Cerebral Infarction Secondary to Temporal Lobe Herniation in Head Trauma: A CT Study*

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— Abstract —

Cerebral infarction is a known complication of temporal lobe herniation caused by a traumatic intracranial lesion. To ascertain the frequency, time of recognition, and influence on mortality of posttraumatic cerebral infarction secondary to temporal lobe herniation, we retrospectively reviewed brain CT scans and clinical records of 55 patients who had CT and clinical signs of temporal lobe herniation on admission date. Cerebral infarctions were recognized in seven (12.7%) patients on CT scans taken within two days after admission (mean: 1.3 days). Cerebral infarctions were in the territories of the posterior cerebral artery in all seven patients, two of whom had infarctions of the anterior choroidal artery as well. Mortality (71.4%) for these seven patients was not statistically significant from that (50%) of patients without cerebral infarction admitted with the same range of Glasgow Coma Scale score.

The result suggests that such cerebral infarction does not greatly influence patient’s mortality.

Index Words: Brain, computed tomography, 13.1211
Brain, trauma, 13.43
Brain, infarction, 13.4352
Brain, herniation, 13.4386

INTRODUCTION

Supratentorial unilateral lesions in head trauma cause temporal lobe herniation (TLH) of the brain through the tentorial notch. Temporal lobe herniation may be accompanied by vascular compression leading to cerebral infarctions (1-4). Since first reported by Meyer in 1920 (5), occipital lobe infarctions following compression of the posterior cerebral artery (PCA) between the herniating medial temporal lobe and the tentorial margin during the period of TLH have been described in several pathologic and radiologic literatures (1-4). Thus, common sites and probable mechanisms of cerebral infarctions complicating TLH in head trauma are rather well known (1-4). However, only a few reports have described the frequency of cerebral infarctions complicating TLH by CT analysis (2,3).
Furthermore, time of CT recognition of these infarctions and their influence on mortality are not well established.

To evaluate the frequency, time of recognition and influence on patient mortality of these infarctions, we retrospectively reviewed brain CT scans and clinical records of traumatic patients who had CT signs of TLH.

**MATERIALS AND METHODS**

Over a three-year period, 55 patients showed had CT signs of TLH among 511 patients who had undergone brain CT due to head trauma. CT signs of TLH were based on the presence of obliteration of the suprasellar cistern, widening of the ipsilateral cerebellopontine angle cistern, dilatation of the contralateral temporal horn, and compression of the midbrain (6,7). These 55 patients underwent at least two CT examinations, first of which was performed within two hours after admission or already performed at an outside institution before transfer to our hospital. 55 patients were operated for evacuation of intracranial hematoma one to three hours after initial CT examination and underwent the first follow-up CT examination within 24 hours after operation. All CT scans were obtained with 10mm slice thickness and interval. All patients with CT signs of TLH exhibited clinical signs of TLH before operation, ranging from subtle anisocoria to dilated nonreactive pupil (s) or deacrebrated posture. The patients consisted of 41 males and 14 females with an age range of 6 to 70 years (mean: 42 years).

We reviewed initial and serial follow-up CT scans of 55 patients in view of the type and location of primary intracranial lesions, frequency and location of cerebral infarctions, and time to CT recognition of cerebral infarctions from the admission date. Cerebral infarctions were diagnosed when a well marginated area of low density in a known arterial territory was noted on initial or follow-up CT scans. Low densities indicating traumatic contusions or infarctions due to surgical complication or systemic hypoxia were excluded. Also low densities identified in the brain stem were excluded because brain stem infarctions secondary to TLH can not be easily distinguished by CT from other lesions such as primary brain stem injuries. To ascertain the neurologic status on admission as reflected by the Glasgow Coma Scale (GCS) score and patient outcome, medical records of 55 patients were reviewed. The mortality of patients with cerebral infarctions secondary to TLH was compared with that of patients without such infarctions admitted with the same range of GCS score.

