Cerebrovascular disease, with its complex anatomy, and ischemia in different parts of the brain can produce the same neurological deficits. Because important decisions, such as revascularization, are often based on the association between symptoms and stenosis in a specific vascular territory, the clinician must be confident in the localization. Traditionally, the Oxfordshire Community Stroke Project classification used a simple clinical scheme with a high correspondence to radiological findings to distinguish posterior circulation infarction (PCI) from anterior circulation infarction (ACI). However, this approach may not accurately localize all ischemic strokes. A substantial proportion of PCIs may not be accurately classified only by symptoms/signs, because they lack typical clinical features. Misdiagnosis usually occurs in the initial phase of patient evaluation, which may lead to erroneous clinical decision-making.

In some studies, PCI simulated involvement of the anterior circulation. Recent evidence from magnetic resonance imaging (MRI)-based studies demonstrated that unilateral limb weakness, unilateral limb numbness, nausea/vomiting, and headache are the major clinical neurological deficits in PCI rather than crossed paralysis, crossed sensory deficits, visual field disturbance, isolated vertigo, and dysphagia. This suggests that the clinical manifestations of PCI and ACI are more alike than dissimilar. A comparison of the symptoms and signs between the 2 main stroke localizations within a large series of acute stroke patients has not been performed. We compared the distribution of clinical manifestations between PCI and ACI with lesions confirmed by MRI. In addition, we tried to determine the diagnostic value of specific symptoms/signs for PCI.
Methods

Subjects
From July 2006 to May 2011, we screened 3405 patients with acute ischemic stroke admitted to the Department of Neurology, West China Hospital, located in Chengdu, China; this is a large tertiary hospital and a principal teaching affiliate of West China Medical School of Sichuan University. Consecutive patients were registered prospectively into the Chengdu Stroke Registry database, and data was collected using methods previously described.12 We excluded patients who were admitted to our hospital >2 weeks after onset (881 patients); patients without a MRI-verified ischemic lesion (337 patients); and patients with multiple infarcts involving both anterior circulation and posterior circulation, including watershed infarction (957 patients). Patients with premorbid conditions, such as previous stroke, cancer, uremia, and degenerative diseases, which could confound the neurological examination (56 patients), were also excluded. We relied on Damasio’s template mapping,13 which charts the major divisions of anterior infarction, and on Bogousslavsky’s standards14 to assist in classification of PCI and ACI as detailed in a previous study.15 After these exclusions, we analyzed 1174 patients and classified them into 2 groups based on infarctions involving only posterior circulation or only anterior circulation. The study was approved by the scientific research department of the hospital that conformed to the local ethical criteria during the study period.

Data Collection
Baseline information collected on admission included age, sex, admission delay, initial stroke severity (assessed using the National Institute of Health Stroke Scale and the Glasgow Coma Scale), and risk factors (atrial fibrillation, hypertension, diabetes mellitus, hyperlipidemia, current smoking, alcohol assumption, previous stroke, coronary heart disease, valvular heart disease, and myocardial infarction).

Clinical features were systematically evaluated and defined based on the American Stroke Association’s published stroke warning signs and the Labiche et al study classification, with some modifications.16,17 A neurologist unaware of the imaging results performed a chart review and completed a standardized form to obtain information regarding the symptoms/signs of acute stroke and the Labiche et al study classification, with some modifications.16,17

Statistical Methods
Differences in demographics, risk factors, and the frequency of categorical variables between the PCI and ACI were evaluated by using t tests for continuous variables and χ² tests for discrete variables. To assess the diagnostic value of symptoms/signs, sensitivity, and specificity, the positive predictive value (PPV) and the OR with 95% CI were calculated. Statistical analyses were carried out using the SPSS package (version 16.0 for Windows, SPSS Inc).

