Case Report

Misidentification of Infarct Core by Computed Tomography Perfusion (CTP) in a Patient with Acute Ischemic Stroke with Hypoxia

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The authors reported a case with acute right, middle cerebral artery infarct in which the early infarct area was detected by CTP recovery later after the treatment. The patient had hypoxia and tachypnea from pulmonary edema during acute ischemic stroke, which may have accentuated the ischemic change in cerebral hemodynamic. With treatment, the causes of hypoxia were corrected and the patient got better and some parts of the brain, previously identified as infarct, turned out to be normal.

Keywords: *Infarct, Computed tomography perfusion, Ischemic stroke, Hypoxia*

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Multimodal computed tomography (mCT), which includes non-contrast CT, CT perfusion (CTP) and CT angiography (CTA), has been increasingly used in research and clinical practice. The time frames for revascularization therapy are limited, with up to 4.5 hours after stroke onset for intravenous thrombolysis, 6 hours for intra-arterial thrombolysis and 8 hours for mechanical thrombectomy[1]. However, the onset of symptoms is not precisely known in a portion of patients or in some situations, such as clinically large, middle cerebral artery infarction, where knowing the size of infarct core may help in guiding the treatment. Advanced imaging techniques, such as CTP, help to identify irreversible infarct and tissue at risk (penumbra). Thus, addition of CTA and CTP may further improve detection of infarct, identify candidates who will have the best functional outcome after thrombolysis and may further widen the time window for endovascular therapy.

Several pitfalls in interpretation of CTP were reported[2]. To my knowledge, there was no report about misidentification of infarct core by CTP in a hypoxic patient with acute ischemic stroke.

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Case Report

A 56-year-old male presented with left hemiparesis 2 hours before arrival. He had underlying hypertension. On physical examination, blood pressure was measured at 142/102 mmHg, respiratory rate 25/min, heart rate 110-130/min, irregular rhythm, no fever, drowsy, dyspnea, right-gaze preference, left hemiparesis grade 2, left-sided neglect and hemisensory loss. National Institute of Health Stroke Score (NIHSS) was 15. Multimodal CT was performed 109 minutes after stroke onset, using 64-slice CT scanner (Philips Brillance 64). Sixteen contiguous slices at 5 mm thickness for a total of 80 mm were covered. Iodinated non-ionic contrast material of 50 ml was used with the injection rate 4 ml/sec. Color-coded perfusion maps revealed cerebral blood volume (CBV), mean transit time (MTT) and cerebral blood flow (CBF). The ischemic tissue (penumbra) showed increased MTT with decreased CBF and normal or mildly increased CBV, whereas infarct tissue showed markedly decreased CBF, CBV and increased MTT[2]. CTP map program used a threshold for infarct core when CBV is less than 2 ml/100 g (2 ml/100 gm) and for ischemic tissue when MTT is over 150%. CTP of the patient showed right, middle cerebral artery distribution ischemia and infarct (Fig.

1A-D). Calculated penumbra and infarct area were 44.16 and 37 cc, respectively (total ischemic volume 81.16 cc). CTA revealed decreased opacification and visualized branches of post bifurcation M1 segment of the right middle cerebral artery, suggestive of severe stenosis (string sign) around bifurcation. Intravenous recombinant tissue plasminogen activator was given at 130 minutes after stroke onset. Oxygen saturation was measured by pulse oximeter, which revealed 80-88% at room air and increased to 90-91%, 92-96% with nasal cannula (3-5 liters of oxygen per minute) and oxygen mask with bag 10 L/min, respectively. Chest x-ray showed bilateral reticulo-nodular infiltration and cardiomegaly. Amiodarone was infused intravenously to slow the rate of atrial fibrillation. Patient was finally intubated and had clinical improvement after the treatment of congestive heart failure. Follow-up CT brain was performed a day after rtPA treatment which showed acute infarct at right frontal lobe. His neurological conditions improved with NIHSS of 3 at discharge. A month after stroke onset, the patient was able to walk and perform all previous activities (modified Rankin Scale 1).

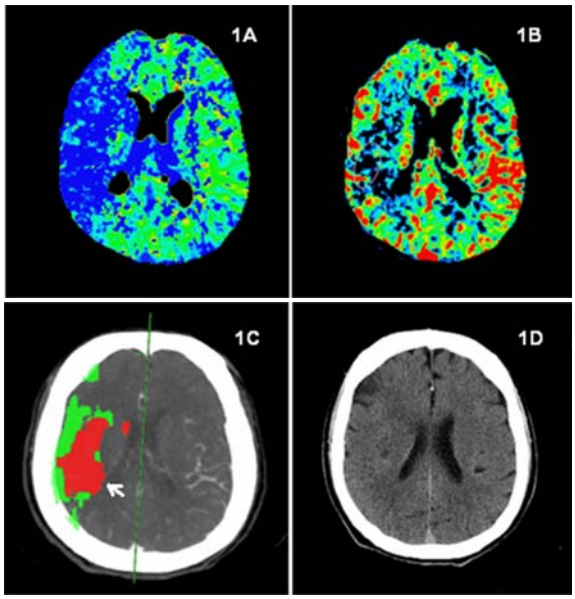

Fig. 1 CTP of a reported patient shows recovery of ischemia at most areas which are previously identified as infarct. A) MTT map, B) CBV map, C) Perfusion map, D) CT a day after rtPA treatment.

