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"CT PERFUSION FOR ASSESING THE RISK OF DELAYED CEREBRAL ISCHEMIA AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE"

Neurosurgery

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ABSTRACT

Objective: To evaluate the usefulness of CT Perfusion study inPatients with aneurysmal SAH by using computed tomography perfusion (CTP)-degree of perfusion abnormalities and toassess its co-relation with occurance of delayed cerebral ischeamia based on Modified Rankin scale(mRS). **Material And Methods:** Prospective evaluation of aneurysmal SAH patientswho were taken for CTP after admission and then on discharge. We evaluated fiveregions of interest (ROI); i.e Orbitofrontal Cortex (OFC), Internal capsule(IC), Thalamus(TH), Caudate Nucleus (CN), and Sensorimotor Cortex for cerebral blood perfusion. The Patient's clinical and radiological findings were analyzed; correlated with CBF (cerebral blood flow) and MTT (mean transient time), and the outcome assessed usingthe modified Rankin scale on 3 months follow-up. **Result:** On admission, cerebral blood flow (CBF), cerebral bloodvolume (CBV), mean transit time (MTT), and time-to-peak (TTP) did not differ between patients who did and did not develop DCI. Inthe DCI time-window (4 to 14 days after subarachnoid hemorrhage (SAH)), DCI was associated with a decreased CBF and an increased MTT. Both CBF and MTT showed a significant correlation with the patients developing DCI. In aSAH patients developing DCI, the mean CBF on admission was 65.63 which reduced to 44.90 on discharge with P<0.0001 showing a significant variation. The meanMTT for DCI+ patients on admission was 6.58 which increased to 7.36 sec on discharge with P<0.0001 variation which was statistically significant. **Conclusion:** We conclude that CBF and MTT are two important radiological parameters of CT perfusion study that can be used as prognostic indicators in patients with aneurysmal SAH to predict development of DCI in patients and treat them accordingly.

KEYWORDS

Subarachnoid Hemorrhage, Delayed Cerebral Ischemia, Cerebral Perfusion, Vasospasm

INTRODUCTION

Subarachnoid haemorrhage (SAH) is still associated with high morbidity and mortality despite technical advances in the treatment of cerebral aneurysms and new discoveries in the treatment of delayed cerebral ischaemia (DCI) due to vasospasm. severity of the initial bleeding, expressed by disturbance of the level of consciousness ,the amount of bleeding in the initial computed tomography (CT), and age are still the major determinants of the outcome.

It is known that after the initial bleeding there is a global decrease in cerebral blood flow that lasts for many hours—even days—after aneurysm rupture(1). These disturbances in the cerebral blood flow seem to be caused by an increase in intracranial pressure (ICP), which initially stops the bleeding.

Another proposed mechanism is global vessel vasoconstriction in response to the presence of blood in the subarachnoid space and intraluminal obstruction because of vascular micro-thrombi formation(2,3,4). All these mechanisms provoke an inadequate brain perfusion that ends in ischaemia, which is mainly responsible for the acute brain injury. DCI is a serious complication following aneurysmal SAH affecting upto 30% patients and remains the leading cause of morbidity and mortality (9).

Symptoms typically develop between 4 and 14 days after aneurysm rupture/ hemorrhage. Vasospasm is considered a major contributing factor to the development of DCI, in which severe arterial narrowing may result in decreased cerebral blood flow.

Early diagnosis and prompt treatment of DCI are primarily focused on preventing its devastating sequelae of permanent neurologic deficits, cerebral infarction, and death, and is crucial in improving outcomes in patients(10).

DCI, defined as a functional disability andc erebral infarction on imaging not attributed to other causes such as aneurysm treatment-clipping/coiling, ventricular catheter placement, or intraparenchymal hematoma, is characterized by a decrease in consciousness, new focal deficit, or both (9). Although vasospasm is traditionally thought to be the main cause of DCI, with arterial narrowing resulting in reduced cerebral blood flow (CBF) and ischaemia, these terms have been erroneously considered interchangeable.

The caveat is that not all patients with vasospasm develop DCI and, conversely, not all patients who experienceDCI have vasospasm. (11,12)as DCI is preceded by a decreased cerebral perfusion, it is thought that cerebral perfusion–computerized tomography (CTP) may be able to detect DCI before cerebral infarction.

The diagnosis of DCI can be difficult, as clinical deterioration can also have other etiologies, such as hydrocephalus, rebleeding, metabolic disturbances, or infections. For diagnosing and guiding treatment of DCI, a diagnostic tool in the acute setting of clinical deterioration is needed, but currently, the diagnosis can only be confirmed by the identification of ischemic lesions on follow-up imaging.

