Original Article

Relationship between Abducens Nerve Palsy and Local Hematoma in a Case Series of Ruptured Vertebral Artery Dissecting Aneurysm

Abstract

Background: Abducens nerve palsy associated with subarachnoid hemorrhage (SAH) has rarely been reported. Its frequency, mechanism of palsy, association with aneurysmal location, and clinical course are poorly described. The purpose of our study was to evaluate patients with abducens nerve palsy caused by SAH occurring from ruptured vertebral artery (VA) dissecting aneurysm and to find aneurysmal location using initial computed tomography (CT) and its association with clinically detected cranial nerve palsy. Methods: Fourteen patients of SAH due to ruptured VA dissecting aneurysm were treated at our hospital from January 2011 to May 2015. The clinical courses and CT findings were reviewed retrospectively. Results: Abducens nerve palsy was observed in 77.8% of cases after excluding patients with decreased levels of consciousness. Clots within the prepontine cistern were significantly thicker in cases of VA dissecting aneurysm than in case of supratentorial aneurysm (P = 0.002). Conclusion: The findings of our study indicated that ruptured VA dissecting aneurysms, even in cases of angio-negative SAH, are likely to present with abducens nerve palsy.

Keywords: Abducens nerve palsy, dissecting aneurysm, hematoma, subarachnoid hemorrhage, vertebral artery

Introduction

Subarachnoid hemorrhage (SAH) is associated with sudden-onset headache, loss of consciousness, convulsion, and hemiparesis. In rare cases, it may cause cranial nerve palsies. The abducens nerve is more likely to be injured than other cranial nerves because, intracranially, it runs a relatively long distance and an anatomically complicated course.[1-4] Abducens nerve palsy associated with SAH has been described according to aneurysmal origin, but the frequency, mechanism, location of aneurysms, and clinical course of this condition have been poorly described. In our study, we focused on patients with SAH caused by ruptured vertebral artery (VA) dissecting aneurysm; some of them had thick hematoma in the prepontine cistern and abducens nerve palsy. Early diagnosis of the hemorrhagic origin is frequently challenging using three-dimensional computed tomography (3DCT), particularly in the cases of ruptured VA dissecting aneurysm. We evaluated the relationship between abducens nerve palsy and local hematoma thickness in patients of SAH due to ruptured VA dissecting aneurysm.

Materials and Methods

We retrospectively reviewed 14 consecutive cases of ruptured VA dissecting aneurysm treated at our hospital between January 2011 and May 2015. Clinical data including age/sex/site of hemorrhage/presence of abducens nerve palsy/duration of abducens nerve palsy, World Federation of Neurosurgery grade, Fisher group, hematoma thickness (on initial CT) in the prepontine cistern, distance of the prepontine cistern after the hematoma was washed out, strategy of treatment, and modified Rankin scale scores were evaluated. Hematoma thickness and the distance of the prepontine cistern after the hematoma was washed out were calculated using axial CT images. The formula used was the ratio of the distance between the posterior clinoid process and the front of pons to the distance between the posterior clinoid process and the posterior cranial fossa [Figure 1]. We named the calculated ratio – “hematoma thickness ratio.” The calculated hematoma thickness ratio was compared and evaluated against age-matched cases of ruptured supratentorial aneurysms, treated at our hospital within the same time period.


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The data were calculated as means ± standard deviation or medians (interquartile range) for continuous variables and numbers (percentage) for categorical variables. Bivariate comparisons between patients, with and without abducens nerve palsy, in the VA dissecting group and supratentorial group were performed using Chi-square test (Fisher’s exact test was used when the expected cell frequency was <5) and Student’s t-test for categorical variables and continuous variables, respectively.

Informed consent was obtained from all patients or their representatives. Our local Ethics Committee approved the study protocol.

Results

A summary of all cases is shown in Table 1, and the details of 14 cases of VA dissecting aneurysm are shown in Table 2.

Of the 14 cases, 11 were male, with a mean age of 49.8 years (range, 42–62). All the 14 cases were treated with endovascular internal unilateral trapping of the involved VA. Eleven cases received operative intervention on an urgent basis after confirming diagnosis; however, three cases were treated after sometime because the initial 3DCT findings were unable to determine a definite diagnosis.

