European Journal of Radiology 116 (2019) 165–173

Contents lists available at ScienceDirect

European Journal of Radiology

journal homepage: www.elsevier.com/locate/ejrad

Research article

# Whole-brain CT perfusion on admission predicts delayed cerebral ischemia following aneurysmal subarachnoid hemorrhage

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ARTICLE INFO

*Keywords:*
CT perfusion
Aneurysmal subarachnoid hemorrhage
Delayed cerebral ischemia
Prediction

ABSTRACT

*Purpose:* The aim of this study is to prospectively evaluate whole-brain CT perfusion (CTP) on admission to predict delayed cerebral ischemia (DCI) in patients with aneurysmal subarachnoid hemorrhage (aSAH).

*Methods:* All 252 consecutive patients with aSAH enrolled in this study underwent one-stop whole-brain CTP scan within 24 h after aneurysm rupture. The qualitative and quantitative CTP parameters and clinical data were compared between patients with and without DCI. Diagnostic performance of clinical data and mean and lowest CTP parameters were evaluated by receiver-operating characteristic (ROC) analyses. Logistic regression analysis was employed to determine predictors of DCI.

*Results:* The study evaluated 191 of 252 consecutive patients, 57 of whom (29.8%) developed DCI during hospitalization. Patients with diffused hypoperfusion had the highest incidence rate of DCI (43%, 46/107). Mean TMax produced the largest area under the curve of 0.726 (95% confidence interval [CI] 0.638–0.814), and a cutoff value of 2.240 s provided sensitivity of 73.7% and specificity of 71.6% for early prediction of developing DCI. Glasgow Coma Scale score (odds ratio [OR] = 0.716, 95% CI 0.565–0.908, P = 0.006), cerebral vasospasm (OR = 6.117, 95% CI 1.427–26.223, P = 0.015), hydrocephalus (OR = 3.795, 95% CI 1.327–10.858, P = 0.013), and qualitative CTP analysis (OR = 3.383, 95% CI 1.686–6.789, P = 0.001) were all significant independent predictors of DCI.

*Conclusions:* Whole-brain CTP within 24 h of admission can qualitatively and quantitatively detect abnormal cerebral perfusion. It is possible to predict the risk of developing DCI after aSAH when the TMax value is larger than 2.240 s.

## 1. Introduction

Delayed cerebral ischemia (DCI) is a serious complication following aneurysmal subarachnoid hemorrhage (aSAH) and leads to poor outcomes [1,2]. DCI affects approximately 30% of patients who survive the initial hemorrhage and usually occurs 3–14 days after the hemorrhage [3,4]. The pathogenesis of DCI has not been elucidated. Several recent studies focused on early brain injury, microcirculatory dysfunction, and impaired autoregulation [3,5,6]. Early prediction of the risk of developing DCI and urgent treatment are critical to reducing mortality and morbidity [7].

Computed tomography perfusion (CTP) provides hemodynamic information and has been applied in brain tumors, ischemic stroke, and aSAH [8,9]. CTP imaging was reported in previous clinical studies to have an important role in detecting hemodynamic disturbances thought to occur in DCI and vasospasm [10]. In a majority of studies, the CTP was performed because of the presence of clinical symptoms more than 72 h after aneurysm rupture [4,5,11].

Data concerning whole-brain CTP in aSAH patients within 24 h after onset are very limited. In our institution, every patient routinely undergoes CT angiography (CTA) at admission to discover the cause of SAH. Because one-stop whole-brain CTP is able to not only evaluate cerebral arteries but also access cerebral perfusion, a one-stop whole-brain CTP scanning protocol was designed for SAH patients on admission. The present study focused on discerning the cause of SAH while investigating whether qualitative and quantitative CTP within 24 h of admission can detect cerebral perfusion abnormalities and predict the occurrence of DCI in patients after aSAH.

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https://doi.org/10.1016/j.ejrad.2019.05.008
Received 2 March 2019; Received in revised form 20 April 2019; Accepted 7 May 2019

## 2. Materials and methods

### 2.1. Patient population and study design

Our prospective study was approved by the institutional review board, and informed consent was obtained from all patients or family members with responsibility. A total of 252 consecutive SAH patients underwent the one-stop whole-brain CTP scan on admission from December 2015 to June 2017; the scan included noncontrast CT (NCCT), CTP, and dynamic CTA derived from CTP data. The following inclusion criteria were used in this study: (1) 18 years of age or older and (2) CTP performed within 24 h after aneurysm rupture. The study exclusion criteria included the following: (1) pregnancy, (2) impaired cardiac or renal function, (3) tumors or other serious artery disease, (4) intracerebral hematoma causing midline structure displacement, and (5) CTP imaging with extensive motion artifacts.

We recorded the following information for all included patients: age, gender, clinical status on admission according to the Glasgow Coma Scale (GCS) score, Hunt–Hess grade, global cerebral edema, hydrocephalus, amount of blood on NCCT according to the Fisher score, and presence of cerebral vasospasm (CV) on dynamic CTA derived from CTP data. The surgical clipping and/or endovascular coiling for aneurysm repair were performed in accordance with current SAH guidelines [12]. A total of 152 patients underwent coil embolization, 21 patients were treated through microsurgical clipping, and 18 patients were treated conservatively. All patients received routine nimodipine intravenously via a micropump at the rate of 2 mL/h during hospitalization. If the CTP results indicated abnormal perfusion, the neurologist analyzed the causes and adjusted treatment options.

