 (46.7)	48 (53.3)	
Weight (kg) at ICU admission				
Mean (SD)	(89) 85.4 (19.5)	(41) 87.7 (22.6)	83.4 (16.6)	0.309
Height (cm) at ICU admission mean (SD)	(87) 172.2 (19.6)	(40) 172.8 (13.3)	(47) 171.6 (23.9)	0.767
STEMI n (%)	42 (100)	14 (0.33)	28 (0.67)	0.018
PCI n (%)	65 (100)	22 (0.34)	43 (0.66)	< 0.001
Comorbidities n (%)				
Hypertension	48 (100)	26 (54.2)	22 (45.8)	0.127
Diabetes	21 (100)	14 (66.7)	7 (33.3)	0.036
Coronary artery disease	30 (100)	17 (56.7)	13 (43.3)	0.179
Peripheral vascular disease	2 (100)	0	2 (100)	
Previous stroke	3 (100)	2 (66.7)	1 (33.3)	0.480
Active smoking	22 (100)	8 36.4)	14 (63.6)	0.056
Smoking history (known past history)	18 (100)	12 (66.7)	6 (33.3)	
Chronic renal failure with dialysis	0	0	0	
Chronic renal failure without dialysis	6 (100)	6 (100)	0	
Other comorbidities	73 (100)	35 (47.9)	38 (52.1)	0.614

ICU, intensive care unit; IQR, inter-quartile range; SD, standard deviation; STEMI, ST elevation myocardial infarction; PCI, percutaneous coronary intervention

Patients who survived the hospital admission were notably younger (58.3 years vs. 66.7 years, $p = 0.003$) compared to those who experienced in-hospital mortality. Additionally, survivors were also more likely to have presented with ST elevation myocardial infarction, STEMI (28% vs. 14%, $p = 0.018$) and to have undergone percutaneous coronary intervention (PCI) (43% vs. 22%, $p < 0.001$) (Table 1). Among those who died in the hospital, there were significantly higher levels of creatinine (122.7 vs. 99.7 µmol/L, $p = 0.004$), blood urea nitrogen (8.4 vs. 6.4 mmol/L, $p = 0.011$), blood CO2 (65.4 vs. 53.6 mmHg, $p = 0.003$) and lower pH (7.1 vs. 7.2, $p = 0.001$), carbonate (15.4 vs. 18.1 mmol/L, $p = 0.001$), and heart rate (83.3 vs. 93.1 beats per minute, $p = 0.041$) compared to those who survived the hospital stay (Table 2).

The multivariate analysis encompassed age (on a continuous variable and scaled by 10 years), sex, pH, blood CO_2 level, STEMI, PCI, and eGFR (Table 1S). Notably, only PCI remained independently associated with a significant reduction in the odds of in-hospital mortality (odds ratio 0.10, $p = 0.004$), underscoring its critical role in patient outcomes.

CTP done at hospital admission demonstrated high specificity (100%; 95% CI 92–100%) and positive predictive value (100%; 95%CI 6.3–100.0%) for the prediction of non-survivable brain injury (Table 3 and Table 2S). This indicates that CTP accurately identified

Table 2 Initial baseline biologic variables at admission between those with and without in-hospital mortality

Biologic parameter	Total N = 90 Mean (SD)	Death N = 42 Mean (SD)	Survival N = 48 Mean (SD)	*P*-value
Blood glucose (mmol/L)	15.9 (11.6)	16.3 (7.1)	15.6 (14.4)	0.778
Hemoglobin (g/L)	135.7 (22.8)	131.5 (25.6)	139.3 (17.2)	0.091
Na+ (mmol/L)	138.2 (4.3)	138.1 (4.3)	138.3 (4.4)	0.855
K+ (mmol/L)	4.0 (0.9)	4.1 (0.9)	3.9 (0.9)	0.349
Creatinine (µmol/L)	110.4 (38.3)	122.7 (47.4)	99.7 (23.8)	0.004
BUN (mmol/L)	7.3 (3.7)	8.4 (4.8)	6.4 (2.0)	0.011
eGFR	62.9 (18.7)	63.5 (20.1)	61.9 (17.4)	0.698
Baseline pH	7.2 (0.1)	7.1 (0.2)	7.2 (0.1)	0.001
HCO_3 (mmol/L)	16.9 (4.2)	15.4 (4.0)	18.1 (3.9)	0.001
PCO_2 (mmHg)	59.1 (19.2)	65.4 (22.4)	53.6 (13.8)	0.003
PaO_2 (mmHg)	66.9 (48.3)	63.0 (40.5)	70.4 (54.4)	0.475
FiO_2 (%)	(52) 69.8 (28.8)	(24) 77.8 (27.2)	(28) 63.0 (28.9)	0.063
Vital signs				
Systolic BP (mmHg)	120.8 (34.3)	120.6 (33.5)	121.0 (35.4)	0.966
Diastolic BP (mmHg)	76.9 (22.0)	76.0 (22.4)	77.6 (21.8)	0.739
MAP (mmHg)	91.3 (24.9)	91.1 (24.7)	91.4 (25.3)	0.952
Heart rate (bpm)	(88) 88.4 (22.5)	83.3 (21.2)	(46) 93.1 (22.8)	0.041
Body temperature (°C)	(41) 36.0 (1.0)	(16) 35.7 (1.3)	(25) 36.2 (0.8)	0.141