**RESULTS**

Primary traumatic intracranial lesions of 55 patients with CT signs of TLH included subdural hematomas (31 cases), epidural hematomas (18 cases), intracerebral hematomas (four cases) and localized cerebral contusions (two cases). All lesions were located in unilateral supratentorial hemispheric regions. Cerebral infarctions were detected in seven (12.7%) of 55 patients, whose traumatic lesions comprised epidural hematomas (three cases), subdural hematomas (three cases) and intracerebral hematoma (one case) (Table 1). Cerebral infarctions appeared on the first follow-up CT scans within one day after admission in four patients and within two days in two patients (mean: 1.3 days). Cerebral infarctions developed in the PCA territories in all seven patients, two of whom had infarctions of the anterior choroidal artery (AChA) territories as well. PCA infarctions were distributed in the ipsilateral occipital lobe in five patients, the ipsilateral occipital and medial temporal lobes in one (Fig. 1), and the ipsilateral hippocampal area in another patient (Fig. 2). Infarctions of AChA territories of two patients were confined to the posterior limb of the internal capsule and the medial portion of the globus pallidus, one ipsilaterally and the other bilaterally (Fig. 3).
Seven patients with cerebral infarctions were admitted with the GCS score of seven or less. Among 48 patients without cerebral infarctions, 20 patients had the GCS score of seven or less. The mortalities of these two groups with the same range of the GCS score (equal to or less than seven) were 71.4% (five of seven cases) and 50% (10 of 20 cases) respectively. By Fisher's Exact test (8), however, there was no statistically significant difference in mortalities between two groups.

**DISCUSSION**

Recently magnetic resonance (MR) imaging has been shown to be more sensitive than CT in depicting many types of traumatic intracranial lesions. In severe head trauma, however, patients generally are not candidates for MR imaging and CT acts a major role in determining its diagnosis and prognosis. CT recognition of TLH secondary to traumatic intracranial lesions facilitates prompt clinical evaluation and subsequent management. Two important possible complications of TLH are brain stem injury and cerebral infarction, of which brain stem injury is clinically more important (9). Although hemorrhagic brain stem lesions are rather easily detected with CT, ischemic brain stem lesions are generally missed on CT due to poor contrast resolution and the presence of posterior fossa artifacts (10,11). Furthermore, brain stem injuries secondary to TLH can not be easily distinguished by CT from primary brain stem injuries (9). For these

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**Fig. 1. Case 2. Cerebral infarction in the territory of the posterior cerebral artery.**

a: CT scan on admission shows subdural hematoma in the left frontotemporal area with marked midline shift. b: More caudal CT scan shows CT signs of severe temporal lobe herniation: obliteration of the suprasellar and perimesencephalic cisterns, distortion of the midbrain, and widening of the right temporal horn (arrow). c,d: CT scan performed immediately after operation shows a well defined low density in the left occipital lobe extending to the left medial temporal lobe, representing infarction in the territory of the left posterior cerebral artery.
Table 1. Summary of Patients with Cerebral Infarction Secondary to Temporal Lobe Herniation