Brain perfusion can be measured with a variety of techniques, including transcranial Doppler (TCD)(5), PET, SPECT, xenon-enhanced CT (Xe CT), and perfusion-weighted MRI(6). TCD, however, cannot be used to quantify cerebral blood flow (CBF) at the tissue level, and the other techniques have limited clinical access, in particular for patients who are restless or ventilated.

CT perfusion (CTP) allows the quantitative measurement of cerebral blood volume(CBV), CBF, mean transit time (MTT), and time topeak (TTP)(7). CTP is also practical in an acute clinical setting because it can easily be combined with the initial noncontrast CT demonstrating SAH and can serve for the delay time for CT angiography (CTA)(8).

CBF values of CTP provided accurate and reliable data compared with Xe CT in patients with various types of cerebrovascular disease. There are many techniques for studying cerebral perfusion deficit, like magnetic resonance perfusion, Xenon computed tomography, positron emission tomography (PET) and single photon emission computed tomography (SPECT), but they are limited by their cost, their availability or patient tolerance Perfusion CT is a rapid technique thatallows qualitative and quantitative evaluation of cerebral perfusion.

The aim of this study was to explore the feasibility of acutely performing CT perfusion in these patients and correlate the CT perfusion parameters with the acute bleeding, their severity and the delayed outcome of patients suffering aneurismal SAH.

Our hypothesis was that there are significant differences in the CTP parameters in patients with DCI and poor outcomes; therefore, CTP may have the potential to provide valuable prognostic information

MATERIAL AND METHODS.

1. Patient/study design

A prospective study was conducted on patients with aneurysmal SAH admitted to the Department of Neurosurgery , Sawai Man Singh Medical College and Associated Group of Hospitals, Jaipur, Rajasthan, India. Patients were enrolled in the study after obtaining informed and written consent. The study was conducted on 50 patients over one year from Jan 2020 to Jan 2021.We excluded patients who died early after admission. Because the risk of dying can be related to the outcome measure DCI, inclusion of these patients would have blurred the relation between CTP and DCI, resulting in an under estimation of the relation. CTP was done within 24 hrs of admission and at the time of discharge.

Modified Rankin score (mRs) was measured at 3 months of discharge to check for DCI.

2. Data collection

Demographic data such as Age and sex was recorded and patients were assessed with Glasgow Coma Scale and the World Federation of Neurological Surgeons scale (WFNS). Diagnostic CT was performed and the presence and amount of cisternal blood was evaluated by using the Fisher scale and Hess and Hunt Scale. The presence and location of the aneurysm responsible for the bleeding and its mode of management- clipping/ coiling was recorded. Delayed cerebral ischaemia (DCI) was defined as clinical deterioration attributable to vasospasm, when other causes of deterioration could be ruled out such as hydrocephalus, rebleeding or electrolyte abnormalities, infection, metabolic disturbances, seizure or by the detection of a new infarct on brain CT related to vasospasm that was not visible on the admission or inmediate post-treatment scan, or both.

3. Outcome measure

Outcome was evaluated 3 months after the bleeding by means of the Modified Rankin score (mRs).

The primary outcome measures included the following:
1) cerebral infarction on CT or MR imaging within 6 weeks after SAH, which was not present on imaging up to 48 hours after aneurysm occlusion and was not attributable to other causes such as surgical clipping, endovascular treatment, ventricular catheter placement, and intraparenchymal hematoma.
2) permanent neurologic deficit on clinical examination, distinct from the deficit at baseline, produced by aneurysm rupture or surgical intervention, and not attributable to other causes.

The secondary outcome measure was DCI, i.e clinical deterioration with the occurrence of focal neurologic impairment (such as hemiparesis, hemiplegia, aphasia, and so forth) or a decrease of at least 2 points on the GCS that was not apparent immediately after aneurysm occlusion and was not attributed to other causes by clinical assessment, CT or MR imaging, and laboratory studies.

To study whether brain perfusion on admission CTP scan relates to the development of DCI, we compared quantitative and semiquantitative perfusion values between patients with and patients without development of DCI after aneurysmal SAH. Additionally, we evaluated which perfusion parameter and approach (quantitative or semiquantitative) is the most promising to study as prognosticator for the development of DCI.