SD, standard deviation; BP, blood pressure; MAP, mean arterial pressure

Table 3 Diagnostic performance of qualitative assessment using non-contrast CT, CT angiogram and CT perfusion in identifying non-survivable brain injury and distinguishing between patients with and without in-hospital mortality

Criteria	SE	95% CI	SP	95% CI	Accuracy	95% CI	PPV	95% CI	NPV	95% CI	AUC
CTP											
Brainstem	19.0	9.0; 34.0	100	92.0; 100	61.4	51.1; 71.6	100	63.0; 100	57.0	46.0; 68.0	60.0
Isolated brainstem	19.0	9.0; 34.0	100	92.0; 100	61.4	51.1; 71.6	100	63.0; 100	57.0	46.0; 68.0	60.0
Whole Brain	2.0	0; 13.0	100	92.0; 100	53.4	44.3; 63.6	100	3.0; 100	53.0	42.0; 64.0	51.0
CTA											
4 points, peak phase	0	0; 8.0	100	93.0; 100	53.3	0	0	0	53.0	43.0; 64.0	50.0
7 points, peak phase	0	0; 8.0	100	93.0; 100	53.3	0	0	0	53.0	43.0; 64.0	50.0
10 points, peak phase	0	0; 8.0	100	93.0; 100	53.3	0	0	0	53.0	43.0; 64.0	50.0
4 points, late phase	0	0; 8.0	98.0	89.0; 100	52.2	37.8; 57.8	0	0; 97.0	53.0	42.0; 63.0	49.0
7 points, late phase	0	0; 8.0	98.0	89.0; 100	52.2	37.8; 57.8	0	0; 97.0	53.0	42.0; 63.0	49.0
10 points, late phase	0	0; 8.0	98.0	89.0; 100	52.2	37.8; 57.8	0	0; 97.0	53.0	42.0; 63.0	49.0
Non-Contrast CT Brain	17.0	7.0; 32.0	100	92.0; 100	60.9	51.7; 71.3	100	59.0; 100	57.0	46.0; 68.0	59.0

CI, confidence interval; CTP, computed Tomography perfusion; CTA, computed tomography angiography

patients who survived the severe brain injury (specificity) in the absence of non-survivable brain injury. When CTP features of non-survivable brain injury were present, none of them survived the hospital stay (positive predictive value). Incidentally, non-contrast CT scan was also found to have a high specificity (100%; 95% CI 92–100%) and positive predictive value (100%; 95%CI 59.0–100.0%) for the prediction of non-survivable brain injury, with slightly lower accuracy (Table 3).

On whole brain assessment, 2.3% of patients had features of non-survivable brain injury. On brainstem assessment, 13.6% of patients had features of non-survivable brain injury. Only 13.6% of patients were found

to have CTP features reflective of isolated brainstem features of non-survivable brain injury.

For CTAs, 4-, 7- and 10-point scales exhibited high specificity but very low PPV and sensitivity as no survivors met the CTA criteria for a non-survivable brain injury. Consequently, the NPV and accuracy value were also low for CTA scales.

For CTP, there was more than 90% agreement between the two neuroradiologists. Bennett's S-score [23], which adjusts for random disagreement, showed excellent agreement between the two readers (s = 0.82–0.95). For CTAs, between 85 and 94% agreement was seen between the two neuroradiologists. Bennett's S-score also showed excellent agreement (s = 0.71–0.89) between the readers, but was lower compared that for CTP.

CTP was found to be safe as there were no complications associated with CTP acquisition in our study.

Of the 42 patients with in-hospital mortality, 40 were declared dead with cardio-circulatory arrest; one had death by neurological criteria (DNC) and only one patient underwent withdrawal of life-sustaining therapy (WLST) after significant anoxic brain injury, status epilepticus, and approximately 2 months of hospital stay. All of the 8 patients, with CTP changes of non-survivable brain injury, were declared dead by cardio-circulatory arrest.

Regarding the denominator analysis, our recruited patients (n = 90) were compared to all CCAP (n = 491) admitted to the hospital in the study duration. Our study patients were similar in mean age (62.2 vs. 61.7; $p = 0.78$) but had fewer female patients (13.5 vs. 29.3%, $p < 0.001$). The proportion of in-hospital mortality was slightly lower, but not statistically different, in our study patient (46.7% vs. 53.2%, $p = 0.26$). We do not have a very definitive explanation for the difference in the proportion of female patients recruited to our study and this could merely be a chance observation.