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr) /Sex</th>
<th>Cause of Temporal Lobe Herniation</th>
<th>GCS Score at Admission</th>
<th>Time Lapse before Infarction* (day)</th>
<th>Cerebral Infarction Site</th>
<th>Vascular Territory</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>70/M</td>
<td>EDH, Rt</td>
<td>5</td>
<td>1</td>
<td>Rt occipital</td>
<td>PCA</td>
<td>Died</td>
</tr>
<tr>
<td>2</td>
<td>63/M</td>
<td>SDH, Lt</td>
<td>4</td>
<td>1</td>
<td>Lt occipital</td>
<td>PCA</td>
<td>Died</td>
</tr>
<tr>
<td>3</td>
<td>50/M</td>
<td>EDH, Rt</td>
<td>5</td>
<td>1</td>
<td>Rt occipital</td>
<td>PCA</td>
<td>Died</td>
</tr>
<tr>
<td>4</td>
<td>33/M</td>
<td>EDH, Rt</td>
<td>5</td>
<td>1</td>
<td>Rt occipital</td>
<td>PCA</td>
<td>Mild headache</td>
</tr>
<tr>
<td>5</td>
<td>46/M</td>
<td>ICH, Rt</td>
<td>7</td>
<td>2</td>
<td>Bi PLI/GP</td>
<td>AChA</td>
<td>Died</td>
</tr>
<tr>
<td>6</td>
<td>15/M</td>
<td>SDH, Rt</td>
<td>6</td>
<td>2</td>
<td>Rt occipital</td>
<td>PCA</td>
<td>Lt homonymous hemianopsia/ Lt hemiparesis</td>
</tr>
<tr>
<td>7</td>
<td>38/M</td>
<td>SDH, Rt</td>
<td>4</td>
<td>1</td>
<td>Rt occipital</td>
<td>PCA</td>
<td>Died</td>
</tr>
</tbody>
</table>

*: time to CT recognition of infarction from the date of admission

GCS = Glasgow Coma Scale, Rt = right, Lt = left, Bi = bilateral, EDH = epidural hematoma, SDH = subdural hematoma, ICH = intracerebral hematoma, PLI = posterior limb of internal capsule, GP = globus pallidus, PCA = posterior cerebral artery, AChA = anterior choroidal artery.

Reasons, we omitted the evaluation of possible brain stem pathologies in this study in spite of their clinical importance.

Cerebral infarctions secondary to TLH have been reported in several pathologic and radiologic literatures with the mention of probable mechanisms of infarctions. Infarctions of the PCA territory following compression of the PCA...
Fig. 3. Case 5. Cerebral infarctions in territories of the posterior cerebral and anterior choroical arteries.

a,b: Admission CT scan shows large right frontal lobe hematoma producing temporal lobe herniation as in Fig. 1b. A focal contusional hematoma in the left frontal lobe (arrow) is noted also.

c: Postop CT scan on the second day of admission shows a well defined low density in the right occipital lobe. Additional focal low densities (arrows) appear bilaterally in the posterior limb of the internal capsule and the medial portion of the globus pallidus, which belong to territory of the anterior choroidal artery.

Table 2. Mortality in Patients with Temporal Lobe Herniation and GCS Score of 7 or less

<table>
<thead>
<tr>
<th></th>
<th>% Mortality (No. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>with Infarction</td>
<td>without Infarction</td>
</tr>
<tr>
<td>71.4% (5/7)</td>
<td>50% (10/20)</td>
</tr>
</tbody>
</table>

GCS: Glasgow Coma Scale.  
p > 0.05 by Fisher’s Exact test

between the herniating medial temporal lobe and the tentorial margin during the period of TLH is the most well recognized one (1-5). Seven patients in our study developed infarctions of the PCA territory with preceding CT signs of severe TLH. The variability in the extent of cerebral infarctions, as in our cases, is thought to be caused by the anatomical variation in the relationship of the PCA to the tentorium: a narrow aperture between the free margins of tentorium is apt to compress the PCA in the more proximal portion, resulting in a wide area of infarction (1,3). The area of the hippocampus is presumably involved by the compression of the hippocampal branches arising from the PCA (3). Some anatomical arrangement favors involvement of the contralateral PCA, which is caught against the tentorial margin by the displaced mibdrain (1-3); however, we have not observed such findings in our cases.

The less well recognized cerebral infarctions of the AChA territory as a complication of TLH have been described in few instances, all of which were located in the ipsilateral AChA territory (2,3). The AChA may be compressed between the herniating medial temporal lobe laterally and the cerebral peduncle medially (1-3). Of our seven patients with cerebral infarctions of the PCA territory, two developed infarctions of the AChA territory as well, one ipsilaterally and the other bilaterally. These patients had preceding CT signs of severe TLH. We think that the contralateral AChA may be also compressed between the displaced midbrain and the contralateral medial temporal lobe.

