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Semiquantitative dynamic computed tomography to predict response to anti-platelet therapy in acute cerebral infarction

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Abstract We investigated whether dynamic computed tomography (CT) in patients with acute cerebral infarction could identify patients likely to respond to anti-platelet therapy. Seventy patients underwent semiquantitative dynamic CT within 6 h as well as cerebral angiography. All then received anti-platelet therapy with a thromboxane A2 synthetase inhibitor. Peak value (pv) and time-to-peak (tp) (time–density curves) for the Sylvian fissure were extracted from dynamic CT data and standardizing interpatient data, two indices, PV/TP index and TP index, were prepared following a standard semiquantitative manner. Both PV/TP index and TP index were effective in discriminating between 48 responders (modified Rankin scale (mRS): 0 to 2) and 22 non-responders (mRS: 3 to 5, or death: 6; both $P < 0.0001$). High PV/TP index (≥0.8) was a strong indicator of favorable response. Most of these patients maintained regional cerebral blood flow (rCBF) via anterograde flow or collaterals, with a TP index ≤ 1.1. Low PV/TP index (≤0.4) predicted non-response associated with increased TP index (>1.1) and non-compensated rCBF. Intermediate PV/TP values could not predict outcome. Dynamic CT prior to therapy can identify patients with acute cerebral infarction who are treatable with anti-platelet therapy alone.

Keywords Anti-platelet therapy · Therapeutic response · Acute cerebral infarction · Stroke · Dynamic computed tomography

Introduction

Several clinical trials have shown that thrombolysis may increase the proportion of patients with acute cerebral infarction who survive and are able to live independently. However, controversy persists concerning whom to treat. On the other hand, effective inhibition of thromboxane A2 (TXA2) synthetase by imidazole and its derivatives was demonstrated in the late 1970s [1]. Such pharmacological evidence led to the development of sodium ozagrel in the 1980s [2]. This agent, a selective inhibitor of TXA2 synthetase, maintains cerebral perfusion to the affected territory by reduction of platelet aggregation and prevention of vasospasm [3]. Use of this agent represents conservative anti-platelet (AP) therapy in contrast to aggressive measures such as thrombolysis.

Ability to predict the effectiveness of AP therapy would help in refining indications for more aggressive therapeutic intervention such as thrombolytic recanalization. At present, most stroke specialists agree that the severity of neurological deficit on admission [i.e., the National Institutes of Health Stroke Scale (NIHSS)] is a reliable predictor of stroke outcome. Radiological prediction of stroke outcome, on the other hand, has been based on demonstration of decreased regional cerebral blood flow (rCBF). Using positron emission computed tomography (PET) and single photon emission computed tomography (SPECT) several investigators related therapeutic responses to rCBF in the ischemic

area [4, 5, 6, 7, 8]. However, since the availability of such methods and the time window for thrombolysis are both limited, many stroke patients are unable to benefit from such nuclear medicine procedures.

Recently, the contribution of diffusion- and perfusion-weighted images using high-speed magnetic resonance imaging (MRI) to the diagnosis of cerebral infarction has been emphasized. Although many clinicians recognize the diagnostic accuracy of these techniques, the need for MRI in therapeutic decision making in acute cerebral infarction has yet to be firmly established [9]. Computed tomography (CT) often remains the first choice for diagnostic imaging in acute cerebral infarction [10, 11, 12]. CT depends on quantitative data expressed per pixel as Hounsfield Units (HU), facilitating quantitative analysis based on concentration of contrast medium and amount of increase in HU in the region of interest (ROI). If the technical advantages of CT are considered, dynamic CT using contrast medium holds promise as a way to provide information reflecting rCBF in the ischemic region and may provide an effective prognostic index. Despite this promise, however, few CT studies have focused on prediction of response to AP therapy.

The present study was undertaken with the aim of first determining whether patients with acute cerebral infarction treatable with AP therapy alone can be identified by dynamic CT, and then elucidating the relationship between therapeutic prognosis according to dynamic CT and specific hemodynamic findings.

Methods

Patients and therapy

One hundred nineteen consecutive patients with acute cerebral infarction in the anterior territory, suspected according to neurological signs and symptoms, underwent routine (non-contrast) as well as dynamic CT within 6 h of onset. Subsequently, to confirm the presence or absence of vascular stenosis and occlusions as well as to assess the degree of collateral formation, conventional cerebral angiography was performed in all patients.