### 2.2. CTP scanning protocol and post-processing

All imaging studies were performed on a dual-source CT scanner (SOMATOM Definition Flash; Siemens Healthcare, Erlangen, Germany). The following parameters were used for the one-stop whole-brain CTP scanning models. An NCCT scan was first performed with a tube voltage of 120 kVp and a tube current–time product of 390 mAs. Each patient received an injection of 55 mL of nonionic contrast agent (Ioversol, 350 mg iodine/mL; Hengrui, Jiangsu, China) in the antecubital vein at a flow rate of 5 mL/s, followed by 40 mL of saline flush at the same rate using a dual power injector (Stellant Injection System; Medrad, Indianola, PA, USA). The CTP acquisitions were obtained using a four-dimensional (4D) spiral mode to cover the range of 15 cm from skull base to the top of head after the contrast material injection. The images were obtained with a time interval of 1.5 s per spiral and 14 cycles in the first phase. The scan time was then 4.5 s per spiral and 6 cycles in the second phase, which is a total of 46.35 s. The CTP scan parameters included the following: tube voltage, 80 kV; tube current–time product, 120 mAs; section collimation, 32 × 1.2 mm with a z-flying focal spot. The perfusion images were reconstructed with a section thickness of 3 mm and an increment of 3 mm by using convolution kernel H30f. Additionally, 1.5-mm thin-slice images (increment, 1.5 mm; convolution kernel H30f) were reconstructed for dynamic CTA analyses. The location of aneurysm was recorded in all patients.

The CTP data were transferred to a post-processing workstation (Syngo.via, version VA30A; Siemens Healthcare). We performed 4D noise reduction and segmentation after completing the optional motion correction. The vessel segmentation thresholds were reviewed with automatic arterial and venous vessel identification. The grayscale and color-coded perfusion parameter maps for cerebral blood flow (CBF), cerebral blood volume (CBV), mean transit time (MTT), time to delay (TTD), time to start (TTS), and transit time to the center of the impulse response function (TMax) were produced for qualitative CTP analysis.

The qualitative CTP analysis on color-coded perfusion parameter maps consisted of three patterns covering the whole brain: (1) normal perfusion maps (Fig. 1C–H), showing a periventricular symmetrical green to yellow area on TTD and TMax images; (2) localized hypoperfusion (Fig. 2C–H), showing a patchy green to yellow or red area on TTD and TMax images; (3) diffused hypoperfusion (Fig. 3C–H), showing a diffused green to yellow or red area on TTD and TMax images. Positive findings of qualitative evaluation consisted of hypoperfused areas that were not localized at the neurosurgical trajectory or directly surrounding an intracerebral hematoma.

Thirty-two hand-drawn regions of interest (ROIs) were created collaboratively on five slices (Fig. 4). The ROIs were drawn on the maximum-intensity projection (MIP) map for each of the five slices in the cortical flow territories of the anterior cerebral artery, middle cerebral artery, posterior cerebral artery, basal ganglia, and cerebellum. Quantitative CTP analysis included the following two models: (1) mean values: the values of 32 ROIs for each parameter were averaged in each patient; (2) lowest values: the minimum values of CBF and CBV, and the maximum values of MTT, TTD, TTS, and TMax of single ROIs, were recorded for each patient. The CTP images were analyzed by two experienced radiologists (with 5 and 10 years of experience) in consensus, who were blind to all clinical and imaging data to limit test-review bias. To minimize the contribution of vascular pixels from large vascular, we excluded CBF values > 100 mL/100 g/min from the analysis [13].

### 2.3. DCI determination

We used the following outcome measurements based on recent expert consensus and previous clinical research [11,14,15]: (1) cerebral infarction on NCCT or magnetic resonance imaging (MRI) within 6 weeks after SAH or on the latest NCCT or MRI within 6 weeks of death, which was not present on imaging up to 48 h after aneurysm occlusion and was not attributable to other causes such as surgical clipping, endovascular treatment, ventricular catheter placement, or intraparenchymal hematoma; and/or (2) focal neurological impairment (such as hemiparesis, hemiplegia, aphasia) and/or global neurological impairment (at least 2-points decrease on GCS) lasting 1 h or longer that was not apparent immediately after aneurysm occlusion and not attributed to other causes. The infarction on NCCT or MRI and neurological impairment were assessed by two authors (one radiologist and one neurologist) blinded to the baseline CTP results.

### 2.4. Statistical analysis

The continuous variables are presented as the mean ± standard deviation, and categorical variables are shown as counts and/or frequencies. The patients were grouped according to whether DCI was present. The differences in clinical and imaging characteristics were determined using independent t-tests for continuous variables with normal distribution. The Wilcoxon rank sum test was used for continuous variables with skewed distribution. The chi-squared analysis was used for categorical variables. Receiver-operating characteristic (ROC) curves were generated for Hunt–Hess grade, Fisher score, age, GCS, and quantitative CTP values (the mean value and lowest value) with its 95% confidence interval (CI). The areas under the curve (AUC) were also calculated. We then selected the parameters with AUC > 0.5. The optimal threshold values were derived from the ROC curves to distinguish patients with and without DCI. A binary logistic regression analysis was performed to determine predictors of DCI. All P values less than 0.05 were considered statistically significant, and all statistical analyses were performed using the SPSS statistics package, version 17.0 (IBM, Armonk, NY, USA).