Results
Baseline Variables
The final adjudicated diagnoses by MRI of the 1174 patients were 872 ACI (74.3%) and 302 PCI (25.7%). Compared with ACI, patients with PCI were more often men (67.2% versus 58.0%; P=0.005), had a lower mean baseline National Institutes of Health Stroke Scale score (4.8±6.1 versus 6.5±5.9; P<0.001), and a higher mean Glasgow Coma Scale (14.3±1.9 versus 13.6±2.2; P<0.001). PCI patients had a greater frequency of diabetes (20.5% versus 13.8%; P=0.005) and smoking (39.7% versus 31.5%; P=0.009), but a lower frequency of atrial fibrillation (3.0% versus 8.7%; P=0.001) and valvular heart disease (2.0% versus 6.4%; P=0.003; Table 1).

Clinical Manifestations
The frequency of each neurological deficit in both groups is presented in Table 2. Among PCI patients, disturbed consciousness at the time of hospital admission was less common (10.3% versus 18.6%; P=0.001), because at the neurological evaluation, they were less often in a somnolent or stuporous state compared with ACI patients. Concerning speech disturbances, aphasia (1.0% versus 22.0%; P=0.001) occurred in a lower percentage of PCI patients, and no statistically significant difference was found in the percent of patients having dysarthria (25.5% versus 25.0%; P=0.864) in both groups.

Neurological deficits that are significantly more common in PCI patients compared with ACI are shown in Figure 1. Nausea/vomiting (33.8% versus 10.4%; P<0.001) was the most frequent sign followed by ataxia (31.5% versus 5.4%; P<0.001), whereas other symptoms/signs with a low prevalence of PCI ranged from 1.3% to 18.9%.

The patients’ neurological deficits in PCI and ACI were divided into 3 subgroups, using their frequency in PCI as the reference group: very common (≥30%), moderately common
Table 2. Main Neurological Deficits by Infarction Localization

<table>
<thead>
<tr>
<th></th>
<th>Posterior Circulation Infarct, No. (%)</th>
<th>Anterior Circulation Infarct, No. (%)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>302</td>
<td>872</td>
<td></td>
</tr>
<tr>
<td>Disturbed consciousness</td>
<td>31 (10.3)</td>
<td>162 (18.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>Confusion/delirium</td>
<td>3 (1.0)</td>
<td>25 (2.9)</td>
<td>0.066*</td>
</tr>
<tr>
<td>Somnolence</td>
<td>20 (6.6)</td>
<td>100 (11.5)</td>
<td>0.017</td>
</tr>
<tr>
<td>Stupor</td>
<td>2 (0.7)</td>
<td>29 (3.3)</td>
<td>0.013</td>
</tr>
<tr>
<td>Coma</td>
<td>6 (2.0)</td>
<td>8 (0.9)</td>
<td>0.213*</td>
</tr>
<tr>
<td>Speech disturbance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>3 (1.0)</td>
<td>192 (22.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>77 (25.5)</td>
<td>218 (25.0)</td>
<td>0.864</td>
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<tr>
<td>Central facial/lingual palsy</td>
<td>123 (40.7)</td>
<td>542 (62.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Motor deficits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mono limb</td>
<td>17 (5.6)</td>
<td>79 (9.1)</td>
<td>0.061</td>
</tr>
<tr>
<td>Homolateral</td>
<td>162 (53.6)</td>
<td>653 (74.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Bilateral limbs</td>
<td>31 (10.3)</td>
<td>48 (5.5)</td>
<td>0.004</td>
</tr>
<tr>
<td>Crossed sign</td>
<td>12 (4.0)</td>
<td>1 (0.1)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Quadriplegia</td>
<td>25 (8.3)</td>
<td>43 (4.9)</td>
<td>0.032</td>
</tr>
<tr>
<td>Sensory deficits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mono limb</td>
<td>15 (5.0)</td>
<td>53 (6.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Homolateral</td>
<td>110 (36.4)</td>
<td>298 (34.2)</td>
<td>0.479</td>
</tr>
<tr>
<td>Bilateral limbs</td>
<td>6 (2.0)</td>
<td>9 (1.0)</td>
<td>0.203</td>
</tr>
<tr>
<td>Crossed sign</td>
<td>9 (3.0)</td>
<td>0 (0)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Visual field deficits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single blind</td>
<td>0 (0.0)</td>
<td>2 (0.2)</td>
<td>1.000*</td>
</tr>
<tr>
<td>Hemianopia</td>
<td>13 (4.3)</td>
<td>11 (1.3)</td>
<td>0.001</td>
</tr>
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<td>Quadrantanopia</td>
<td>4 (1.3)</td>
<td>0 (0)</td>
<td>0.005*</td>
</tr>
<tr>
<td>Diplopia</td>
<td>22 (7.3)</td>
<td>6 (0.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Eye movement disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oculomotor nerve palsy</td>
<td>12 (4.0)</td>
<td>0 (0)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Gaze palsy</td>
<td>8 (2.6)</td>
<td>96 (11.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>36 (11.9)</td>
<td>7 (0.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Walking instability</td>
<td>40 (13.2)</td>
<td>22 (2.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ataxia</td>
<td>95 (31.5)</td>
<td>47 (5.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Seizures</td>
<td>1 (0.3)</td>
<td>4 (0.5)</td>
<td>1.000*</td>
</tr>
<tr>
<td>Syncope</td>
<td>5 (1.7)</td>
<td>29 (3.3)</td>
<td>0.136</td>
</tr>
<tr>
<td>Horner’s syndrome</td>
<td>12 (4.0)</td>
<td>0 (0)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Patient complaints</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td>53 (17.5)</td>
<td>83 (9.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>102 (33.8)</td>
<td>91 (10.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Vertigo</td>
<td>57 (18.9)</td>
<td>15 (1.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>8 (2.6)</td>
<td>8 (0.9)</td>
<td>0.051*</td>
</tr>
</tbody>
</table>