Although it was not possible to evaluate five cases because of unconsciousness, in the rest nine cases, seven (77.8%) cases demonstrated abducens nerve palsy. In these seven cases, bilateral abducens nerve palsies were observed in four cases, ipsilateral to the aneurysmal location in two cases, and contralateral abducens nerve palsy was observed in one case.

The hematoma thickness ratio was significantly higher in VA dissecting aneurysm patients than in supratentorial aneurysm patients [0.14 ± 0.035 vs. 0.11 ± 0.026, respectively; *P* = 0.002; Figure 2]. In cases of VA dissecting aneurysm, no statistical significance in the hematoma thickness ratio was observed irrespective of whether abducens nerve palsy was observed or not. However, there was a trend toward an increased hematoma thickness ratio in patients of abducens nerve palsy [0.16 ± 0.031 vs. 0.11 ± 0.017; *P* = 0.09; Figure 2]. In abducens nerve palsy patients, hematoma thickness ratio was significantly higher in cases of vertebral artery dissecting aneurysms than in cases of supratentorial aneurysms. In cases of vertebral artery dissecting aneurysm, there was a trend toward an increased hematoma thickness ratio after hematoma was washed out.
Table 2: Summary of the cases with ruptured vertebral artery dissecting aneurysms

<table>
<thead>
<tr>
<th>Case</th>
<th>Age/sex</th>
<th>Location of dissecting aneurysm</th>
<th>Abducens nerve palsy</th>
<th>Pre-HTR</th>
<th>WFNS grade</th>
<th>Fisher group</th>
<th>Treatment</th>
<th>Post-mRS</th>
<th>Period of abducens nerve palsy</th>
<th>Post-HTR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60/male</td>
<td>BA</td>
<td>Bilateral</td>
<td>0.23</td>
<td>3</td>
<td>4</td>
<td>Trapping</td>
<td>2</td>
<td>Not recover</td>
<td>0.13</td>
</tr>
<tr>
<td>2</td>
<td>59/female</td>
<td>Right VA</td>
<td>Contralateral</td>
<td>0.17</td>
<td>4</td>
<td>4</td>
<td>Trapping</td>
<td>5</td>
<td>1 week</td>
<td>0.09</td>
</tr>
<tr>
<td>3</td>
<td>48/male</td>
<td>Right VA</td>
<td>Bilateral</td>
<td>0.13</td>
<td>2</td>
<td>3</td>
<td>Trapping</td>
<td>3</td>
<td>2 weeks</td>
<td>0.12</td>
</tr>
<tr>
<td>4</td>
<td>51/female</td>
<td>Right VA</td>
<td>Bilateral</td>
<td>0.13</td>
<td>2</td>
<td>4</td>
<td>Trapping</td>
<td>0</td>
<td>6 months</td>
<td>0.06</td>
</tr>
<tr>
<td>5</td>
<td>51/male</td>
<td>Left VA</td>
<td>-</td>
<td>0.14</td>
<td>5</td>
<td>4</td>
<td>Trapping</td>
<td>6</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>62/male</td>
<td>Right VA</td>
<td>-</td>
<td>0.13</td>
<td>4</td>
<td>3</td>
<td>Trapping</td>
<td>6</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>41/male</td>
<td>Right VA</td>
<td>-</td>
<td>0.09</td>
<td>4</td>
<td>3</td>
<td>Trapping</td>
<td>2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>56/male</td>
<td>Left VA</td>
<td>Ipsilateral</td>
<td>0.14</td>
<td>2</td>
<td>4</td>
<td>Trapping</td>
<td>0</td>
<td>1 week</td>
<td>0.11</td>
</tr>
<tr>
<td>9</td>
<td>45/male</td>
<td>Left VA</td>
<td>-</td>
<td>0.18</td>
<td>5</td>
<td>4</td>
<td>Trapping</td>
<td>5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>35/male</td>
<td>Left VA</td>
<td>Ipsilateral</td>
<td>0.16</td>
<td>2</td>
<td>4</td>
<td>Trapping</td>
<td>6</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>49/female</td>
<td>Right VA</td>
<td>-</td>
<td>0.13</td>
<td>5</td>
<td>4</td>
<td>Trapping</td>
<td>0</td>
<td>-</td>
<td>0.10</td>
</tr>
<tr>
<td>12</td>
<td>44/male</td>
<td>Bilateral VA</td>
<td>-</td>
<td>0.08</td>
<td>5</td>
<td>4</td>
<td>Trapping</td>
<td>6</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>53/male</td>
<td>Right VA</td>
<td>-</td>
<td>0.14</td>
<td>5</td>
<td>3</td>
<td>Trapping</td>
<td>6</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>14</td>
<td>43/male</td>
<td>Right VA</td>
<td>Bilateral</td>
<td>0.17</td>
<td>2</td>
<td>4</td>
<td>Trapping</td>
<td>0</td>
<td>1 week</td>
<td>0.10</td>
</tr>
</tbody>
</table>

