MRI Pontine Hyperintensity After Supratentorial Ischemic Stroke Relates to Poor Clinical Outcome

Riitta Mäntylä, MD; Tarja Pohjasvaara, MD; Risto Vataja, MD; Oili Salonen, MD, PhD; Hannu J. Aronen, MD, PhD; Carl-Gustaf Standertskjöld-Nordenstam, MD, PhD; Markku Kaste, MD, PhD; Timo Erkinjuntti, MD, PhD

Background and Purpose—MRI studies in patients with atherosclerosis often reveal ill-defined hyperintensity in the pons on T2-weighted images. This pontine hyperintensity (PHI) does not fulfill the criteria of a brain infarct, and its clinical relevance is not established. We examined the frequency, as well as the radiological and clinical correlates, of PHI in poststroke patients.

Methods—Three hundred nineteen patients were studied 3 months after supratentorial ischemic stroke with the use of 1.0-T MRI. Brain infarcts, atrophy, white matter hyperintensities, and PHI were registered. The clinical outcome was assessed 3 and 15 months after the stroke.

Results—Of the patients, 152 (47.6%) had PHI. The risk factors for stroke did not differ in patients without or with PHI. PHI was related to a higher frequency (P<0.002) and larger volume (P<0.001) of supratentorial brain infarcts, to parietal (P=0.020) and temporal (P=0.002) atrophy, to central atrophy (P=0.040), and to white matter hyperintensity grade (P<0.001). Brain infarcts that affected the corpus striatum (putamen, caudate, and pallidum) (P≤0.011) or pyramidal tract (P<0.001) were more frequent in patients with PHI. The 3- and 15-month outcomes were worse in patients with PHI (P=0.004). The total volume of brain infarcts (OR 1.22), mean atrophy (OR 3.59), and PHI (OR 3.76) were independent correlates of a poor 15-month outcome.

Conclusions—PHI after supratentorial ischemic stroke deserves attention because it relates to poor clinical outcome. (Stroke. 2000;31:695-700.)

Key Words: brain stem ■ magnetic resonance imaging ■ stroke, ischemic ■ wallerian degeneration ■ white matter

MRI is sensitive in the detection of brain stem pathology, and it has been used to detect a group of poststroke patients presenting with pontine hyperintensity (PHI) who do not fulfill the criteria of a brain stem infarct.1,2 PHI can be seen as areas of ill-defined hyperintensity on T2, proton density (PD), or fluid-attenuated inversion recovery sequences, without or with only minor corresponding hypointensity on T1-weighted images (Figure 1). The cause and clinical significance of PHI remain less well characterized.

We performed the present study to examine the frequency, risk factors, and radiological and clinical correlates of PHI in patients with ischemic supratentorial stroke.

Subjects and Methods

Patients

The study group consisted of 319 patients from the Helsinki Stroke Aging Memory (SAM) Study, a prospective cross-sectional study of 486 consecutive persons with ischemic stroke who were aged 55 to 85 years. The detailed clinical3–5 and imaging6,7 procedures have been previously described. A total of 396 (81.5%) of the patients in the SAM cohort underwent MRI and a structured clinical and neurological examination 3 months after ischemic stroke. Of these patients, 77 had a brain stem infarct on MRI and were analyzed separately. The mean age of the patients was 70.8 years (SD 7.7, range 55 to 85 years), and 169 were women.

Risk Factors

In each patient, the case history was obtained regarding the presence of arterial hypertension, myocardial infarction, heart failure, atrial fibrillation, carotid atherosclerosis, and diabetes; previous or current smoking; and daily or weekly use of alcohol. A history of hypertension was defined as a systolic blood pressure of ≥160 mm Hg or a diastolic blood pressure of ≥95 mm Hg. Diabetes was defined as a previously documented diagnosis, the current use of insulin or oral hypoglycemic medication, or a fasting blood glucose level of >7.0 mmol/L. Carotid atherosclerosis was considered to be...
present if there was an occlusion or a clear stenosis or other atherosclerotic plaque, including an ulceration of a major extracranial or intracranial artery as seen on a carotid sonogram or angiogram. Education was divided into 2 categories: low (0 to 6 years of formal education) and high (>6 years of formal education).