## 3. Results

We analyzed 191 aSAH patients (134 women and 57 men; mean age 60 ± 11 years) of the 252 consecutive patients enrolled in the prospective trial. All clinical, demographic, and imaging characteristics of the population are summarized in Table 1. Fifty-seven of 191 patients

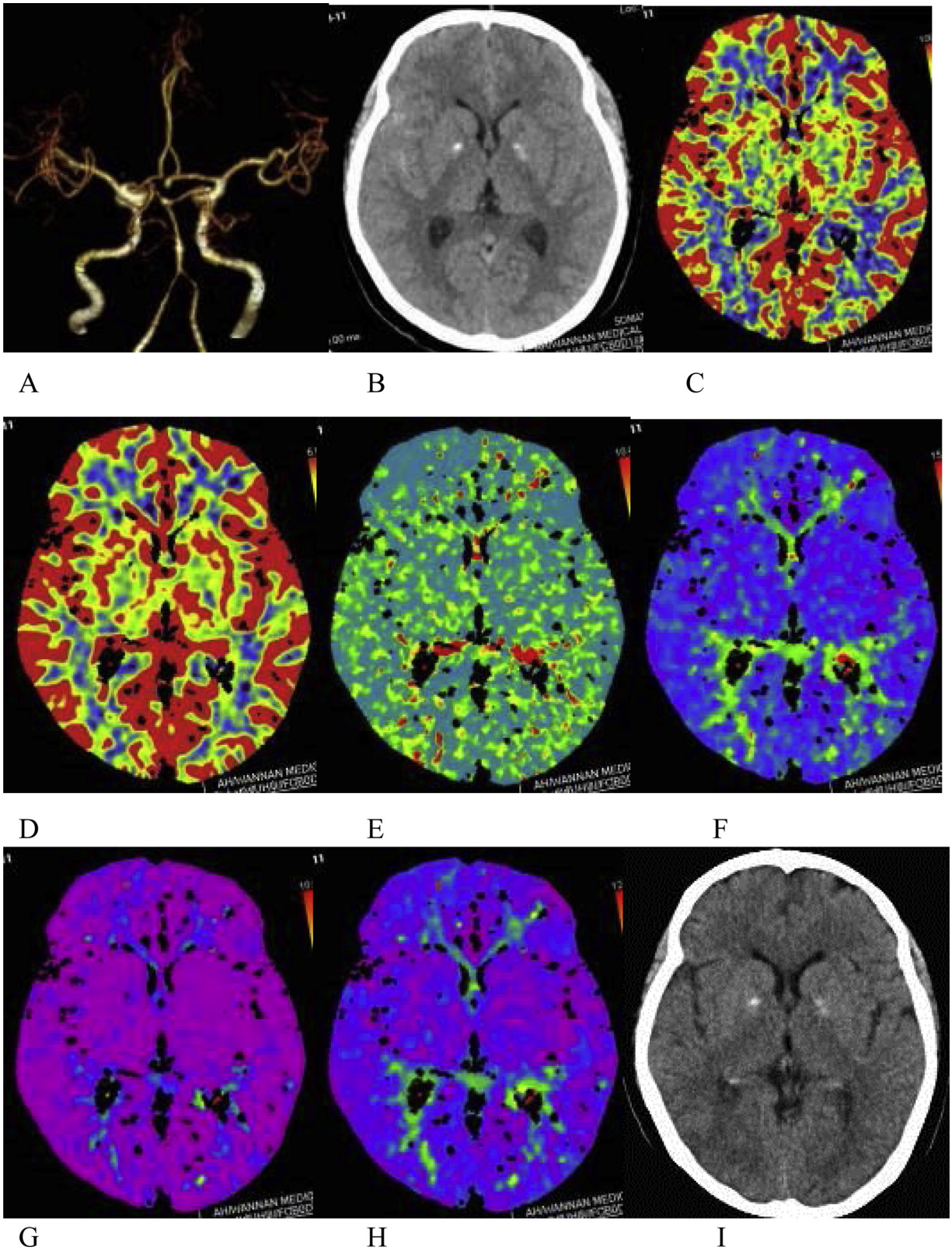


**Fig. 1.** A 52-year-old female patient with ruptured anterior communicating aneurysm on admission and without DCI. CTA (A) showed anterior communicating aneurysm. The Fisher score was 2 on NCCT (B). Color-coded maps of CTP parameters including CBF (C), CBV (D), MTT (E), TTD (F), TTS (G), and TMax (H) revealed no perfusion abnormalities. There were no signs or symptoms of DCI on follow-up CT (I).

(29.8%) developed DCI during hospitalization.

The data indicated that DCI tended to occur in patients with older age ($P = 0.002$), higher Hunt–Hess grade ($P < 0.001$), higher Fisher score ($P < 0.001$), and lower GCS ($P < 0.001$) on admission. The CTP scan within 24 h after aneurysm rupture indicated significant differences between DCI and non-DCI patients in CV ($P = 0.010$), global cerebral edema ($P = 0.045$), and hydrocephalus ($P < 0.001$). There were significant differences between patients with and without DCI in