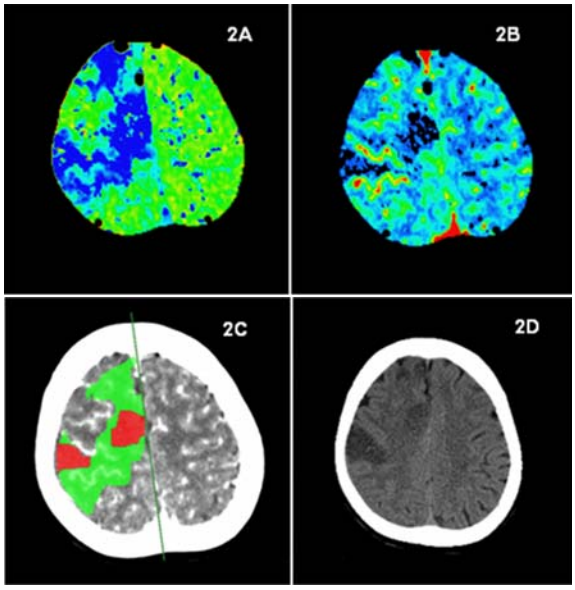

Fig. 2 CTP of another patient, a 68-year-old female, who presents with clinical right middle cerebral ischemia, shows well-matched infarct area which is identified from CTP. A) MTT map, B) CBV map, C) Perfusion map, D) CT a day after rtPA treatment.

Discussion

CTP is able to detect hemodynamic lesions within cerebral hemispheres with the sensitivity of 90%[3]. Infarct core, early detected by CBV, seems to correlate well with the true infarct (Fig. 2A-D). Some infarcts can be missed on the CTP, especially in the case of small infarcts or small subcortical infarcts or when the infarct area is outside the volume of coverage[4]. Campbell BC et al suggested that specificity of detection of infarct core reduced due to low CBF/CBV in non-infarcted white matter in cases with reduced contrast bolus intensity and leukoaraiosis[5]. The authors reported a case in which abnormality in CBV did not represent the early infarct or might not represent irreversible tissue injury, especially if the patient had hypoxia. Studies about cerebrovascular reactivity in patients during acute stroke are very limited. However, studies show that cerebrovascular reactivity is impaired following subarachnoid hemorrhage. In most patients with subarachnoid hemorrhage, the response to hypercapnea is weak because of maximal vasodilatation of small arteries and arterioles compensating for ischemia, but the reaction to hypocapnia is normal[6]. In the patient in our case report, hypoxia induced tachypnea as a compensatory mechanism, which may have caused hypocapnia in this patient. A PaCO2 drop may have induced vasoconstriction in small arteries and added to the effects of previously decreased

cerebral blood flow from the tight blockage in proximal large artery. In addition, this patient was complicated by poor cardiac pumping due to congestive heart failure with rapid-rate atrial fibrillation. This may explain overestimation of the infarct area. After correction of underlying systemic conditions, hemodynamic improved, and also due to the effect of thrombolysis, the ischemic brain tissue partly recovered with the residual infarct of 51 cc from a follow-up CT.

The authors reported a case with acute right, middle cerebral artery infarct in which early infarct area detected by CTP recovered later after treatment.

Potential conflicts of interest

None.

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ความผิดพลาดในการวินิจฉัยเนื้อสมองตายจากการตรวจเอกซเรย์คอมพิวเตอร์เพอร์ฟิวชันในผู้ป่วยโรคหลอดเลือดสมองตีบและอุดตันระยะเฉียบพลันที่มีภาวะออกซิเจนต่ำ

พรภัทร ธรรมสโรช, อาวีมาศ วัชรากร

รายงานผู้ป่วยโรคหลอดเลือดสมอง middle cerebral ด้านขวาตีบและอุดตันระยะเฉียบพลัน ซึ่งได้รับการตรวจพบรอยโรคสมองขาดเลือดและสมองตายด้วยเอกซเรย์คอมพิวเตอร์เพอร์ฟิวชัน ผู้ป่วยในขณะนั้นมีอาการหอบเหนื่อยและระดับออกซิเจนต่ำจากปอดบวมน้ำร่วมด้วย โดยภาวะร่วมดังกล่าวอาจส่งผลให้เกิดการเปลี่ยนแปลงในระบบไหลเวียนโลหิตในสมอง หลังจากผู้ป่วยได้รับการรักษาด้วยยาสลายลิ่มเลือด ทางหลอดเลือดดำและแก้ไขภาวะออกซิเจนต่ำ ผู้ป่วยอาการดีขึ้น ผลการตรวจติดตามด้วยเอกซเรย์คอมพิวเตอร์สมองพบว่าบางส่วนของสมองตายที่พบในการตรวจด้วยเอกซเรย์คอมพิวเตอร์เพอร์ฟิวชันได้กลับมาเป็นปกติ