MRI Examinations
MRI was performed 3 months after stroke at 1.0 T (Magnetom; Siemens). The imaging protocol included transaxial T2-weighted (repetition time [TR] 3000 ms, echo time [TE] 90 ms, number of excitations [NEX] 1), PD- (TR 3000 ms, TE 15 ms, NEX 1), and T1- (TR 400 ms, TE 15 ms, NEX 2) weighted images with conventional spin-echo technique. The angulation of slices was bicommissural, with slice thickness of 5 mm, gap of 0, field of view of 230 mm, matrix size of 256×256 pixels, and 26 slices on every pulse sequence. A 3-dimensional gradient-echo (TR 30, TE 5, α 40, NEX 1) sequence that yielded 64 coronal sections (3 mm thick) was also used.

Brain Infarcts
All MR images were reviewed by the same neuroradiologist (R.M.), who was blinded to the clinical data. The number, size, site, and type of focal lesions were recorded. Lesions that were equal to the signal characteristics of cerebrospinal fluid on T1-weighted images and measured >3 mm in diameter, as well as wedge-shaped corticosubcortical lesions, were regarded as brain infarcts. The size of the lesion was classified into 4 groups according to the diameter: 3 to 9, 10 to 29, 30 to 59, and ≥60 mm. The radii used for brain infarct volume calculations were 3, 10, 20, and 30 mm, respectively (formula for calculating the volume of ball).

The sites included lobes (corticosubcortical lesions in frontal, parietal, temporal, and occipital lobes), vascular territories (deep and superficial anterior cerebral artery [ACA], middle cerebral artery [MCA], and posterior cerebral artery [PCA] areas, as well as internal carotid artery [ICA] and border zone areas), and specific locations (putamen, caudate, pallidum, thalamus, genu of internal capsule, anterior and posterior capsules, anterior and posterior corona radiata, and anterior and posterior centrum semiovale).3-11

The histories of the patient’s previous and present strokes were reviewed together with the board-certified neurologist (T.P.) and neuroradiologist (R.M.), and the infarcts compatible with the patient’s present symptoms were defined as related infarcts.

White Matter Hyperintensities
White matter (WM) hyperintensities (WMHIs) were rated on PD-weighted images in 6 WM areas: around the frontal and posterior horns, along the bodies of lateral ventricles, and in deep, watershed, and subcortical WM areas.7

Periventricular hyperintensities (PVHIs) around the frontal and posterior horns were classified on the basis of size and shape into small cap (≤5 mm), large cap (6 to 10 mm), and extending cap (>10 mm). PVHIs along the bodies of lateral ventricles were classified on the basis of thickness and shape into thin lining (≤5 mm), smooth halo (6 to 10 mm), and irregular halo (>10 mm).

WMHIs in the subcortical, deep, and watershed areas were classified on the basis of size (greatest diameter) and shape into small focal (≤5 mm), large focal (6 to 10 mm), focal confluent (11 to 25 mm), diffusely confluent (>25 mm), and extensive WM change (diffuse hyperintensity without distinct focal lesions affecting the majority of WM area). The number of each type of WMHIs was counted, and extensive WM change was rated as absent or present.5,7

The extent of WMHIs was graded with the 4-point scale proposed by Fazekas et al.6,12,13 and the mean WMHI score was the mean of WMHI grades in different WM regions.

Pontine Hyperintensity
PHI was defined as hyperintensity without distinct borders on T2-weighted images, without or with only minor corresponding hypointensity on T1-weighted images (Figure 1). Well-demarcated hyperintense lesions in the pons on T2-weighted images with a corresponding T1-weighted hypointensity that approached the signal characteristics of cerebrospinal fluid were regarded as infarcts1 (Figure 2), and patients with these lesions were not included in the first analysis.