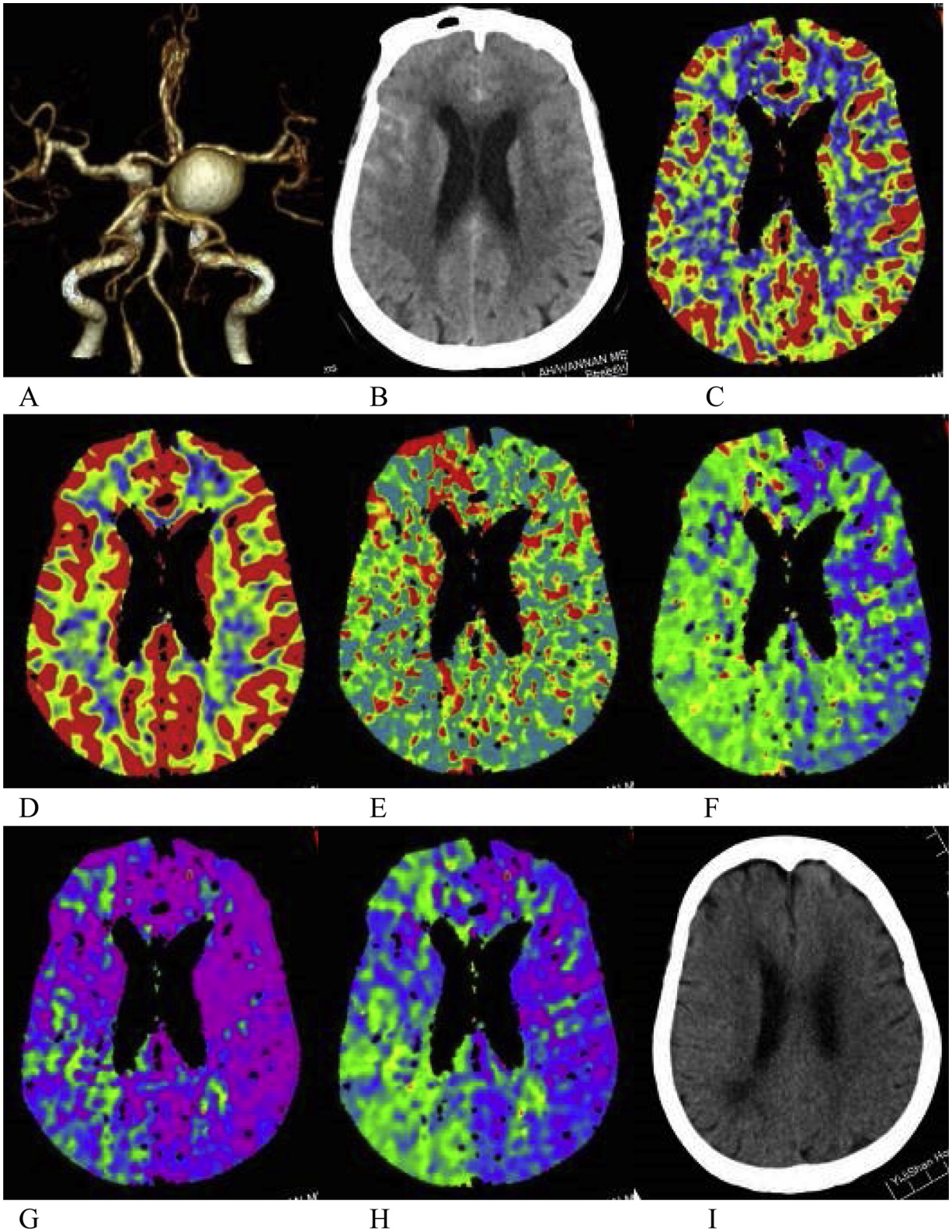


**Fig. 2.** CTA (A) showed right posterior communicating aneurysm. The Fisher score was 2 on NCCT (B). Color-coded maps of CTP parameters (C–H) revealed that compared with the left hemisphere, the right hemisphere showed mild decreased CBF (C) and normal CBV (D), mild prolonged MTT (E), and significantly prolonged TTD (F), TTS (G), and TMax (H). Left hemiplegia occurred on day 18 after admission. There were multiple infarctions in the right hemisphere on follow-up CT (I).

all mean CTP parameter values. However, there were no differences in sex or minimum CBV parameter values. The results of the analysis of qualitative perfusion maps are shown in Table 1. The incidence rate of DCI in patients with diffused hypoperfusion, localized hypoperfusion, and normal perfusion maps were 43%, 26%, and 2%, respectively. There were significant differences ($P < 0.001$) in incidence rate of DCI among the three types of qualitative analysis. We found that there were lower CBF and CBV and longer MTT, TTD, TTS, and TMax in early