Patients with acute cerebral infarction in the territory of the middle cerebral artery (MCA) including lacunar strokes, who were treated conservatively with a specific inhibitor of TXA2 synthetase, were eligible. Under the strict condition that a patient preferring thrombolysis could refuse AP therapy, the hospital ethics committee approved the study protocol. The number of eligible patients who gave informed consent was 70 (45 male, 25 female; mean age: 68.3 years). This eligible group included 26 patients with acute lacunar stroke in territory of perforating branches. The remaining 49 patients were excluded because of ischemic stroke in the posterior territory (not related to the MCA), non-ischemic disease (hemorrhage, tumor, or seizure), patient's preference, thrombolytic intervention, or early CT findings in acute cerebral infarction (obscured outline or partial disappearance of the lentiform nucleus, slight decrease in tissue density, and/or effacement of cortical sulci).

The AP therapy provided to the eligible group consisted solely of intravenously administered sodium ozagrel (160 mg/day for 2 weeks). Distinct from aggressive thrombolytic recanalization therapy with urokinase or tissue plasminogen activator (tPA), AP therapy with sodium ozagrel, which inhibits TXA2 synthetase and platelet aggregation, also is a commonly established therapy in acute cerebral infarction.

Protocol of dynamic CT

The slip-ring CT scanner used (Lemage; GE-YMS, Tokyo, Japan) was capable of high-speed helical scanning. All patients first underwent baseline CT without contrast enhancement. Subsequently, dynamic scanning began at 7 s following bolus injection (8 ml/s) of non-ionic contrast medium (100 ml of Iohexol 300 (iodine, 300 mg/vial); Daiichi Pharmaceuticals, Tokyo, Japan) via the right antecubital vein, with a scan time of 1 s and scan interval of 1 s. For each procedure, 20 continuous scans were obtained. The CT slice including the basal ganglia and pineal body was used for this study. ROIs were placed symmetrically as circles with a maximum diameter of 3 cm at the center of both the Sylvian fissures, where branches of the MCA can easily be recognized. No patient had an adverse reaction to contrast medium.

Angiographic evaluation

Two neurosurgeons without access to any clinical information evaluated all angiograms. Compensation of perfusion to the stroke area was angiographically categorized as anterograde (nearly normal anterograde perfusion); sufficient (well-developed collateral circulation without a delay of contrast); insufficient (poorly developed collateral circulation with delayed contrast); and absent (no collateral circulation).

Semiquantitative analysis and therapeutic evaluation

Raw data from dynamic CT ROIs were processed with a gamma-variate function, and time-density curves were prepared. Both peak value (pv) and time-to-peak (tp) were extracted. These two values indicate the maximum fractional vascular volume and transit time, respectively (Fig. 1).

Based on these values, PV/TP index and TP index of the affected side relative to the non-affected side were calculated using the following equations: PV/TP index = pv/tp ratio on affected side/pv/tp ratio on non-affected side; TP index = tp on affected side/tp on non-affected side. Time required to complete both CT examinations (routine non-contrast and dynamic CT) and calculation of CT indices (PV/TP index and TP index) was approximately 15 min.

Stroke severity prior to initiation of AP therapy was evaluated according to the NIHSS by two experienced neurologists. Therapeutic outcome was assessed using the modified Rankin Scale (mRS) at 30 days (independence: 0 to 2, vs. dependence: 3 to 5, and death: 6). Responders and non-responders were defined as having mRS scores of 0 to 2 and 3 to 6, respectively. Two neurosurgeons without knowledge of baseline NIHSS score, CT and angiographic findings, or clinical events, made this outcome evaluation.

Statistical analysis

Whether PV/TP index and TP index were significant predictors of response and non-response was investigated retrospectively. Comparisons of data regarding the two dynamic CT indices were performed using the Mann-Whitney U-test between groups. Statistical significance was defined as $P < 0.05$.

Results

No eligible patient showed hyperacute stroke signs on baseline routine CT. Among the 70 eligible patients

analyzed, baseline NIHSS was 9.71 ± 8.52 (mean ± SD) and average mRS at 30 days was 1.97 ± 1.80 (mean ± SD). The number of stroke-related deaths (mRS = 6) at 30 days was 2. No deaths occurred from other diseases.