* P value obtained from Fisher exact test.

(10%–30%), and less common (≤10%). Homolateral paralysis (PCI, 53.6% versus ACI, 74.9%; P<0.001), central facial/lateral palsy (PCI, 40.7% versus ACI, 62.2%; P<0.001), and hemisensory deficits (PCI, 36.4% versus ACI, 34.2%; P=0.479) were the 3 most common neurological deficits in PCI and ACI; however, a large proportion of specific neurological deficits that occurred significantly more often in PCI patients were in the moderately and less common subgroups with frequency <30% (Figure 2).

Predictive Value of Neurological Deficits in Diagnosing Patients With PCI

Table 3 shows the value of single neurological deficit for a PCI diagnosis. The neurological deficits with the highest predictive values favoring the diagnosis of PCI were in the less common subgroup, including Horner’s syndrome (PPV=100.0%; OR=4.00), crossed sensory deficits (PPV=100.0%; OR=3.98), quadrantanopia (PPV=100.0%; OR=3.93), oculomotor nerve palsy (PPV=100.0%; OR=4.00), and crossed motor deficits (PPV=92.3%; OR=36.04). However, all of them occurred with an extremely low sensitivity ranging from 1.3% to 4.0% of PCI patients, which means that the probability of detecting neurological deficits in patients with PCI was very low. Neurological deficits with a relatively good predictive value such as ataxia (PPV=66.9%; OR=8.06), nausea/vomiting (PPV=52.8%; OR=4.38), nystagmus (PPV=83.7%; OR=16.72), vertigo (PPV=79.2%; OR=13.29), and diplopia (PPV=78.6%; OR=11.34), also displayed low sensitivities that decreased their diagnostic value.