Brain Atrophy
Brain atrophy was rated visually from 0 to 3 (none, mild, moderate, or severe) through a comparison with standard images. Cortical and central brain atrophy were rated separately: cortical atrophy in frontal, parietal, and occipital lobes, in temporal neocortex; and entorhinal cortex (parahippocampal gyrus) and hippocampal formation as well as in cerebellum and vermis; and central atrophy in temporal, frontal, and occipital horns of the lateral ventricles, as well as the bodies, and in the third and fourth ventricles. Temporal atrophy was evaluated on 3 coronal slices (the slice showing the interpeduncular cistern and ±1 slice).14,15 The mean of the atrophy
scores (central, cortical, and temporal) was regarded as the mean cerebral atrophy value.

The intraobserver and interobserver reliabilities for the rating of PHI, WMHIs, and atrophy were tested through an independent review of 60 MRI scans by the same rater (R.M.) and by a board-certified neuroradiologist (O.S.) and a general radiologist (H.J.A.). Assessments were made in different sessions, and for the intraobserver reliability, the time interval between first and second ratings was ≥4 weeks. The intraobserver and interobserver agreements for PHI were very good (intraobserver \( \kappa =0.97 \), interobserver \( \kappa =0.93 \); R.M. and O.S.). The weighted \( \kappa \) values for intraobserver agreement were 0.90 to 0.95 for WMHIs and 0.75 to 0.82 for atrophy. The corresponding \( \kappa \) values for interobserver agreement were 0.72 to 0.84 for WMHIs and 0.61 to 0.74 for atrophy (R.M., O.S., and H.J.A.).

**Outcome**

The clinical outcome was assessed by a board-certified neurologist (T.P. or R.V.). Social functioning was assessed on the basis of patient’s ability to perform the instrumental activities of daily living (IADL) and activities of daily living (ADL) on the basis of an interview with the patient and with a knowledgeable informant, and the neurologist’s examination. Assessments were used that reflected functions before and 3 months after the index stroke. Scales that were used included the Index of ADL (rating from 0 to 7),16 the IADL Scale (maximum 8),17 the Functional Activities Questionnaire (maximum 30),18 and the Blessed Functional Activity Scale (maximum 17).19,20 In addition, the neurologist completed the Barthel Index (maximum 100).21

Stroke-related impairment was also assessed with the National Institutes of Health Stroke Scale,22 the Scandinavian Stroke Scale (maximum 58),23 and the Rankin scale (maximum 5).24 The clinical outcome at 15 months after stroke was assessed in 289 patients (25 patients had died and 5 patients could not be reached). The outcome was assessed by an interview (R.V.) with the patient, and the scales included the Barthel Index and Rankin scale.

**Statistical Analysis**

We compared patients without and with PHI. The \( \chi^2 \) test was used for categorical data, and the \( t \) test was used for continuous data. Differences in WMHI grades and in atrophy in patients without and with PHI were assessed with the Mann-Whitney \( U \) test.

Statistical tests were performed with BMDP New System 1.1, BMDP Classic,25 and SPSS for Windows 7.0.26 A level of \( P<0.05 \) was regarded as statistically significant.

**Results**

A total of 152 (47.6%) of the patients had T2 hyperintensity in the pons. In 69.1% of these patients, PHI was bilateral; in 16.4%, only the right side was affected; and in 14.5%, only the left side was affected. In 57 patients (37.5% of the patients with PHI), hyperintensity was also seen in the ipsilateral cerebral peduncle and internal capsule, suggesting Wallerian degeneration (WD) of the pyramidal tract.

The mean age, sex, level of education, and risk factors for stroke did not differ between patients without or with PHI (Table 1).

Of the PHI-positive patients, 61.6% had ≥1 cortical infarct, and 78.8% had ≥1 subcortical infarct. The corresponding values for PHI-negative patients were 59.3% and 70.1%. The difference in the prevalence of cortical (\( P=0.674 \)) or subcortical (\( P=0.075 \)) infarcts between PHI-positive and PHI-negative patients was not significant.