In 48 responders (mRS = 0 to 2) among 70 treated patients, AP therapy was considered effective; in the remaining 22, AP therapy was ineffective (non-responder; mRS = 3 to 6). PV/TP index and TP index in responders and non-responders are shown, respectively, in Fig. 2a and b. Both PV/TP index and TP index were effective prognostic indices for response to AP therapy ($P < 0.0001$ for each). Baseline NIHSS scores of responders and non-responders were 5.8 ± 4.8 and 18.2 ± 8.6, respectively, again showing a significant difference ($P < 0.0001$). We arbitrarily classified 70 eligible patients into three groups (A: NIHSS 0–9, B: NIHSS 10–20, C: NIHSS 21–42) based on NIHSS scores. This classification is equally predictive of favorable and poor responses to AP therapy in 42 of 47 (89%) patients with group A and in 10 of 11 (90%) patients with group C, respectively.

Figure 3 shows the relationship between therapeutic outcome as assessed by mRS and dynamic CT data (PV/TP index and TP index). In most patients showing a high PV/TP index (≥0.8) a favorable therapeutic outcome (mRS 0 to 2) was obtained, while in those with a low PV/TP index (≤0.4) AP therapy was ineffective (mRS 5 to 6).

In the intermediate range of PV/TP index (0.4 to 0.8) responders coexisted with non-responders, with most non-responders in this range showing an mRS of 3 to 4. Referring to Figs. 2b and 3, we selected 1.1 as a cut-off value for TP index.

The high PV/TP index group (≥0.8, n = 42) was characterized by almost no decrease in perfusion. Thirty-nine of the 42patients (92.8%) with a high value responded well to therapy. The remaining three patients

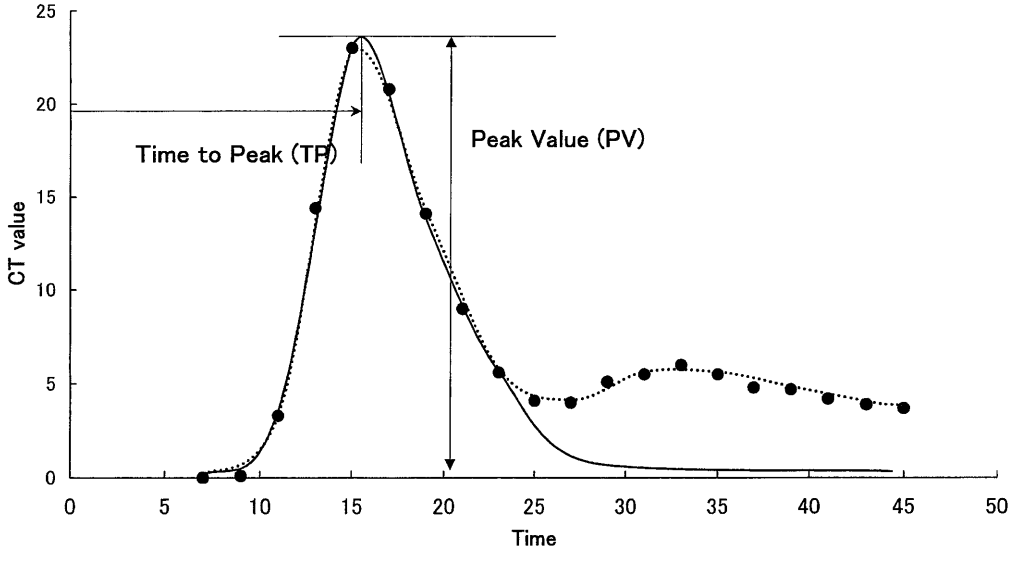

Fig. 1. Example of time-density curve (TDC). Peak value (*PV*) and time-to-peak (*TP*) were extracted from ROIs. *Dotted line* raw data obtained at ROI, *solid line* TDC