Discussion

Our study represents the first prospective, well-powered investigation to validate CTP for the diagnosis of in-hospital mortality in CCAP, achieving remarkable specificity and PPV of 100%. These findings closely align with results from our previously conducted pilot study [12]. This underscores the pivotal role CTP can play in identifying patients at high risk of in-hospital mortality. Early identification of such patients may help initiate a timely and frank discussions with substitute decision-makers including end-of-life considerations, reducing redundant diagnostic testing, shortening ICU admissions, and potentially reducing family stress during a difficult time with the potential of saving healthcare resources [24]. In comparison to CTA, CTP exhibited superior accuracy in diagnosing patients with in-hospital mortality. Diagnostic performance of non-contrast CT head similar to that of CTP was likely incidental as this has not been supported by other studies [14, 18].

The excellent inter-rater agreement observed between the two readers underscores the ease of interpretation of the relatively novel use of CTP in non-survivable brain injury. Although further education for radiologists on interpreting CTP findings in the context of declaring non-survivable brain injury is imperative.

Qualitative assessment of CTP outperformed quantitative assessment, likely for two reasons. Firstly, there is a need for better-defined cutoff values in future studies, as many current cutoffs are derived from stroke studies and may not be directly applicable to CCAPs. Additionally, streak artifacts commonly found in the posterior fossa could potentially influence these cutoff values, particularly for quantitative assessments. On the other hand, qualitative assessment may be less affected by these factors. A recent animal study attempted to characterize CBF during open cardiac massage [20], though it did not correlate these findings with that of non-survivable brain injury. Future study of this may aid in better-defining cutoff values for CBF and CBV. Various other factors were associated with in-hospital mortality in our study underscoring the multifactorial nature of this outcome.

With consistent results from two studies, the role of CTP in the management of CCAP warrants further discussion. It may be time to consider incorporating CTP into CCAPs care protocols, similar to its established use in acute ischemic stroke. While the low sensitivity observed in our study suggests that CTP may not be ideal as a general screening tool, it could be particularly valuable in patients with severe ischemic injury who are not regaining consciousness. In such cases, identifying non-survivable brain injury may aid in guiding management decisions. However, implementation would require workflow adjustments at individual institutions. For example, we propose that CTP be performed prior to any coronary interventions. While this presents logistic challenges, modifying the STEMI care workflow could support its integration. Although timely coronary intervention remains the treatment of choice in the acute stage [25], post-intervention evaluation with CTP could be considered in the future.

PCI was found to have a protective effect on patients in our study and those who had PCI were reported to have lower in-hospital mortality. In the absence of STEMI, early angiography is not recommended in OHCA as it has been shown to be of no benefit. Our findings may reflect bias with early angiography performed in patients with other markers suggesting a better neurologic outcome, rather than any benefit of PCI [26].

A notable limitation of our study is that non-survivable brain injury was not determined at the time of hospital admission and could only be assessed after initial coronary intervention. This was primarily due to two reasons. Firstly, CTP could not be performed before PCI due to logistical constraints, and priority to timely life-saving interventions for these critically ill patients must be maintained. Secondly, comparison of the CTP findings with a thorough neurological examination immediately after the CTP was not feasible, as the patients were intubated and sedated. Therefore, we compared CTP features of non-survivable brain injury with in-hospital mortality. Consequently, the results may have been confounded by events occurring during the patients' hospitalization. However, our study provides valuable insights suggesting that patients, showing CTP features of non-survivable brain injury, are unlikely to survive beyond their hospital stay. In the future, developing a model that integrates CTP findings with other clinical parameters may enhance our ability to predict outcomes more accurately.

Conclusion

In conclusion, our study represents the first well-powered investigation to validate the CTP findings of non-survivable brain injury in diagnosing in-hospital mortality among CCAPs. We found that CTP demonstrated very high specificity and positive predictive value, emphasizing its potential as a valuable diagnostic tool for identifying patients at high risk of mortality.

Moving forward, integrating CTP findings with other clinical parameters may aid in developing more accurate diagnostic models for patient outcomes. Additionally, further research is needed to optimize the use of CTP in CCAP care protocols, potentially leading to improvements in patient management and resource allocation.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13054-025-05454-z.

Supplementary Material 1.

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Author contributions
Statement of authorship—JS-conceptualized the study, wrote the grant, monitored the study conduct, analyzed the data and wrote the first draft of the study; SA—monitored the conduct of the study and reviewed the final manuscript; MA- analyzed the data and reviewed the final manuscript; EW, JP, NS, BB, RT and RM—Collected data and reviewed the final manuscript; NS—Helped with the grant writing and reviewed the final manuscript; AT—monitored the conduct of the study and reviewed the final manuscript; IK—monitored the conduct of the study and reviewed the final manuscript; ME—Image interpretation and reviewed the final manuscript; SS—monitored the conduct of the study, reviewed the final manuscript.

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Availability of data and materials
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The study was approved by our institutional research ethics board (REB number- HS23646 B2020:017) and the study was conducted in accordance with the Declaration of Helsinki.

Consent for publication
Consent was obtained or waived by the research ethics board for all patients included in the study.

Competing interests
I am PI of EMMA Can randomized control trial that is funded by Medtronic Canada.

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