Patients with PHI had a higher frequency and volume of both all and related ipsilateral brain infarcts. Higher frequencies of infarcts were found in the frontal (\( P<0.001 \)), parietal (\( P<0.001 \)) and temporal (\( P=0.017 \)) lobes, as well as in the vascular territories of the superficial branches of ACA (\( P=0.018 \)) and MCA (\( P=0.002 \)) and the deep branches of

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>PHI Absent (n=167)</th>
<th>PHI Present (n=152)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (SD), y</td>
<td>70.1 (8.1)</td>
<td>71.6 (7.3)</td>
<td>0.087</td>
</tr>
<tr>
<td>Male, %</td>
<td>46.7</td>
<td>47.4</td>
<td>0.906</td>
</tr>
<tr>
<td>Low education, %</td>
<td>32.5</td>
<td>29.6</td>
<td>0.427</td>
</tr>
<tr>
<td>Risk factor, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>44.9</td>
<td>49.3</td>
<td>0.428</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>17.4</td>
<td>18.4</td>
<td>0.806</td>
</tr>
<tr>
<td>Heart failure</td>
<td>25.7</td>
<td>19.1</td>
<td>0.164</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>21.0</td>
<td>18.4</td>
<td>0.552</td>
</tr>
<tr>
<td>Carotid atherosclerosis</td>
<td>18.6</td>
<td>20.4</td>
<td>0.680</td>
</tr>
<tr>
<td>Diabetes</td>
<td>22.8</td>
<td>26.3</td>
<td>0.460</td>
</tr>
<tr>
<td>Ever smoking</td>
<td>47.3</td>
<td>46.1</td>
<td>0.725</td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td>24.0</td>
<td>23.1</td>
<td>0.873</td>
</tr>
<tr>
<td>Serum-cholesterol, mmol/L (SD)</td>
<td>6.3 (5.5)</td>
<td>5.9 (4.5)</td>
<td>0.438</td>
</tr>
</tbody>
</table>

**TABLE 1. Characteristics of 319 Poststroke Patients Without and With PHI on T2-Weighted Images**

Figure 2. Pontine infarct. Well-demarcated hyperintensity in the pons on T2-weighted images (a) with a corresponding hypointensity approaching the signal characteristics of cerebrospinal fluid on T1-weighted images (c).
MCA ($P<0.002$) and ICA ($P<0.001$). Lesions that affected the corpus striatum (putamen $P<0.001$, caudate $P=0.011$, pallidum $P<0.001$), as well as structures related to the pyramidal tract (posterior capsule $P<0.001$, corona radiata $P<0.001$), were more common in patients with PHI.

Patients with PHI had more severe cortical atrophy in parietal ($P=0.020$) and temporal ($P=0.002$) lobes, as well as in entorhinal ($P=0.006$) and hippocampal ($P=0.011$) areas. The lateral ventricles ($P=0.001$ to 0.040), temporal horns ($P<0.001$), and third ventricle ($P=0.008$) were larger in patients with PHI, reflecting more severe central atrophy. The extent of supratentorial white matter changes was more severe ($P<0.001$) in all WM areas in patients with PHI.

The prestroke ADL assessed with the ADL, IADL, Blessed Functional Activity Scale, and Barthel index scales did not differ between the 2 patient groups (Table 2). However, the 3-month outcome assessed with all 8 outcome scales and the 15-month outcome assessed with the Rankin scale and Barthel Index were worse in patients with PHI ($P=0.004$). There was no difference in mortality rates between patients with and without PHI ($P=0.661$).

To determine whether PHI has an independent impact on a poor clinical outcome, we used a multivariate logistic regression analysis (Table 3). The patients were divided into 2 groups according to disability as measured with the Rankin scale (Rankin 1 to 2 and 3 to 5), and the variables in the model were age, mean cerebral atrophy, mean WMHI score, total volume of all and related brain infarcts, and PHI. The independent predictors for a poor 15-month outcome (Rankin 3 to 5) were the total volume of brain infarcts (OR 1.22, 95% CI 1.139 to 1.315), mean cerebral atrophy (OR 3.59, 95% CI 2.252 to 5.729), and PHI (OR 3.76, 95% CI 2.004 to 7.074).