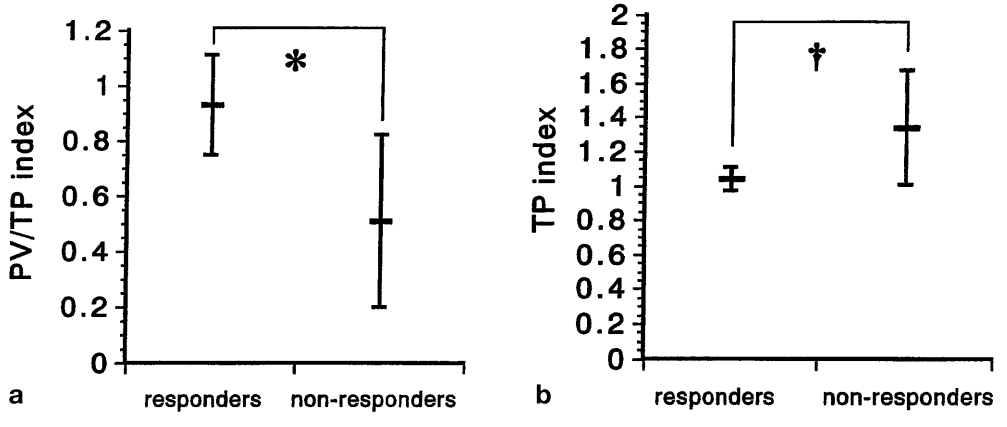

Fig. 2. **a** PV/TP index (mean ± SD) in responders (0.92 ± 0.18) and non-responders (0.50 ± 0.31). Significant difference in PV/TP index was observed between the two groups (* $P < 0.0001$), showing that PV/TP index is informative for predicting the therapeutic outcome. **b** TP index (mean ± SD) in responders (1.04 ± 0.07) and non-responders (1.34 ± 0.33). Response to therapy could be clearly separated by referring to TP index, with a statistical difference (*dagger* $P < 0.0001$)

were non-responders, despite having PV/TP values in a range where a favorable outcome would be expected. Although all had very small (lacunar) infarcts, these were located in the posterior limb of the internal capsule, a site critical to motor function. In the high PV/TP index group, TP index was ≤1.1. Cerebral angiography performed in this group indicated nearly normal anterograde flow (40/42; Fig. 4) or sufficient collateral formation (2/42; Fig. 5).

In the low PV/TP index group (≤0.4, $n = 10$), decreased perfusion was found over a wide area including the affected cortical and subcortical regions. Of the ten patients in this group, nine did not respond to AP therapy. Only one patient with a low PV/TP index responded to therapy. TP index in this group always exceeded 1.1. Angiography in this group revealed nearly total absence of collateral formation (Fig. 6).

In the intermediate PV/TP index group (0.4 to 0.8, $n = 18$), at least moderately decreased perfusion was noted, and only insufficient collateral formation could be confirmed by cerebral angiography, while compensation of the cortical blood flow in the ischemic region was decreased (Fig. 7). Eighteen patients belonged to this group; the eight responders and the ten non-responders could not be distinguished by PV/TP index ($P = 0.24$; not significant). TP index was similarly unable to distinguish therapeutic outcome (response vs. non-response) in this group ($P = 0.16$). Various angiographic findings (anterograde: 4, sufficient: 5, insufficient: 9) also were not useful in predicting outcome in this group.

Baseline NIHSS score and CT indices (PV/TP index and TP index) were significant predictors of therapeutic outcome (all $P < 0.0001$). In general, high and low baseline NIHSS scores were respectively noted in the low and high PV/TP index range, with an inverse correlation of $r = -0.69$. Of 42 in the high PV/TP index group, only five had a high baseline NIHSS (>10) despite the inverse correlation noted above. These five patients included four responders to AP therapy. Of ten patients in the low PV/TP index group, only one had a low NIHSS score (<10), suggesting a relatively minor stroke. Nevertheless, this patient did not respond to therapy, in accord with the prediction of poor outcome from dynamic CT. Baseline NIHSS scores in patients with intermediate PV/TP index remained promising as a prognostic index (responders: 5.7 ± 4.2; non-responders: 17.0 ± 7.0, $P = 0.0039$).

Discussion

In this dynamic CT study using widely available CT equipment, we succeeded in establishing indices

