Ischemic Stroke in Evolution: Predictive Value of Perfusion Computed Tomography

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Background: Various perfusion computed tomography (PCT) parameters have been used to identify tissue at risk of infarction in the setting of acute stroke. The purpose of this study was to examine predictive value of the PCT parameters commonly used in clinical practice to define ischemic penumbra. The patient selection criterion aimed to exclude the effect of thrombolysis from the imaging data. *Methods:* Consecutive acute stroke patients were screened and a total of 18 patients who initially underwent PCT and CT angiogram (CTA) on presentation but did not qualify to receive thrombolytic therapy were selected. The PCT images were postprocessed using a delay-sensitive deconvolution algorithm. All the patients had follow-up noncontrast CT or magnetic resonance imaging to delineate the extent of their infarction. The extent of lesions on PCT maps calculated from mean transit time (MTT), time to peak (TTP), cerebral blood flow, and cerebral blood volume were compared and correlated with the final infarct size. A collateral grading score was used to measure collateral blood supply on the CTA studies. *Results:* The average size of MTT lesions was larger than infarct lesions ($P < .05$). The correlation coefficient of TTP/infarct lesions ($r = .95$) was better than MTT/infarct lesions ($r = .66$) ($P = .004$). *Conclusions:* A widely accepted threshold to define MTT lesions overestimates the ischemic penumbra. In this setting, TTP with appropriate threshold is a better predictor of infarct in acute stroke patients. The MTT/TTP mismatch correlates with the status of collateral blood supply to the tissue at risk of infarction. **Key Words:** Computed tomography—stroke—perfusion—mean transit time—time to peak.
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Introduction

Perfusion computed tomography (PCT) has been increasingly advocated to guide optimal management strategies in acute stroke. PCT is widely accessible in emergency rooms, and its advantages over perfusion magnetic resonance imaging (MRI) include shorter imaging times and a lower cost.[1] PCT allows rapid evaluation of cerebral perfusion by generating maps of cerebral blood flow (CBF), cerebral blood volume (CBV), mean transit time (MTT), and time to peak (TTP). These parameters are derived from PCT source data based on central volume principles.[2-6]

PCT parameters are used to distinguish irreversibly damaged infarct core tissue from potentially reversible ischemic tissue (ie, the ischemic penumbra).[7] The presence and extent of salvageable ischemic penumbra is the mainstay of appropriate patient selection for thrombolytic therapy especially beyond the 3-hour time window.[8-13] In recent years, various methods have been proposed to estimate the amount of at-risk ischemic tissue, using different CBF, MTT, TTP, and CBV thresholds.[14-26] One proposed method uses relative MTT values above 145% to define at-risk ischemic tissue.[20] MTT values are increased in at-risk ischemic tissue because of reduced blood flow, and CBV values are lowered within the infarct core because of loss of autoregulation.[20]

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Current methods used for MTT calculation have recently been scrutinized regarding their accuracy.[27-29] The sensitivity of processing algorithms to contrast delay is a potential pitfall that could result in overestimation of the ischemic penumbra by some software packages.[28,29] In the present study, we examined the predictive value of PCT parameters for identifying the evolution of at-risk tissue using a commercially available delay-sensitive software. For this purpose, we selected a group of acute stroke patients who underwent PCT imaging and did not qualify for thrombolytic therapy. This selection criterion aimed to represent the natural history of infarct evolution and exclude the effect of thrombolysis from the imaging data. For each patient, we compared the size of ischemic lesions derived by various PCT parameters to the size of the infarct on follow-up imaging studies (noncontrast CT or MRI performed in 1-7 days later).

Materials and Methods

Patients

PCT imaging data were obtained as part of standard clinical stroke care and were retrospectively reviewed with the approval of institutional review board at the University of Massachusetts. Patients with suspected acute stroke and no known renal insufficiency or allergy to contrast agent underwent the following imaging protocol on presentation: noncontrast CT, a single PCT slab of 4 cm width, and computed tomographic angiogram (CTA) of the cervical and intracranial vessels. This study was based on patients who presented from January to December 2009. We excluded all patients who received tissue plasminogen activator. A total of 18 acute stroke patients were found who initially underwent PCT and CTA on presentation but later did not qualify to receive thrombolytic therapy. All these patients had follow-up noncontrast CT or MRI to delineate the extent of their acute infarct.

Imaging and Data Processing

The PCT images were obtained using a Brilliance 64-channel scanner (Philips Medical Systems) and consisted of 4 axial 10-mm-thick sections obtained above the orbits toward the vertex to protect the lenses. Images were acquired and reconstructed at a temporal sampling rate of 1 image per second, resulting in a series of 45 images for each assessed section. According to our PCT imaging protocol, a 40-mL bolus of nonionic iodinated contrast agent (Isovue-370; Bracco Inc., Monroe Township, NJ) were used in all patients, administered into an antecubital vein by a power injector at an injection rate of 4-5 mL/sec. The acquisition parameters were 80 kVp and 100-150 mAs. CT scanning was initiated 6-7 seconds after start of the contrast bolus injection. Follow-up imaging used in this study was noncontrast CT images (5-mm-thick sections obtained by Brilliance 64-channel CT scanner; Philips Medical Systems) or diffusion-weighted MR sequence (5-mm-thick sections obtained by a General Electric HDxt 1.5-T MR imaging system). The follow-up imaging for each patient (CT or MRI) and the time it was obtained from onset of stroke symptoms are listed in Table 1.

PCT data were analyzed using a deconvolution software developed by Philips Medical Systems (EBW version 3.0.1.3200). For each axial section, the software generates color-coded maps of different PCT parameters including MTT, CBF, CBV, and TTP. CBV is measured in units of milliliters of contrast material per 100 g of brain and is defined as the volume of blood for a given volume of brain. MTT and TTP are measured in seconds and defined, respectively, as the time contrast material takes to transit through a given volume of brain and achieve maximum enhancement. CBF is measured in units of milliliters of contrast per 100 g of brain tissue per minute and is defined as the volume of blood moving through a given volume of brain in a specific amount of time (CBF = CBV/MTT). The PCT maps along with follow-up CT (or MR) images were imported into Image J (Image processing software; version 1.43 m; National Institutes of Health, Bethesda, MD) for further analysis. All the imported images were in a 512 × 512 pixel matrix. The CT (or MR) images were coregistered to the corresponding PCT images by using ImageJ volume viewer plugin. The process included (1) volumetric conversion of the CT and MR images and (2) 3D adjustment of the cross-section plane orientation to match the PCT images. The selected slices were visually inspected for proper anatomical orientation by overlapping to corresponding PCT images. Infarct lesion in each section was defined as areas of hypodensity in CT and hyperintensity in diffusion-weighted images (confirmed by decrease in the apparent diffusion coefficient). These areas were demarcated manually by ImageJ freehand selection tool (measured in pixels) and subsequently converted to square centimeter. For PCT parameters, lesion area was identified as the color pixels representing the maximum 20% range of values on MTT and TTP color-coded maps and the minimum 20% range of values on CBF color-coded maps. The MTT, TTP, and CBF lesions were then defined relative to the contralateral normal hemisphere. For this purpose, (1) areas in the corresponding regions of the contralateral hemisphere were subtracted from the lesion if they had values similar to the lesion area and (2) remaining values within the lesion were expressed as percentage of values from the corresponding regions of the contralateral (normal) hemisphere. These values were above 145% for MTT lesions, above 113% for TTP lesions, and below 20% for CBF lesions in all the patients. The CBV lesions were defined by their absolute size on the CBV maps, and the values obtained were below 2 mL/100 g in all the

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