4. CTP Protocol-

CTP is performed on 256 slice Axial MDCT scan, PHILIPS, GERMANY, acquisition of images 4secs after bolus injection of iodinated contrast 40 ml at flow rate 4ml/secCTP parameter 80 kV, 380-430 mAs, rotation time 0.35 sec, max pitch 15 and collimation 4 mm. CTP data was acquired and collected at PHILIPS work station and analysed using LINEX software. A reconstruction of axial and coronal Maxim intensity Projection (MIP) was performed for analysis of CTP data sets and Different ROIs. In CT Perfusion five regions of interest (ROI) i.e Orbito-frontal Cortex (OFC), Internal capsule(IC), Thalamus (TH), Caudate Nucleus (CN), and Sensorimotor Cortex were drawn corresponding to white matter related to the territory of the major cerebral arteries in both hemispheres as well as deep ROIs corresponding to basal ganglia these were evaluated for cerebral blood perfusion and related indices with the assistance of manufacture's software kit of CT perfusion in the ipsilateral and contralateral hemisphere. measurements of mean transient time (MTT), time to peak (TTP), cerebral blood volume (CBV) and cerebral blood flow (CBF) were performed in each ROI.CBF was generated for each patient and expressed in ml/ 100g/ min.

CBF was measured in five elliptical regions of interest (ROI) at a size of 1-3 cm2, manually drawn on plain CT brain and averaged CTP images in the ipsilateral hemisphere and then automatically projected onto the contralateral hemisphere**(figure 1-4).** The post processing technique was standardized for all patients according to recommended guidelines with the arterial input function as the A2 segment of theACA and venous function as the superior sagittal sinus.

CTP Definitions:
CBV (cerebral blood volume) is defined as the volume occupied by intravascular blood within a particular quantity of brain tissue.7 CBV was measured in milliliter(ml) of blood per 100 gram of brain tissue.

CBF (cerebral blood flow) is defined as the volume of blood that passes through a specific quantity ofbrain tissue during a particular period of time. CBF is measured in units of mililiter of blood per 100 gram of tissue per minute.

MTT (Mean transit time)is the average period of time that blood, or an element of blood such as a single red blood cell, spends within the blood vessels in a particular part of the brain. MTT is measured in seconds and is typically on the order of 6 seconds in normal brain tissue.

Tmax- Time at which this maximum amplitude of perfusion is reached, and produce maps of that parameter, which is often simply called "Tmax."

5. Statistical analysis

The data were coded and entered into a Microsoft Excel spreadsheet. The statistical Analysis was done using SPSS version 20 (IBM SPSS Statistics Inc., Chicago, Illinois, USA) Windows software program.

CTP test characteristics (sensitivity and specificity and Positive and negative predictive value) were determined and 95% confidence internal was calculated.

Quantitative CTP data were analyzed by calculating the mean CBF, CBV, and MTT and their SDs for each outcome group comparing them from "on admission" to "on discharge". For patients with focal perfusion deficits, ROIs within the affected region were isolated and arithmetic means were calculated. In patients without focal deficits, all ROIs for all 4 section locations were included in the arithmetic means. A Student *t* test was used to determine statistical significance(for quantitative data to compare before and after observations), accepted at $P < .05$.CBF and MTT Pearson correlation was calculated with mRS.

Figure 2 shows the ROI and quantitative analysis of CBF,CBV,MTT and TTP of a patient in study.

RESULTS

A total of 50 patients were included in the study. Three patients were excluded from the study for the following reason: CTP examinations were not performed before treatment for DCI (n=3). No iv contrast related adverse reactions were encountered in any patient while paerforming CTP.

The clinical and demographic characteristics of the study population are shown in **Table 1**.

The mean age of patients was 50.0 (20–73) years. 33 patients were women and 14 were men. In all patients, a SAH was diagnosed within 24 hours after symptom onset, which was considered the moment of aneurysm rupture. Multiple aneurysms were identified in 4 patients. By dichotomizing the Hunt and Hess grade into good (Grades I–III)

and poor (Grades IV and V), we found a good Hunt and Hess grade in 20 of the 47 patients (42.5%) and 57.5% patients (27 out of 47) with poor Hunt and Hess grades. Thirty-one of the 47 patients (66%) had a Fisher Grade 3 or 4 on the initial CT scan. The ruptured aneurysm was clipped in 22 patients and coiled in 25 patients. Among 47 patients, 10 patients (21%) were DCI+. At the patient level, DCI+ was significantly associated with higher Fisher Grade.

Quantitative CTP revealed significantly reduced CBF and prolonged MTT for DCI, (P<0.05) permanent neurologic deficits, and infarction. ROC analysis showed that CBF and MTT had the highest accuracy (13).

Table 2
It is quite obvious that mean MTT at discharge has higher values as compared to admission and mean CBF at discharge has lower values as compared to admission in DCI+ patients.