Discussion

Given the limited information deriving from small case series with highly selected PCI patients, as well as the low performance rate of MRI in these studies, there have not been reliable clinical criteria for PCI. In the current study, we initially compared the frequency of neurological deficits between the 2 groups and found a similar distribution among the most common symptoms and signs between PCI and ACI. Second, we assessed the ability of a single neurological deficit to discriminate PCI from ACI, and we observed that despite some symptoms and signs having approximately 100% specificity for diagnosing PCI, symptoms/signs with a higher predictive value had a very low prevalence. These findings emphasize the fact that the clinical manifestations between PCI and ACI have a high degree of similarity.
In this study, we observed that the most common neurological deficits were homolateral paralysis, central facial/lingual palsy, and hemisensory deficits in PCI; and also, that these were the 3 most common in ACI. Previous studies, the New England Medical Center Posterior Circulation Registry, and a Qatar study presented within-study comparisons of the frequency of the neurological deficits in PCI cohorts; but, these studies did not compare PCI with ACI patients. The frequency appeared to vary in some clinical manifestations in these previous studies, as compared with that in our study; however, homolateral paralysis and facial palsy were also found previously to be the most common signs in PCI patients. The divergent frequencies may partly caused by differences in the severity of vertebrobasilar ischemia among stroke populations and the different definitions used when classifying the neurological deficits. In a series of articles from the New England Medical Center Posterior Circulation Registry, Caplan et al reported that unilateral limb weakness (38%) was the most frequent sign in the whole PCI cohort, hemiparesis and tetraparesis (62%) were the most common signs for patients with midbrain infarction, and hemiparesis was present in more than half of patients (50.6%), along with basilar artery occlusive disease. Furthermore, in a study that analyzed the clinical manifestations of pontine infarction, pure motor hemiparesis or hemiplegia was found in a large proportion of patients instead of classical crossed syndromes, and cannot be distinguished from the internal capsule-corona radiata region infarctions by clinical signs alone. These findings are consistent with our view that PCI patients with homolateral paralysis are common in clinical practice.

Besides the nonspecificity of most clinical manifestations in PCI versus ACI, we should also emphasize that there are still certain symptoms/signs favoring a diagnosis of PCI, such as crossed motor/sensory deficits, oculomotor nerve palsy, visual field deficits, and Horner’s syndrome, which are highly suggestive. Another point to consider is that these signs occurred infrequently (<10%); hence, it is difficult to identify most PCI patients based on these relatively rare clinical manifestations. One previous study with the observation that approximately 10% to 20% of patients with a diagnosis of presumed ACI actually have a PCI supports our contention. The misdiagnosis rate in the previous study might be underestimated because of the small number of patients included and the low sensitivity of computed tomography in detecting multiple infarctions involving the posterior fossa.

According to the literature, crossed signs such as ipsilateral motor and sensory cranial nerve signs or symptoms plus contralateral hemiplegia or hemianesthesia are specific symptoms that point to brain stem involvement. However, in the present cohorts, crossed motor deficits occurred infrequently (13 patients; 1.1%), with only 1 patient diagnosed with ACI having MRI-confirmed bilateral middle cerebral artery infarction that happened suddenly after cholecystectomy. Crossed sensory deficits occurred in only 9 PCI patients (3.0%) and in 0 patients with ACI. The crossed sensory deficits correlated well with lesion locations involving the medulla, whereas crossed motor deficits were associated with lesions in medulla (n=8), pons (n=2), and midbrain (n=2). A study focusing on pure lateral medullary infarction reported that the

Figure 2. The frequency of single clinical symptom/sign in posterior circulation infarction and anterior circulation infarction.
rate of the crossed sensory deficits was high at approximately 26%, but the correlation between infarct topography and the crossed motor/sensory signs in PCI patients were not well-described and need additional investigation. Contrary to current belief, our results do not support the conventional wisdom that holds that disturbed consciousness is highly specific for PCI. Since the initial description of the clinical findings in a small group of select patients with brain stem infarction by Kubik and Adams, disturbances of consciousness were considered to be an important feature of PCI. This was followed by studies of patients with specific vertebrobasilar lesions, including the midbrain, cerebellum, and basilar artery occlusive disease separately; the rate of disturbed consciousness was high, frequently >20%. In the most extensive of these studies, Carol et al reported 20 patients with basilar artery occlusion and found 19 of them had various impairment of consciousness. In contrast, Sato et al reported a much lower rate of disturbed consciousness in patients with PCI than with ACI (18% versus 41%; \( P<0.001 \)); this was in accordance with our cohorts that disturbed consciousness accompanied with a corresponding lower Glasgow Coma Scale score occurred in 10.3% of PCI, which was less prevalent than that in ACI (18.6%). Similarly, 2 recent studies showed that the prevalence of altered level of consciousness in PCI patients was much lower (10% and 18%, respectively) than was suggested by previous studies. These controversies might be caused by a different distribution of stroke subtypes or different age groups. At the least, the present study revealed that additional investigations are required to determine whether consciousness disturbance should be considered diagnostic for PCI.