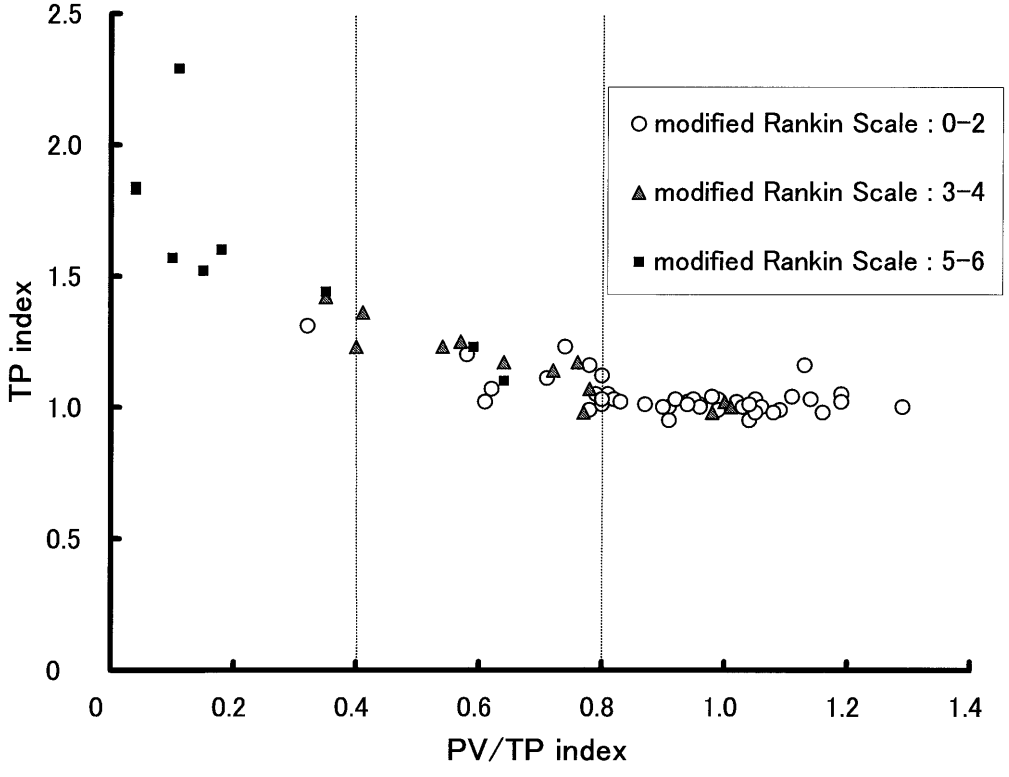

Fig. 3. Distribution of PV/TP index and TP index in relation to the therapeutic outcomes. Patients with mRS: 0–2 were defined as responders and those with mRS: 3–6 were defined as non-responders

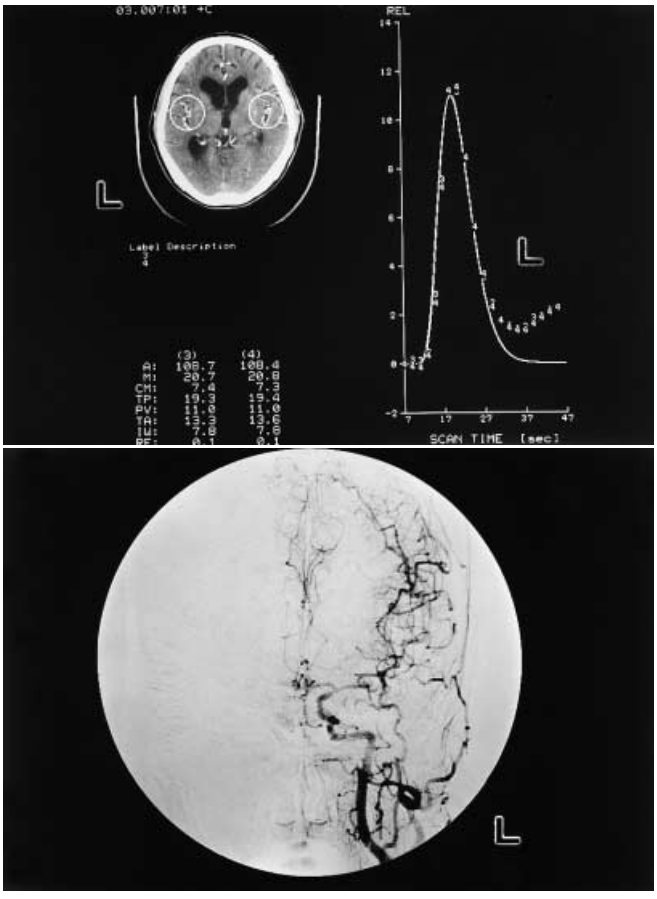

Fig. 4. High PV/TP index (1.0) patient with left cerebral infarction. *Above* time-density curve on two ROIs had no shift, which implied favorable response to AP therapy. *Below* hemodynamically, the maintenance of anterograde perfusion was found, without vascular impairment. mRS 30 days later actually showed neurological improvement (mRS: 0)