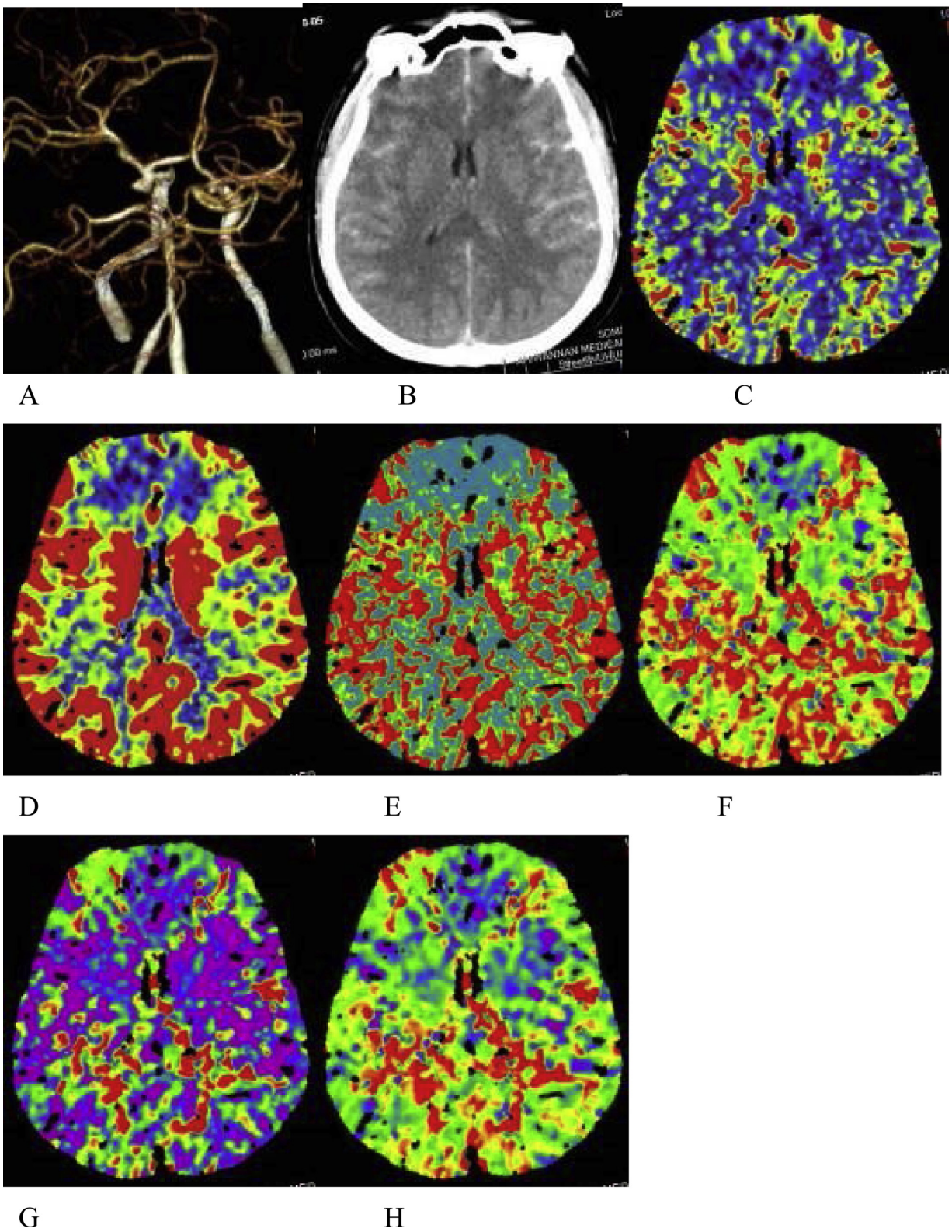


**Fig. 3.** A 43-year-old male patient with ruptured left posterior communicating aneurysm on admission who died 11 days later. CTA (A) showed left posterior communicating aneurysm. The Fisher score was 3 on NCCT (B). Color-coded maps of CTP parameters (C–H) revealed whole-brain perfusion abnormalities showing decreased CBF (C) and CBV (D), and significantly prolonged MTT (E), TTD (F), TTS (G), and TMax (H).

whole-brain CTP scans from the patients with DCI compared with patients without DCI during hospitalization (Figs. 1–3).

The ROC curves of Hunt–Hess grade, Fisher score, age, GCS, and all quantitative CTP values are shown in Fig. 5. The AUCs, thresholds, sensitivities, and specificities of quantitative CTP parameter values and clinical data are summarized in Table 2. The mean TMax had the largest AUC of 0.726 (95% CI 0.638–0.814), and the cutoff value of 2.240 s provided sensitivity of 73.7% and specificity of 71.6% for early