After the first analysis, we included patients with pontine infarcts in the PHI-positive group ($n=77$); this did not change the results. The 3-month outcome assessed with all 8 outcome scales and the 15-month outcome assessed with the Rankin scale and the Barthel Index were worse in patients with PHI.

### Discussion

Conventional MRI offers a high sensitivity but, unfortunately, quite a low specificity for brain stem signal changes. PHI may represent osmotic myelinolysis, multiple sclerosis, encephalitis, or neoplasm or be sequelae of radiation or chemotherapy. In poststroke patients, the most probable causes of PHI, however, are pontine ischemic rarefaction and WD. The concept of “pontine ischemic rarefaction” was introduced in 1995 by Pullicino et al. in a series of 85 elderly patients. Of these patients, 16 had PHI, and 2 patients were examined histopathologically. The pathological changes in the pons were consistent with a subcortical arteriosclerotic encephalopathy–like pathology, characterized with myelin

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**TABLE 2. Outcome Among 319 Patients Without and With PHI**

<table>
<thead>
<tr>
<th>Outcome Variable</th>
<th>Before Index Stroke</th>
<th>3 mo After Index Stroke</th>
<th>15 mo After Index Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>NIH</td>
<td>PHI –</td>
<td>PHI +</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.5 (2.3)</td>
<td>4.7 (6.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>55.9 (5.0)</td>
<td>49.9 (11.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>1.0 (0.7)</td>
<td>2.1 (1.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>6.4 (2.2)</td>
<td>4.8 (3.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Index of ADL (1–7)</td>
<td>7.2 (1.4)</td>
<td>7.0 (1.5)</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>2.0 (4.4)</td>
<td>3.4 (6.0)</td>
<td>0.020</td>
</tr>
<tr>
<td></td>
<td>0.5 (1.1)</td>
<td>0.7 (1.3)</td>
<td>0.190</td>
</tr>
<tr>
<td>BFAS (0–17)</td>
<td>78.4 (5.8)</td>
<td>78.4 (5.7)</td>
<td>0.995</td>
</tr>
<tr>
<td></td>
<td>75.0 (11.9)</td>
<td>64.6 (22.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Barthe Index (0–100)</td>
<td>1.9 (0.9)</td>
<td>2.5 (1.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Rankin scale</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 3. Correlates of Independent (Rankin 1–2) and Dependent (Rankin 3–5) Living Among 319 Patients 3 and 15 Months After Supratentorial Ischemic Stroke**

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SEM</th>
<th>P</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 mo after stroke</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume of related infarcts</td>
<td>0.229</td>
<td>0.038</td>
<td>&lt;0.001</td>
<td>1.257</td>
<td>1.168–1.353</td>
</tr>
<tr>
<td>Mean brain atrophy</td>
<td>0.986</td>
<td>0.246</td>
<td>&lt;0.001</td>
<td>2.681</td>
<td>1.656–4.340</td>
</tr>
<tr>
<td>PHI</td>
<td>1.126</td>
<td>0.318</td>
<td>&lt;0.001</td>
<td>3.084</td>
<td>1.653–5.755</td>
</tr>
<tr>
<td>Age</td>
<td>0.60</td>
<td>0.023</td>
<td>0.008</td>
<td>1.062</td>
<td>1.016–1.110</td>
</tr>
<tr>
<td>15 mo after stroke</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume of all infarcts</td>
<td>0.202</td>
<td>0.037</td>
<td>&lt;0.001</td>
<td>1.223</td>
<td>1.139–1.315</td>
</tr>
<tr>
<td>Mean cerebral atrophy</td>
<td>1.279</td>
<td>0.238</td>
<td>&lt;0.001</td>
<td>3.592</td>
<td>2.252–5.729</td>
</tr>
<tr>
<td>PHI</td>
<td>1.326</td>
<td>0.322</td>
<td>&lt;0.001</td>
<td>3.765</td>
<td>2.004–7.074</td>
</tr>
</tbody>
</table>

Multiple logistic regression analysis: age, mean cerebral atrophy, mean WMHI score, total volume of related and all brain infarcts, and PHI on MRI are included in the model.
pallor and reactive astrogliosis compatible with ischemic damage. A small portion of the PHI represented WD of the crossing fibers.

