The evaluation of whole-brain CT perfused blood volume following the acute onset of an aneurysmal subarachnoid hemorrhage

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Authors: S. Li; Shenyang/CN
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Purpose

It is crucial to determine the degree and the extent of cerebral vasospasm following acute onset of SAH and assist clinicians and physicians in developing acute management plans in a timely manner. As a standardized clinical practice, nonenhanced CT (NECT) combined with either perfusion CT (PCT) \cite{1} with CT angiography (CTA)\cite{2} or multisection CTA (MSCTA) has become available in most medical hospitals and served as a reliable and more accurate method with high sensitivity and specificity to investigate anatomic vasculature following ischemic insults including SAH. However, depending on whether 16 or 64 slice CT is employed, the spatial resolution or anatomical coverage of PCT is limited to 2 to 4cm, therefore, it is impossible to evaluate the status of perfusion of the whole brain following ischemic insults. As a result, the brain perfusion deficit is usually estimated for a small region which supplied by the affected blood vessels, and little information regarding hemodynamic changes secondary to SAH in whole brain is provided yet. More recently, two studies from the same group have demonstrated that using whole brain perfused blood volume (PVB) CT, PVB maps representing changes in whole brain perfusion following ischemic insults can be acquired simultaneously during perfusion CT\cite{3,4}.

In the present study, we assessed changes in perfused blood volume following acute onset of SAH using whole brain perfused blood volume imaging techniques.
Methods and Materials

29 patients with SAH and 5 patients without SAH were first confirmed by nonenhanced CT scan. All PBV maps were acquired with 48 hours after the onset of SAH. Patients with pathological changes that could affect the accuracy of measurement of PBV values were excluded including occlusion or stenosis of cerebral or carotid arteries. A total of 28 aneurysms were detected in 27 out of 29 SAH patients. The remaining two were diagnosed with spontaneous SAH. All SAH patients underwent the standardized treatment including acute pain management, correction of electrolytes imbalance and hypovolemia as well as administration of nimodipine. SAH patients with aneurysm were treated with surgical clipping. All patients underwent NECT first to establish the baseline imagining and the following parameters were employed: 120KVP, 139mAs, slice thickness 1.0 mm, interval for reconstruction of imaging: 0.7 mm, dose-length product (DLP): 210mGy.cm). Scan direction was from the skull base to the vertex to ensure the coverage of the entire brain. For each series, a 80ml bolus of nonionic contrast was intravenously injected at a rate of 4ml per second into an antecubital vein using a power injector, and followed by a 50ml of saline washout at the same rate. Whole brain PVB Imaging acquisition begun 10 seconds post-injection of the contrast and the same parameters mentioned above were used. Then whole brain PVB maps were reconstructed and the sensitivity for detecting hypoperfusion deficts using NECT imagining and PVB map was assessed and compared. Additionally, PVB value for the regions of interest (ROIs) selected from each of five lobes of the brain and cerebellum in the SAH and non-SAH patients as well as SAH survival and non-survival patients were compared, respectively. Furthermore, correlation of the blooding dominance hemisphere detected on NECT and PVB hypoperfusion was examined.
Results

PVB maps were obtained from all 34 patients who underwent imaging studies successfully. Among 29 SAH patients, areas with hypoperfusion can be identified visibly in 18 patients whose NECT scan revealed no abnormalities. Chi-square test indicated that whole brain CT PVB can serve a sensitive indicator for detecting a subtle and focal ischemic lesion following acute onset of SAH ($\chi^2 = 26.526, p=0.000$).

Mean PVB value obtained from those SAH patients (n=24) for whom anti-spastic agent was not administrated is lower than that of non-SAH patients (n=5) in all the lobes of the brain measured, with statistical significant difference being observed in the frontal, parietal and occipital lobes (p<0.05), respectively. The results of mean PVB values of each lobe of the brain and the cerebellum from SAH and non-SAH patients is shown in Table 1 on page . Additionally, we assessed mean PVB value from SAH patients who survived or died following acute onset of SAH, the results was shown in Table 2 on page . Although mean PVB value tends to be lower in all ROIs in non-survival group (n=5) when compared to the survival group (n=24), no statistical difference was determined.

When examining NECT imaging of SAH patients, the bleeding dominance hemisphere with hyperattenuating lesions can be detected in 17 SAH patients and the distribution of bleeding dominance hemisphere and PVB hypoperfusion was shown in Table 3 on page . Interestingly, the hypoperfusion observed in PVB maps are not always seen in the same side where bleeding is dominated following SAH. For example, the bleeding dominance hemisphere and PVB hypoperfusion were observed in the same right side in only 5 (29%) and the same left side in only 3 (18%) SAH patients, respectively, and the typical example of PVB imaging obtained from a SAH patient was shown in Figure-2 on page . The mismatch of bleeding dominance hemisphere and corresponding PVB value was observed in the remaining 9 SAH patients. Fisher Exact test shows PVB hypoperfusion abnormalities does not always occur in the same side where bleeding is initiated ($\chi^2 = 0.0283, p=0.1940$, Phi Coefficient=0.4994).
Conclusion

Our results suggested that PVB maps can reveal hemodynamic changes in perfusion of the entire brain in the patients with acute onset of SAH, and whole brain PVB CT is a very useful technique that can assess the status of cerebral hemodynamics and the whole brain perfusion following acute onset of SAH.
References