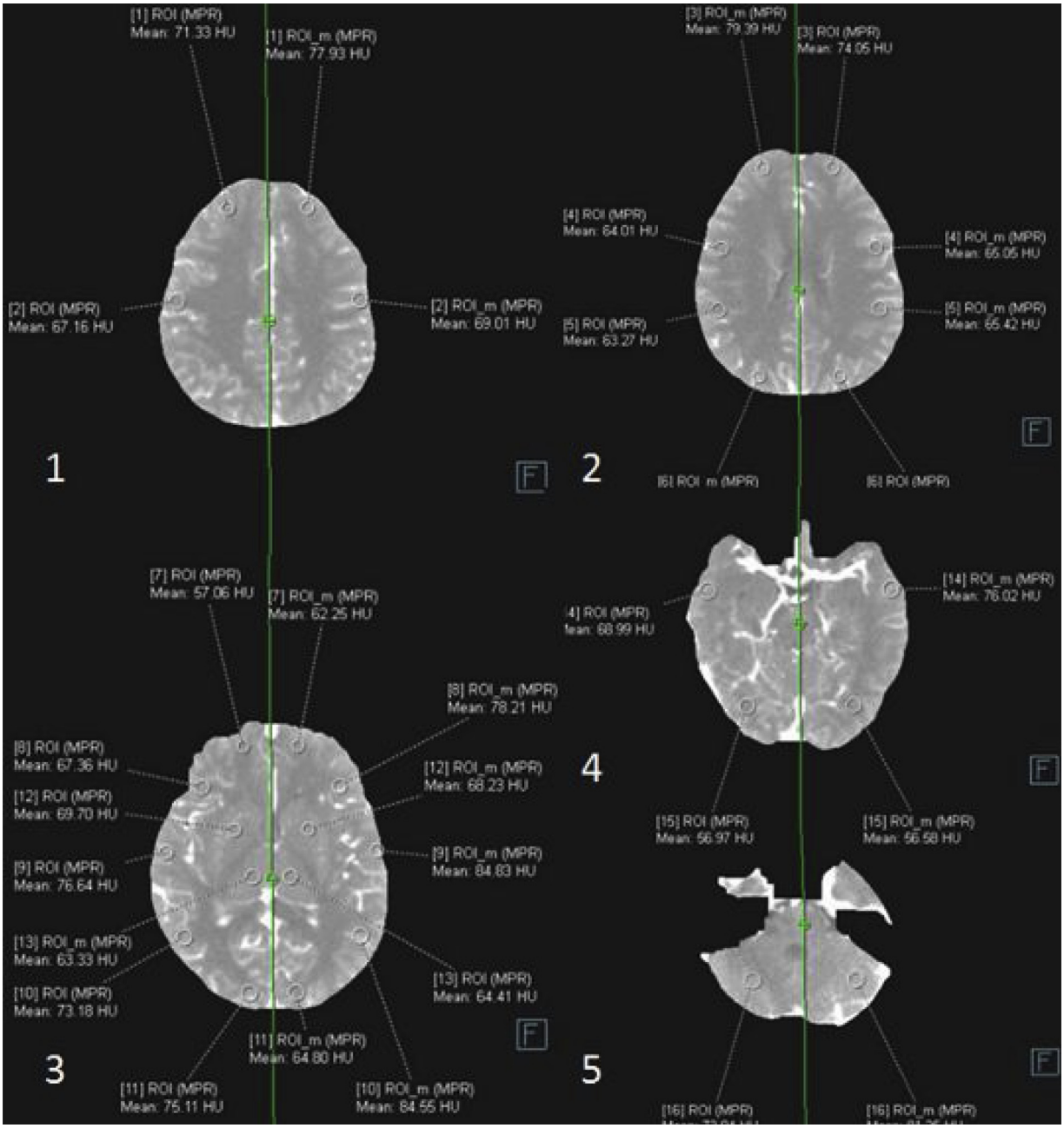


**Fig. 4.** An example of placement of 32 regions of interest (ROIs) on five slices at MIP imaging. The post-processing software automatically calculates the values of CBF, CBV, MTT, TTD, TTS, and TMax for each ROI.

prediction of DCI on admission.

We investigated patient age, GCS, Hunt–Hess grade, Fisher score, CV, global cerebral edema, hydrocephalus, qualitative CTP analysis, and mean TMax using a logistic regression model to determine significant predictors of DCI. The results show that GCS (odds ratio [OR] = 0.716, 95% CI 0.565–0.908, P = 0.006), CV (OR = 6.117, 95% CI 1.427–26.223, P = 0.015), hydrocephalus (OR = 3.795, 95% CI 1.327–10.858, P = 0.013), and qualitative CTP analysis (OR = 3.383, 95% CI 1.686–6.789, P = 0.001) were significant independent predictors of DCI.

## 4. Discussion

This prospective study focused on whole-brain CTP on admission to predict DCI following aSAH and enrolled the maximum number of cases. The majority of previous investigations reported data derived from cohorts with fewer than 100 patients [2,9,14,16–18].

The main results of our study demonstrated that whole-brain CTP can qualitatively and quantitatively detect hypoperfusion on admission within 24 h, and it is possible to predict the risk of developing DCI using quantitative parameters, among which the mean TMax value seems to be a better parameter for the early prediction of DCI. Previous studies confirmed that time-related parametric maps (MTT, TTP, and TTD) are highly sensitive in CTP analyses [19,20]. The present study also included TMax, which is rarely noted in other studies.

In the present study, the AUCs were slightly less than those reported in other investigations using CTP for DCI [14,21–23]. We speculate that there are several reasons for these differences. First, the duration and extent of DCI influenced our results. All our cases underwent the CTP scan within 24 h after aneurysm rupture. As this is not a period of high incidence for DCI [3], microcirculation changes were not obvious during this period. The timing of the CTP examination in most similar