The term “wallerian degeneration” refers to a process of anterograde degeneration of an axon and its myelin sheath after connection with the cell body has been disrupted. In the central nervous system, WD is most often detected in the corticospinal tract after brain infarction, but it can also occur in association with WM diseases, such as multiple sclerosis or progressive multifocal leukoencephalopathy, central nervous system neoplasm, surgery, arteriovenous malformation, and hemorrhage, or it can involve other WM tracts.

The chronological changes of WD on MRI have been well documented. Approximately 4 weeks after ictus, decreased signal intensity on T2- or PD-weighted images can be observed in the ipsilateral brain stem. This hypointensity approaches an isointense stage during 70 to 80 days on all pulse sequences. After 81 days, WD can be detected as hyperintensity on T2-weighted images, with or without decreased signal intensity on T1-weighted images. This bright signal intensity on T2-weighted images is said to persist and can be seen as late as 29 years after brain infarction.

Although the diagnostic problem of PHI has been recognized, the studies reporting the frequency and radiological and clinical correlates of PHI are still quite few. In a Japanese study, bilateral PHI was found in 29 of 211 (12.9%) poststroke patients. Kwa et al. reported a frequency of 23% in a series of 229 patients with symptomatic atherosclerosis, and in the series of Pullicino et al., the frequency of PHI was 19%. A recent series of 68 patients with cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy reported the corresponding frequency to be 45%.

In our poststroke cohort, the frequency of unilateral PHI was 14.7%, the frequency of bilateral PHI was 32.9%, and the frequency of any PHI was 47.6%. The high frequency of PHI in our poststroke population suggests that a portion of the changes in the pons represented WD. This impression was also supported by the greater frequency of pyramidal tract lesions in patients with PHI. In this elderly poststroke cohort, even the presence of bilateral PHI could not rule out the possibility of WD, because 63.8% of the patients with bilateral PHI also had bilateral brain infarcts of varying ages. However, a clear, slice-to-slice continuous pyramidal tract hyperintensity, suggesting classic pyramidal tract WD, could be detected in only 37.5% of the patients with PHI.

The mean age and sex and risk factors for stroke did not differ in patients without or with PHI. Instead, PHI was clearly associated with a larger supratentorial infarct load, more advanced WM changes, and more severe parietal and temporal cortical atrophy and supratentorial central atrophy. Similar results in regard to risk factors and WM changes have been reported in previous studies.

Ever since the recognition of PHI, a continuous effort has been made to find clinical correlates. In atherosclerotic patients, PHI has been related to problems with equilibrium. However, it has been admitted that the clinical significance of PHI is difficult to estimate, because PHI usually relates to advanced supratentorial ischemic damage and seldom occurs as an isolated phenomenon.

High signal intensity along the pyramidal tract after supratentorial hemorrhage or ischemic stroke has been connected with poor prognosis in some studies but not in all. Most of the outcome studies have contained only patients presenting with a classic WD of the corticospinal tract, a limited number of subjects, or a short follow-up after stroke. Our study of 319 poststroke patients is thus far the largest well-defined consecutive series that examined the MRI and clinical correlates of PHI as such and its prognostic value on clinical outcome.

In our study, the total volume of brain infarcts (OR 1.22), mean cerebral atrophy (OR 3.59), and PHI (OR 3.76) were independent correlates of poor clinical outcome measured with the Rankin scale. PHI was also associated with poor clinical outcome regardless of corresponding hypointensity on T1-weighted images. For a patient whose medical treatment has reached its limits, the quality of life probably can be improved only by reducing handicap. This can be done best by making realistic plans for the future of the patient with families and caregivers, who constitute an integral part of the poststroke rehabilitation and adaptation process.

In this elderly poststroke cohort, PHI was independently associated with poor clinical outcome. Although nonspecific, poststroke PHI deserves attention, because it clearly prognosticates persistent clinical disability.

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