In the present study, nausea/vomiting, vertigo, and headaches were found to be the symptoms that occur more frequently in patients with PCI. Because circulation to the inner ear arises from the vertebrobasilar system, vertebroartery disease can cause vertigo that is usually accompanied

<table>
<thead>
<tr>
<th>Symptoms/Signs</th>
<th>No. of Patients</th>
<th>No. With PCI</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Positive Predictive Value (%)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>1174</td>
<td>302</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>The very common subgroup</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Homolateral paralysis</td>
<td>815 (69.4)</td>
<td>162</td>
<td>53.6</td>
<td>25.1</td>
<td>19.9</td>
<td>0.39 (0.29–0.51)</td>
</tr>
<tr>
<td>Central facial/tongue paralysis</td>
<td>665 (56.5)</td>
<td>123</td>
<td>40.7</td>
<td>37.8</td>
<td>18.5</td>
<td>0.42 (0.32–0.55)</td>
</tr>
<tr>
<td>Hemisensory deficits</td>
<td>408 (34.8)</td>
<td>110</td>
<td>36.4</td>
<td>65.8</td>
<td>27.0</td>
<td>1.10 (0.84–1.45)</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>193 (16.4)</td>
<td>102</td>
<td>33.8</td>
<td>89.6</td>
<td>52.8</td>
<td>4.38 (3.17–6.04)</td>
</tr>
<tr>
<td>Ataxia</td>
<td>142 (12.1)</td>
<td>95</td>
<td>31.5</td>
<td>94.6</td>
<td>66.9</td>
<td>8.06 (5.50–11.80)</td>
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<tr>
<td>The moderately common subgroup</td>
<td></td>
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<td></td>
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<td></td>
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<tr>
<td>Dysarthria</td>
<td>295 (25.1)</td>
<td>77</td>
<td>25.5</td>
<td>75.0</td>
<td>26.1</td>
<td>1.03 (0.76–1.39)</td>
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<tr>
<td>Vertigo</td>
<td>72 (6.1)</td>
<td>57</td>
<td>18.9</td>
<td>98.3</td>
<td>79.2</td>
<td>13.29 (7.40–23.89)</td>
</tr>
<tr>
<td>Headache</td>
<td>136 (11.6)</td>
<td>53</td>
<td>17.5</td>
<td>90.5</td>
<td>39.0</td>
<td>2.02 (1.39–2.94)</td>
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<tr>
<td>Walking instability</td>
<td>62 (5.3)</td>
<td>40</td>
<td>13.2</td>
<td>97.5</td>
<td>64.5</td>
<td>5.90 (3.44–10.12)</td>
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<td>Nystagmus</td>
<td>43 (3.7)</td>
<td>36</td>
<td>11.9</td>
<td>99.2</td>
<td>83.7</td>
<td>16.72 (7.36–38.02)</td>
</tr>
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<td>Disturbed consciousness</td>
<td>193 (16.4)</td>
<td>31</td>
<td>10.3</td>
<td>81.4</td>
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<td>0.50 (0.33–0.76)</td>
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<td>Bilateral motor weakness</td>
<td>79 (6.7)</td>
<td>31</td>
<td>10.3</td>
<td>94.5</td>
<td>39.2</td>
<td>1.96 (1.23–3.15)</td>
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<tr>
<td>The less common subgroup</td>
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<tr>
<td>Quadriplegia</td>
<td>68 (5.8)</td>
<td>25</td>
<td>8.3</td>
<td>95.1</td>
<td>36.8</td>
<td>1.74 (1.04–2.90)</td>
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<tr>
<td>Diplopia</td>
<td>28 (2.4)</td>
<td>22</td>
<td>7.3</td>
<td>99.3</td>
<td>78.6</td>
<td>11.34 (4.55–28.25)</td>
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<tr>
<td>Mono limb weakness</td>
<td>95 (8.1)</td>
<td>17</td>
<td>5.6</td>
<td>91.1</td>
<td>17.9</td>
<td>0.61 (0.35–1.04)</td>
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<td>Hemianopia</td>
<td>24 (2.0)</td>
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<td>4.3</td>
<td>98.7</td>
<td>54.2</td>
<td>3.52 (1.56–7.95)</td>
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<td>Horner’s syndrome</td>
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<td>12</td>
<td>4.0</td>
<td>100.0</td>
<td>100.0</td>
<td>4.00 (3.63–4.43)</td>
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<td>12</td>
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<td>100.0</td>
<td>100.0</td>
<td>4.00 (3.63–4.43)</td>
</tr>
<tr>
<td>Crossed motor deficits</td>
<td>13 (1.1)</td>
<td>12</td>
<td>4.0</td>
<td>99.9</td>
<td>92.3</td>
<td>36.04 (4.67–278.38)</td>
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<td>Crossed sensory deficits</td>
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<td>9</td>
<td>3.0</td>
<td>100.0</td>
<td>100.0</td>
<td>3.98 (3.60–4.39)</td>
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<td>Tinnitus</td>
<td>16 (1.4)</td>
<td>8</td>
<td>2.6</td>
<td>99.1</td>
<td>50.0</td>
<td>2.94 (1.10–7.90)</td>
</tr>
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<td>Gaze palsy</td>
<td>104 (8.9)</td>
<td>8</td>
<td>2.6</td>
<td>89.0</td>
<td>7.7</td>
<td>0.22 (0.11–0.46)</td>
</tr>
<tr>
<td>Bilateral sensory deficits</td>
<td>15 (1.3)</td>
<td>6</td>
<td>2.0</td>
<td>99.0</td>
<td>40.0</td>
<td>1.94 (0.69–5.51)</td>
</tr>
<tr>
<td>Syncope</td>
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<td>5</td>
<td>1.7</td>
<td>96.7</td>
<td>14.7</td>
<td>0.49 (0.19–1.28)</td>
</tr>
<tr>
<td>Quadrantanopia</td>
<td>4 (0.3)</td>
<td>4</td>
<td>1.3</td>
<td>100.0</td>
<td>100.0</td>
<td>3.93 (3.56–4.33)</td>
</tr>
<tr>
<td>Aphasia</td>
<td>195 (16.6)</td>
<td>3</td>
<td>1.0</td>
<td>78.1</td>
<td>1.5</td>
<td>0.04 (0.01–0.11)</td>
</tr>
<tr>
<td>Seizures</td>
<td>5 (0.4)</td>
<td>1</td>
<td>0.3</td>
<td>99.5</td>
<td>20.0</td>
<td>0.72 (0.08–6.48)</td>
</tr>
</tbody>
</table>