**Table 1**
Clinical, demographic and imaging characteristics in patients with and without DCI.

| Characteristics | All (n = 191) | DCI(57) | Without DCI(n = 134) | *P* |
|---|---|---|---|---|
| No. of female | 134(70%) | 37(65%) | 97(72%) | 0.302 |
| Age (mean ± SD) | 60 ± 11 | 64 ± 11 | 59 ± 11 | 0.002* |
| GCS(mean ± SD) | 12.79 ± 3.34 | 10.32 ± 4.31 | 13.84 ± 2.09 | < 0.001* |
| Hunt-Hess grade(%) | | | | < 0.001* |
| 1 | 69(36%) | 9(16%) | 60(45%) | |
| 2 | 64(34%) | 18(33%) | 46(34%) | |
| 3 | 40(21%) | 16(28%) | 24(18%) | |
| 4 | 15(8%) | 11(19%) | 4(3%) | |
| 5 | 3(2%) | 3(5%) | 0(0%) | |
| Fisher score(%) | | | | < 0.001* |
| 2 | 55(29%) | 8(14%) | 47(35%) | |
| 3 | 81(42%) | 18(32%) | 63(47%) | |
| 4 | 55(29%) | 31(54%) | 24(18%) | |
| CV | 13 | 8 | 5 | 0.010* |
| Edema | 34 | 15 | 19 | 0.045* |
| Hydrocephalus | 57 | 32 | 25 | < 0.001* |
| Aneurysm location | | | | |
| anterior circulation | 96 | 32 | 64 | |
| posterior circulation | 75 | 19 | 56 | |
| infratentorial region | 13 | 4 | 9 | |
| multiple | 7 | 2 | 5 | |
| Qualitative CTP analysis | | | | < 0.001* |
| normal | 46 | 1 | 45 | |
| localized hypoperfusion | 38 | 10 | 28 | |
| diffused hypoperfusion | 107 | 46 | 61 | |
| Mean CTP value | | | | |
| Mean CBF,mL/100 g/min | 72.72 ± 10.28 | 67.28 ± 10.55 | 75.04 ± 9.28 | < 0.001* |
| Mean CBV,mL/100 g | 4.72 ± 0.44 | 4.57 ± 0.48 | 4.78 ± 0.41 | 0.004* |
| Mean MTT,seconds | 4.53 ± 0.66 | 4.84 ± 0.80 | 4.40 ± 0.54 | < 0.001* |
| Mean TTD,seconds | 4.42 ± 1.08 | 4.97 ± 1.37 | 4.18 ± 0.84 | < 0.001* |
| Mean TTS,seconds | 0.57 ± 0.42 | 0.77 ± 0.56 | 0.48 ± 0.31 | < 0.001* |
| Mean TMax,seconds | 2.17 ± 0.78 | 2.57 ± 1.00 | 2.00 ± 0.60 | < 0.001* |
| Lowest CTP value | | | | |
| Minimum CBF,mL/100 g/min | 38.68 ± 9.08 | 35.86 ± 9.20 | 39.88 ± 8.79 | 0.005* |
| Minimum CBV,mL/100 g | 2.71 ± 0.58 | 2.63 ± 0.67 | 2.74 ± 0.54 | 0.223 |
| Maximum MTT,seconds | 7.42 ± 1.78 | 8.19 ± 2.13 | 7.10 ± 1.51 | 0.001* |
| Maximum TTD,seconds | 7.26 ± 2.20 | 8.13 ± 2.52 | 6.89 ± 1.95 | 0.001* |
| Maximum TTS,seconds | 2.05 ± 1.32 | 2.42 ± 1.50 | 1.90 ± 1.21 | 0.012* |
| Maximum TMax,seconds | 4.09 ± 1.61 | 4.64 ± 1.82 | 3.86 ± 1.45 | 0.002* |

*DCI*: delayed cerebral ischemia, *GCS*: the Glasgow Coma Scale score, *CV*: cerebral vasospasm *CTP*: computed tomography perfusion, *SD*: standard deviation.
* Significant difference ($P < 0.05$).

investigations was later than 24 h. A scan performed closer to the onset period leads to more obvious perfusion abnormalities [11,14]. Second, our study comprised prospective research, and we used the grayscale MIP map to draw standard ROIs. Therefore, the perfusion ROI measurements may not be centered on the ischemic area and depend on color-coded perfusion parameter maps. Selecting the ROI in visible areas of hypoperfusion could result in a larger AUC [24]. The sensitivity and specificity in our study were slightly lower than the values previously reported for CTP during the period of clinical deterioration or more than 72 h later. These time points could be too late for intervention to enable avoidance of permanent neurological impairment or cerebral infarction. A routine whole-brain CTP performed in the acute phase of aSAH might provide early identification and guide effective treatment.

Our logistic regression model analysis identified admission GCS, CV, hydrocephalus, and qualitative CTP analysis as significant independent predictors of DCI. Aldakkan et al. [25] confirmed that CV was the only predictor in their univariate analysis. However, CV was challenged as the sole causal mechanism of DCI in earlier investigations [6,16,26]. The DCI occurred in approximately 10%–50% of patients with CV [10]. The DCI might be considered as an abnormal and prolonged contraction of vascular smooth muscle arising from the subarachnoid blood and its products [26]. In addition, our results were partly different from those of the study conducted by de Rooij et al. [27]. These authors confirmed that clinical condition on admission, amount of cisternal and intraventricular blood on CT, and age were the strongest predictors. In the multivariate analysis model, the different factors may affect each other and lead to different results. Thus, further studies with more expansive sample sizes are needed, and more complex multivariate models are required to provide more reliable predictive capabilities in our institution.

There are several limitations to our study. First, we used only the absolute values rather than the relative values in the evaluation of perfusion parameters. Although the relative values are conducive to the unity and comparison among different research institutions, they are not suitable for patients with bilateral-hemisphere diffuse perfusion abnormalities. Second, artifacts affect the perfusion values in the infratentorial region (cerebellum and brainstem) [28,29], which can affect the accuracy of the whole-brain perfusion parameter values to some extent. We artificially excluded the ROI value of the artifact area in quantitative analysis. Third, we did not investigate the relationship between cardiac dysfunction and cerebral perfusion in patients with aSAH. Several studies have reported that aSAH patients with cardiac dysfunction show decreased focal and global cerebral perfusion [30].

## 5. Conclusions

Whole-brain CTP within 24 h of admission can qualitatively and quantitatively detect abnormalities of cerebral perfusion, and it is possible to predict the risk of developing DCI after aSAH when the TMax value is larger than 2.240 s.