On admission, cerebral blood flow (CBF), cerebral blood volume (CBV), mean transit time (MTT), and time-to-peak (TTP) did not differ between patients who did and did not develop DCI. In the DCI time-window (4 to 14 days after subarachnoid hemorrhage (SAH)), DCI was associated with a decreased CBF of 7.6 mL/min/100 g (100 % sensitivity, 71.4 % specificity, standard error: 0.02, 95 % CI: 0.84–0.88, P = 0.0001) and an increase of MTT of 0.91 seconds (83.9 % sensitivity, 79.5 % specificity, standard error 0.03, 95% CI: 0.82–0.86, P = 0.0001), respectively.

Longer perfusion times correlated to worse clinical grade at admission, higher haemorrhage volume, the occurrence of DCI and poorer outcome. This relationship is not altered if correlations are adjusted for age or the presence of hydrocephalus by the use of partial correlations. The presence of DCI is also related to lower CBF.

DISCUSSION

Our main result was the demonstration that variations of MTT and CBF values between baseline and day4 after aSAH may serve as an imaging surrogate for prediction of DCI.

In line with previous studies, we showed that aSAH patients with CTP deficits, defined as focal regions of reduced CBF and/or prolonged MTT, are more likely to experience DCI than patients with normal CTP results [14, 15, 16]

Considering as a threshold for ischaemic damage a MTT of 5.9, as suggested by Dankbaar et al. (16), the percentage of patients suffering a poor outcome is significantly larger if mMTT is 5.9 s or longer (90%poor outcome in patients with mMTT≥5.9; 30% poor outcome in patients with mMTT<5.9). Thus, having an mMTT of 5.9 or more increases the risk of suffering a poor outcome by 20(22).

Within the first 4 days after SAH, Sanelli et al. [17] demonstrated, based on a unique measure obtained at day2 after onset of SAH, that patients who further develop vasospasm have CBF reduction and MTT prolongation. Similar findings, based on a unique measure obtained at day2, in animal models [18] and patients [19–21], supported the concept of abnormal perfusion at baseline in patients who further develop vasospasm during their hospital course.

Using 1–3 months of MR-defined infarction as the primary endpoint, we found that variations of CTP values (increased MTT and decreased CBF) can be detected between day 0 and day 4/ on discharge in SAH patients who later develop DCI.

Few studies in the literature have evaluated CTP by using DCI defined as clinical deterioration(23,24,25).

Dankbaar et al investigated the diagnostic value of CTP for DCI after clinical deterioration in 39 patients with SAH*. Qualitative CTP was reported to have 84% (65%–94%) sensitivity, 79% (52%–92%) specificity, and 88% (69%–96%) positive and 73% (48%–89%) negative predictive values.

In our study, CTP deficits occurred more often in patients with DCI (P_.0001) and had test characteristics similar to those previously reported. We found 81% (68%–90%) sensitivity, 83% (70%–91%) specificity, and 83% (70%–91%) positive and 82% (69%–90%) negative predictive values.

Dankbaar et a (l4,15) reported significantly reduced CBF and prolonged MTT in patients with DCI.

Our study suffered from several limitations. First, DCI may occur outside the scanned area, although no such case occurred in the present study. The hypoperfused areas on CTP may have been only transient either because the patient received treatment or because they resolved spontaneously. The accuracy of CBF and MTT calculations may have been affected by the data processing.

Also We acknowledge several other limitations in this study.However, emerging CT technology should continue to improve and provide broader coverage to which these findings may apply. Another limitation is that the region of CTP imaging was not coordinated with the location of the region referable to symptoms.

Therefore, it is conceivable that this region may not have been imaged on CTP or was possibly averaged with a larger region of normal perfusion, resulting in increased false-negatives and lower accuracy in our study. We did not use clinical information to select the region of imaging, to reduce observer bias before CTP scanning and interpretation.

CONCLUSION

In conclusion, assessment of early MTT and CBF value variations between the day the aneurysm ruptured and day4 after SAH/ on discharge, obtained on CTP, may serve as an imaging surrogate for prediction of DCI in patients with SAH. Future research is needed to further evaluate these findings in a prospective clinical trial assessing the impact of CTP on patient outcomes.CTP may provide hemodynamic evidence to support the diagnosis of DCI and prognostic information of poor outcomes in patientswith aneurysmal SAH. CTP deficits occurred more often inpatients who developed DCI, permanent neurologic deficits, and infarction.