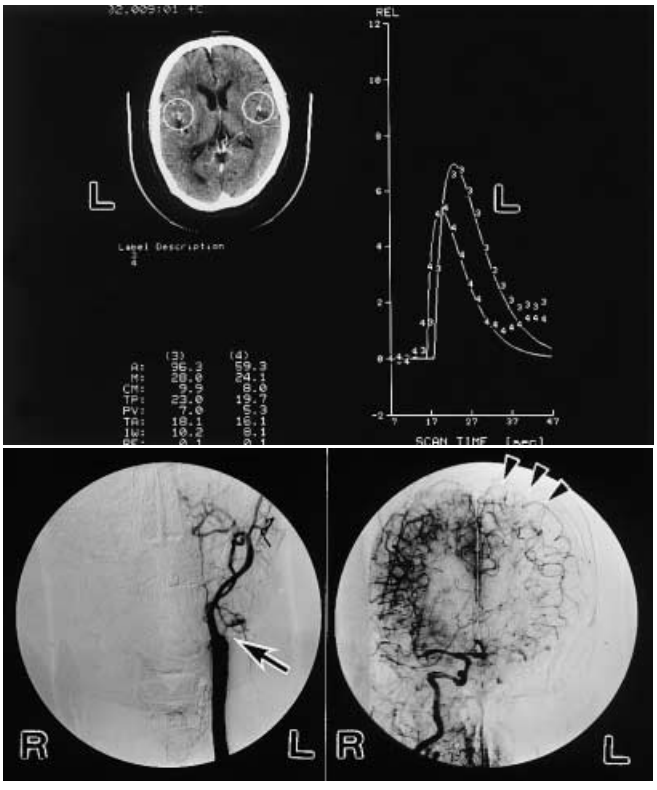

Fig. 5. Another high PV/TP index (1.13) patient with left cerebral infarction. *Above* time-density curve from affected side showed increased blood volume with prolongation of time-to-peak. *Below* left carotid angiography shows the occlusion of the internal carotid artery (*bold arrow*). Right carotid angiography also showed collateral flow sufficient for perfusion in the lesion (*arrowheads*). These findings were indicative of response to AP therapy. Therapeutic outcome was favorable response (mRS: 1)

predictive of favorable and poor responses to AP therapy in 39 of 42 (92%) patients with high PV/TP index and in nine of ten (90%) patients with low PV/TP index, respectively. The two indices that we adopted, PV/TP index and TP index, could classify most patients according to prognosis. First, a high PV/TP index group (≥0.8) could be expected to have a good outcome. Most patients in this group showed a TP index of ≤ 1.1. Second, no response to therapy could be anticipated in a low PV/TP index group (≤ 0.4). TP index in this group was ≥1.1. Third, in an intermediate PV/TP index group (0.4 to 0.8), patients responsive and not responsive to AP therapy coexisted, and could not be distinguished by TP index. Prediction using TP index alone was statistically significant, but because it combined two parameters, PV/TP index could predict the clinical outcome more reliably. Accordingly, our patients were classified mainly by PV/TP index.

As indicated in Fig. 2a, the PV/TP index cut-off value was set at 0.8, based on the distribution of groups with good and poor therapeutic outcomes. The low PV/TP index group showing no response to therapy and progression to an irreversible deficit worse than mRS 3 to 4 was defined by a cut-off value of 0.4 on the scatter plot. In the same way, referring to Fig. 2b, the cut-off value for TP index was empirically set at 1.1, based on comparison of subjects and response to therapy. Detailed mathematical analysis of methods to establish cut-off values was beyond the scope of the study.

Results of the present study suggested several important points regarding strategy of stroke treatment. A patient subgroup requiring an aggressive option such as thrombolysis can be defined more strictly if one could precisely predict outcome of more conservative (AP) therapy. We found that systematic semiquantitation of dynamic CT can identify patients treatable with AP therapy alone. However, this observation should not be misinterpreted as indicating that patients showing likelihood of response to AP therapy should be excluded