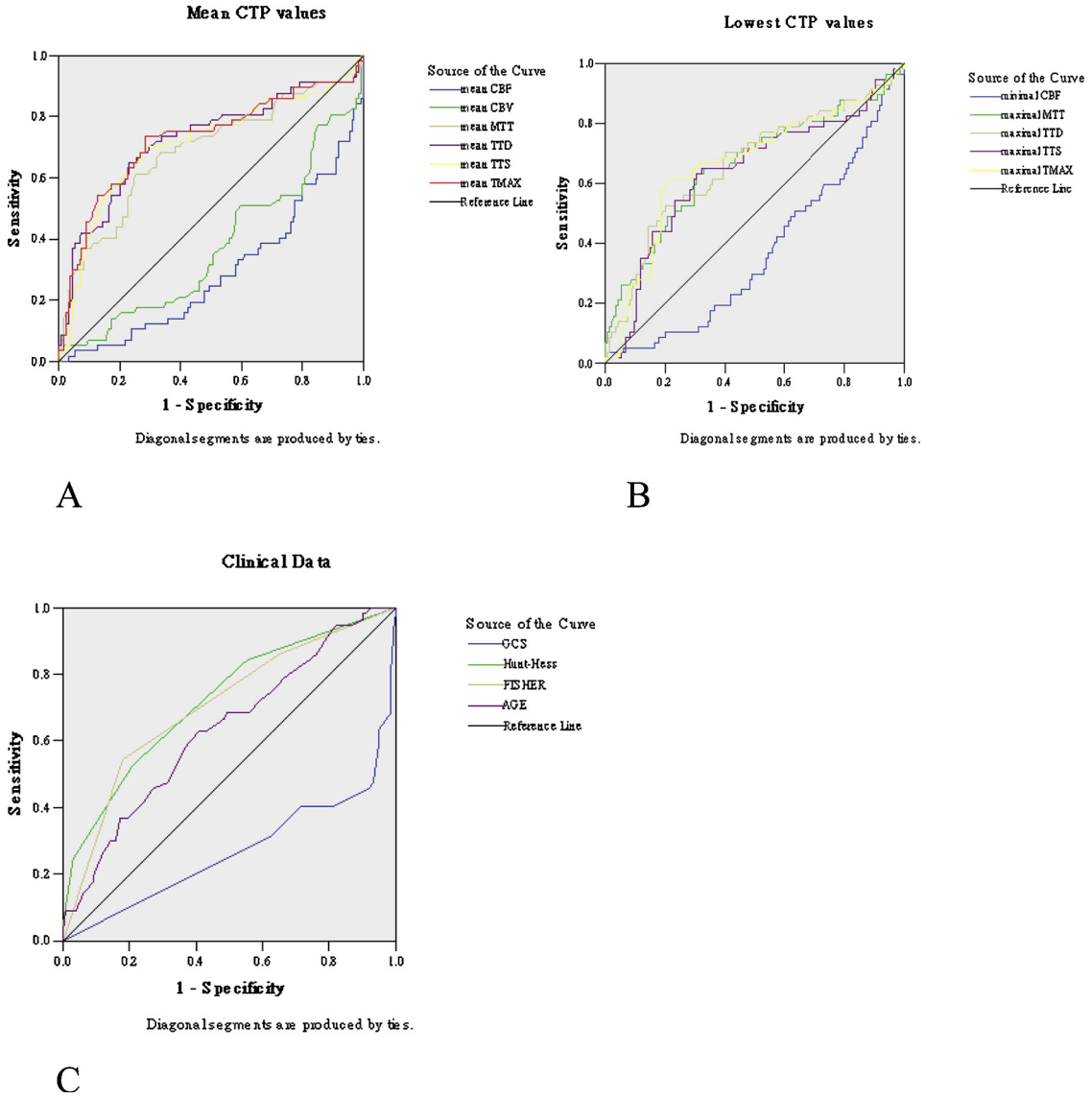


**Fig. 5.** Receiver-operating characteristic curves of mean (A) and lowest (B) CTP parameter values, and clinical data (C).

**Table 2**
AUCs, thresholds, sensitivities and specificities of CTP parameters, and clinical data for the early predicton of DCI.

| Characteristics | AUC(95%CI) | Thresholds | Sensitivities | Specificities |
|---|---|---|---|---|
| Mean MTT,seconds | 0.686(0.597-0.776) | 4.665 | 61.4% | 74.6% |
| Mean TTD,seconds | 0.723(0.636-0.810) | 4.685 | 64.9% | 76.9% |
| Mean TTS,seconds | 0.710(0.621-0.799) | 0.575 | 70.2% | 71.6% |
| Mean TMax,seconds | 0.726(0.638-0.814) | 2.240 | 73.7% | 71.6% |
| Maximum MTT,seconds | 0.661(0.570-0.751) | 7.765 | 63.2% | 69.4% |
| Maximum TTD,seconds | 0.664(0.574-0.753) | 8.040 | 52.6% | 79.9% |
| Maximum TTS,seconds | 0.638(0.547-0.729) | 1.955 | 64.9% | 67.9% |
| Maximum TMax,seconds | 0.665(0.575-0.755) | 4.445 | 61.4% | 79.1% |
| Hunt-Hess grade | 0.721(0.640-0.802) | 2.5 | 52.6% | 79.1% |
| Fisher score | 0.705(0.622-0.788) | 3.5 | 54.4% | 82.1% |
| age | 0.632(0.545-0.718) | 62.5 | 63.2% | 59.0% |

*AUC*: area under the curves, *CTP*: computed tomography perfusion, *DCI*: delayed cerebral ischemia, *CI*: condifence interval.

## Conflict of interest

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

## Declarations of interest

None.

## Acknowledgement

We thank Hugh McGonigle, from Liwen Bianji, Edanz Group China (www.liwenbianji.cn/ac), for editing the English text of a draft of the manuscript.

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