Even with current treatment regimens, patients with DCI are more likely to have worse outcomes, with increased morbidity and mortality. Hence, CTP is a rapid imaging technique that allows early identification of patients at risk for developing DIND and tissue at risk for vasospasm-induced infarction within days after aSAH. On the basis of this study's findings, a routine use of whole-brain CTP in the acute phase of aSAH seems to be useful.

Tables and Figures
Table 1:

Table 1 Clinical characteristics of 47 patients with SAH and Early CT perfusion

	Total Population	DCI+ Population	DCI- Population	P
No. of patients	47	10 (21 %)	37 (79 %)	-
No. of women	33	6 (13 %)	27 (57 %)	0.45
Age, years, mean (range)	50 (20–73)	53 (36–63)	50 (20–73)	0.48
Admission WFNS score				
1–2	31 (66 %)	2 (4 %)	29 (62 %)	
3–5	16 (34 %)	7 (15 %)	9 (19 %)	0.004
Fisher score				
1–2	16 (34 %)	1 (2 %)	15 (32 %)	1
3–4	31 (66 %)	9 (19 %)	22 (47 %)	0.13
Aneurysm location				
Anterior circulation	42 (89 %)	10 (21 %)	32 (68 %)	
Posterior circulation	5 (11 %)	0	5 (11 %)	1
Aneurysm treatment				
Endovascular	25 (53 %)	4 (8.5 %)	21 (44.5 %)	1
Aneurysmal clipping	22 (47 %)	6 (13 %)	16 (34%)	1
3-month mRs				
0–2	40 (85 %)	5 (11 %)	35 (74 %)	0.07
3–5	7 (15 %)	5 (11 %)	2 (4 %)	0.003

DCI = delayed cerebral infarction;

mRs = modified Rankin score;
WFNS = World Federation of Neurological Surgeons

Table 2:
Table 2 CT Perfusion values

	DCI+	DCI-	P
Patients, n	10	37	-
Regions of Interest, n	32	897	-
CBF values (ml/100gm/min)			
On Admission			
Mean values ± SD, sec	62.24 ± 14.55	62.05 ± 24.84	0.89
On Discharge			
Mean values ± SD, sec	40.80 ± 12.84	70.98 ± 30.25	<0.0001
MTT values (sec) On Admission			
Mean values ± SD, sec	6.54 ± 0.77	6.32 ± 1.48	0.82
On Discharge			
Mean values ± SD, sec	7.28 ± 0.68	5.89 ± 1.19	<0.0001

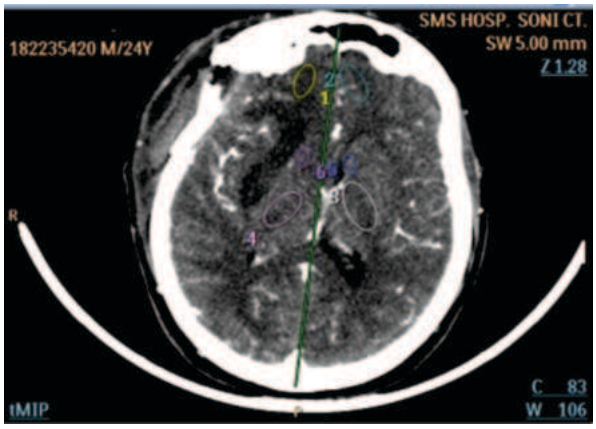

Figure 1:

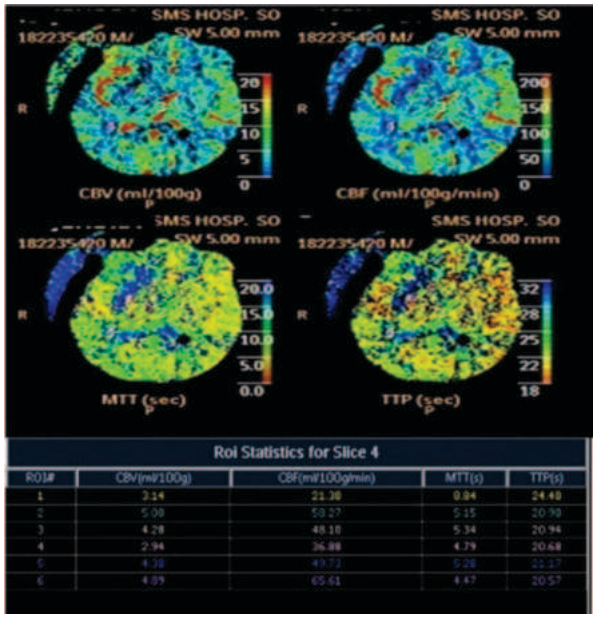

Figure 2:

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