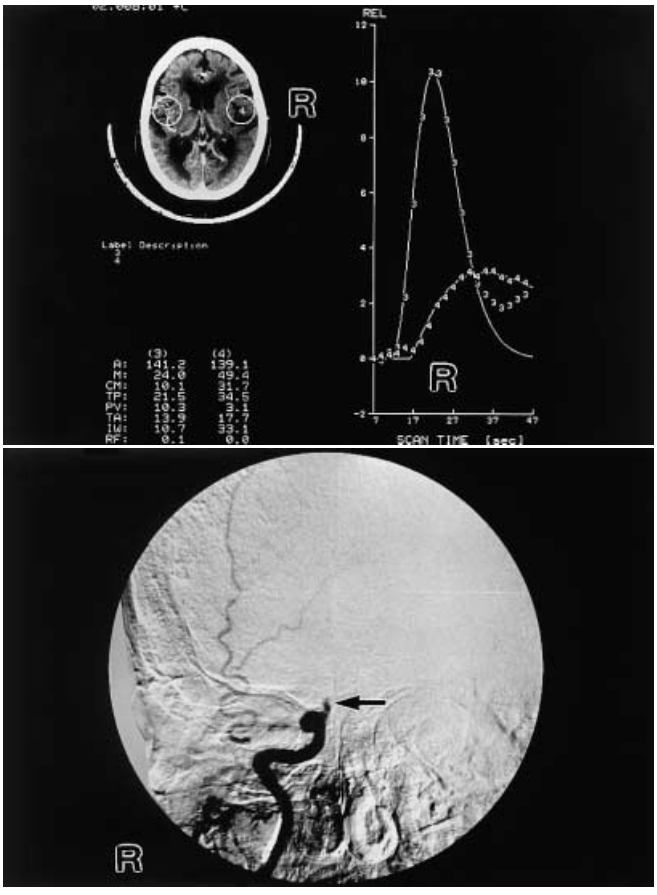

Fig. 6. Low PV/TP index (0.18) patient with right cerebral infarction. *Above* time-density curves had remarkable dissociation and phase-gap, which was suggestive of severe hemodynamic impairment. *Below* total occlusion of right internal carotid artery without compensation by collateral formation was angiographically noted (*bold arrow*). AP therapy did not improve this patient's hemiparesis (mRS: 5)

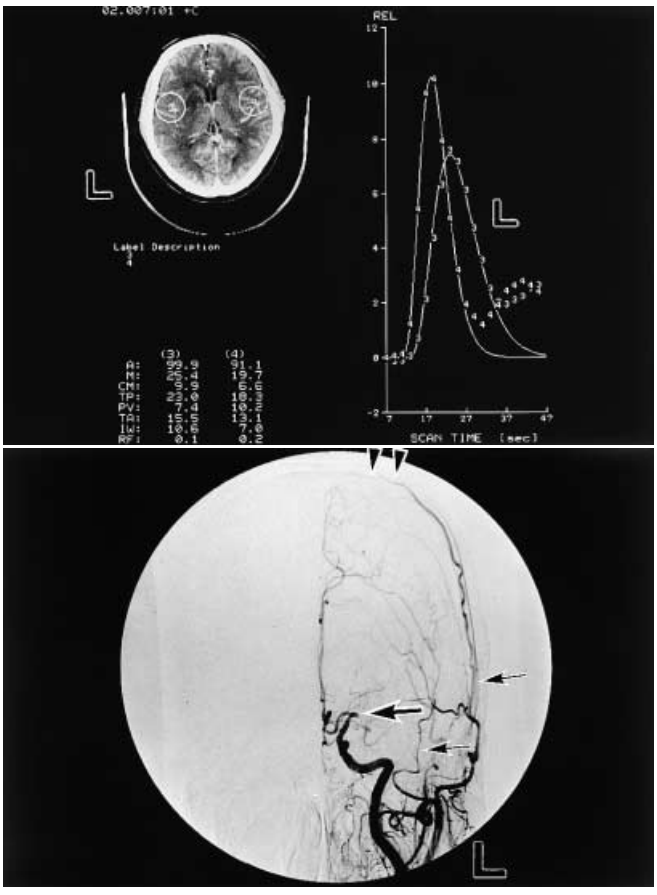

Fig. 7. Middle PV/TP index (0.57) patient with left cerebral infarction due to total occlusion of middle cerebral artery. *Above* time-density curves revealed both slight decrease in peak and phase-gap as prolongation of time-to-peak. *Below* angiography showed occlusion of M1 portion (*bold arrow*) and the presence of collateral flow (*arrowheads*) via anterior cerebral artery. Note the visualization of branches of the external carotid artery (*arrows*). Consequently, neurological deficits in this patient did not improve (mRS: 4)

from thrombolysis, since no direct comparison was made between thrombolysis and AP therapy.

Coexistence of different responses in the intermediate PV/TP index range limits the prognostic usefulness of dynamic CT. In particular, prognostic prediction within this problematic range is of importance for future stroke trials in terms of selecting candidates likely to benefit from thrombolytic recanalization. An attempt to introduce yet another CT index in this unpredictable range makes little sense and complicates the situation. In the patients analyzed here, however, baseline NIHSS score seemed to be a reliable prognostic factor of AP therapy outcome in the intermediate PV/TP index range ($P = 0.0039$).