The development of cerebral infarctions secondary to TLH may depend on how rapidly herniation occurs and therefore, there might not be a chance for collaterals to supply ischemic regions (12). In fact, we have not found similar infarctions in patients with a slowly developing TLH from the tumor or abscess although recently Wernick et al (13) reported a case of the infarction of the PCA territory secondary to TLH produced by a large meningeal sarcoma.
In regard to the frequency of cerebral infarctions secondary to TLH, Sato et al (3) described the development of infarctions of the PCA territory in nine(9%) of 100 patients with CT and clinical signs of TLH. Another recent report by Mirvis et al(2) described posttraumatic cerebral infarctions developed in 25 (1.9%) of 1332 patients who underwent brain CT due to head trauma; however, the precise frequency of cerebral infarctions secondary to TLH was not developed because their posttraumatic cerebral infarctions might be caused not only by TLH but also by other type of cerebral herniation, increased intracerebral pressure, direct pressure effect from overlying extraaxial hematoma and direct vascular injury. Our study disclosed the development of cerebral infarctions secondary to TLH in seven(12.7%) of 55 patients with CT and clinical signs of TLH, similar to Sato’s result, and in 1.4% of 511 patients who required brain CT for head trauma.

Cerebral infarctions secondary to TLH in our cases were recognized on CT scans taken one or two days after admission(mean:1.3 days). Cerebral infarctions secondary to TLH reported by Sato et al (3) appeared on CT scan 2 to 22 days after admission(mean:9.6 days). Mirvis et al (2) diagnosed posttraumatic cerebral infarctions by CT within 24 hours of admission in 40% of patients and up to 14 days after admission(mean:3 days) although their result could not exactly represent the time of recognition of infarction secondary to TLH. We think that our earlier follow-up of brain CT after surgical operation enabled the earlier recognition of cerebral infarctions than in others.

A statistically significant increase in mortality of patients with cerebral infarctions secondary to TLH could not be demonstrated over that of patients without infarctions when matched with the same range of GCS score. This may be explained by the fact that patients with TLH are already associated with a high mortality due to severe head injury. We think that the development of cerebral infarctions in traumatic patients with TLH is not greatly related to the mortality. However, additional clinical studies need to be performed to evaluate exactly the influence of such infarctions on mortality.

REFERENCES

12. Rothfus WE, Goldberg AL, Tabas JH, Deeb ZL. Callosomarginal infarction secondary to transfalcial herniation. AJNR 1987;8:1073-1076
두부 외상 환자에서 측두엽 허니아에 의한 뇌경색: 전산화단층촬영 소견

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두개내 외상성 병변에 의한 측두엽 허니아의 합병증으로 뇌경색이 생길 수 있다는 것은 잘 알려져 있다. 저자들은 전산화단층촬영 소견을 중심으로 이러한 뇌경색의 발생 빈도, 시기 및 사망율에 미치는 영향을 알아보기 위하여, 입원 당시 전산화단층촬영 소견 및 임상적으로 측두엽 허니아의 증후를 보였던 55예 환자의 전산화단층촬영 사진 및 의무기록을 후향적으로 분석하였다. 뇌경색은 7예 (12.7%) 에서 발생하였고 모두 입원 후 2일 이내 (평균: 1.3일) 전산화단층촬영에서 나타났다. 뇌경색은 7예 모두에서 후뇌동맥 영역에서 나타났으며, 이 중 2에는 전맥락총동맥 영역의 뇌경색을 동반하였다. 뇌경색이 발생한 7예의 사망율 (71.4%) 은 입원 당시 같은 Glasgow Coma Scale score를 가지면서 뇌경색이 발생하지 않은 환자군의 사망율 (50%)에 비해 통계적으로 유의한 차이가 있었다.

이러한 결과로 두개내 외상성 병변에 의한 측두엽 허니아의 합병증으로 뇌경색이 발생하더라도 사망율에는 큰 영향을 미치지 않는 것으로 생각된다.