PCI indicates posterior circulation infarction.
by other brain stem or cerebellar symptoms. A previous study emphasized that isolated vertigo is rarely attributable to vascular events and it was in accordance with our findings that only 4 patients with PCI had isolated vertigo.

We are aware of the limitations of the present study. First, to elaborate the clinical features of pure PCI and ACI, we excluded patients with multiple lesions involving both posterior circulation and anterior circulation without MRI confirmation; this may have resulted in a selection bias. Second, the delineation of clinical symptoms and signs was completely dependent on chart review, and thus, the authors were unable to confirm rare manifestations. Nonetheless, most of the medical charts within the present study contained high-quality data. Third, as a hospital-based study, subjects who experienced more severe or minor stroke were not admitted to the hospital, although this hospital is the largest one in the region. Given the large sample and statistical significance of the observed effects in the present study, these limitations are unlikely to affect the results.

Conclusion

In this observational study, we found that there was no apparent difference in the frequency of the most common symptoms/signs between PCI and ACI. Some neurological deficits were highly specific for diagnosing PCI, but their sensitivity suggests that symptoms or signs considered typical of PCI are uncommon. Inaccurate localization would be common if clinicians only relied on clinical symptoms/signs to differentiate PCI from ACI, and this observation suggests that neuroimaging findings, particularly MRI, are vital for accurate localization.

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Disclosures

None.

References

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