In our study, a relationship between baseline NIHSS and PV/TP index was confirmed, with an inverse correlation at $r = -0.69$. Detailed future observation of the occasional patients who deviate from this pattern may enhance the predictive potential of dynamic CT.

Notable angiographic features in the high PV/TP index group included maintenance of cortical blood flow via arterial anterograde flow or collateral pathways. Since, in these patients, cerebral hemodynamics remain nearly undisturbed, the neurological course is likely to be favorable. In contrast, in the low PV/TP index group, virtually no anterograde blood flow or collaterals were demonstrated, while the TP index of this group was increased to ≥1.1; this suggests that the velocity of blood flow to the affected region was markedly decreased. In such patients cerebral hemodynamics are greatly disturbed, and little response to AP therapy can be expected. In the intermediate PV/TP index group, anterograde blood flow was lacking, but slight compensation was achieved via collaterals, suggesting that in this group the therapeutic time window may be somewhat wider than in the low PV/TP index group.

The most pressing issue for clinicians is how to decide swiftly and correctly from among the therapeutic options available to treat cerebral infarction in an individual patient. Despite the value of diagnostic imaging in this setting, very few studies have examined dynamic CT to determine indications for AP therapy. Using dynamic CT, Shih and Huang classified clinical outcome at 6 weeks as good, fair, or poor, in a differential analysis of their patients' PV and TP values, but these authors did not clearly state the criteria used to define outcome [13]. They emphasized that the degree of increase in CT density (HU) was a powerful predictor of outcome. In contrast to their report, we demonstrated that not only PV/TP index, which reflects increases in CT density, but also TP index (related to cerebral blood flow velocity), is a powerful predictor.

According to several representative reports relating therapeutic outcome to pretreatment rCBF measured by radioisotope imaging methods such as PET and SPECT [4, 5, 6, 14], maintenance of rCBF determined the results of non-thrombolytic therapy. Our analysis based on dynamic CT is highly consistent with the conclusions derived from these perfusion studies. However, unlike scintigraphic methods that reflect the volume of decrease of rCBF, our study expanded the scope of investigation to include hemodynamic mechanisms underlying the maintenance of rCBF, specifically formation of collaterals and observation of anterograde blood flow. Dynamic CT indices, consistent with NIHSS results, were found to be excellent predictors of outcome in our study. In comparison with NIHSS for stroke severity, the use of several kinds of dynamic CT data can identify the extent of ischemic penumbra as an area at risk.

One technical limitation of our method is that comparison of ROIs placed in the two hemispheres is of little use in patients with bilateral lesions. Although the present predictive method is based on semiquantitative numerical data, several unpredictable cases, particularly in the high PV/TP index group, highlighted the importance of the functional neuroanatomy of the lesion. Furthermore, since our method evaluates only a single tomographic slice, cases in a high-perfusion group could have perfusion deficits in more distal regions, so prognosis may be misjudged. We believe that such limitations could be overcome by obtaining multi-slice scans by multi-detector CT [15, 16].

Recently, a perfusion CT technique with radiolabeled microspheres has been proposed as a way to measure cerebral perfusion [17], while actual rCBF measurement was beyond the scope of our outcome prediction by simple semiquantification of contrast enhancement.

Considering that neurological deficits from acute cerebral ischemia sometimes show spontaneous recovery, a non-treated or placebo control group would be needed to strictly define benefits and timing of AP therapy, but this is not ethical, since potentially effective therapies exist. Likewise, our study did not include a direct comparison with the results of aggressive therapy, so the limits of AP therapy cannot be precisely defined. If thrombolytic therapy were directly compared with AP therapy, a group might be identified for whom thrombolytic therapy would be desirable despite potential response to AP therapy. However, a thrombolytic-therapy group would not be identical to the patient group studied here, and matching of subjects for multiple factors including underlying risk factors, gender, age and baseline NIHSS would be difficult.

This study had an aim of rethinking therapeutic strategies in cerebral infarction, not from a standpoint of aggressive thrombolytic measures, but from the standpoint of conservative management with an AP aggregation agent. Even in community hospitals, semiquantitative calculation by dynamic CT can be performed as a simple, practical prognostic tool. Despite inevitable basis weaknesses of our study design, the method presented may become part of future triage strategy in acute cerebral infarction.

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