BA – Basilar artery; VA – Vertebral artery; WFNS – World Federation of Neurosurgery; Pre-HTR – Hematoma thickness ratio in initial CT; Post-HTR – Hematoma thickness ratio after hematoma was washed out; mRS – Modified Rankin scale; CT – Computed tomography

Discussion

Abducens nerve palsy may be observed as a false localizing sign in patients with raised intracranial pressure (ICP) due to any cause. The vulnerability of abducens nerve palsy in patients of raised ICP is generally attributed to its long intracranial course.\(^\text{[1-4]}\) Patel et al. reported the annual incidence of abducens nerve palsy of 11.3 per 100,000 patients.\(^\text{[5]}\) In their study, 5 of 137 cases of abducens nerve palsy were related to the presence of an aneurysm. In cases of SAH, the reported prevalence of abducens nerve palsy varies between 2.43% and 5.90%.\(^\text{[6]}\) In particular, in the cases of SAH associated with ruptured VA dissecting aneurysms, abducens nerve palsy was observed in 26.9% of cases.\(^\text{[6]}\) In the present case series, abducens nerve palsy was observed in 50% of cases (7/14), which was higher than previously reported. Moreover, if we exclude those patients in whom an initial neurological examination was not possible because of unconsciousness, the actual prevalence of abducens nerve palsy in our study was 77.8% (7/9). This finding indicates a relationship between abducens nerve palsy and VA dissection.

We were able to identify 14 previous studies, which investigated the cause of abducens nerve palsy in cases of ruptured VA dissecting aneurysms \([\text{Table 3}]\text{[1,4,6-15]}\). The following etiologies have been postulated: (1) direct compression by aneurysm;\(^\text{[1,9]}\) (2) direct mechanical pressure of hematoma;\(^\text{[3,4,6,11,12]}\) (3) vasospasm of the pontine branches of the basilar artery;\(^\text{[16]}\) (4) direct arterial jetting caused by aneurysmal rupture, adjacent to the nerve;\(^\text{[2]}\) (5) increased ICP due to acute hydrocephalus.\(^\text{[10,14]}\)

As suggested by a number of authors, we considered the direct mechanical pressure of hematoma against the brainstem and thereby stretching adjacent nerves lying between the brain and the skull as the most frequent cause of abducens nerve palsy associated with SAH. Till date, no previous studies have reported statistical evaluations of hematoma thickness within the prepontine cistern. To the best of our knowledge, this is the first report to do so.

We considered direct aneurysmal compression unlikely in the present case series because of the small size of identified aneurysms. Vasospasm of the pontine branches of the basilar artery has been shown to cause abducens nerve palsy of the nuclear type, which is associated with gaze paresis and/or facial paresis due to the close anatomical relationship between these nuclei. We could not find any ischemic lesions on diffusion-weighted magnetic resonance imaging. Although direct arterial jetting due to aneurysmal rupture may cause nerve injury, this mechanism is thought to result in permanent symptoms. SAH leading to acute obstructive hydrocephalus has been reported as a cause of abducens nerve palsy due to raised ICP. However, increased ICP often occurs more commonly in cases of ruptured supratentorial aneurysm. Therefore, we concluded that direct mechanical pressure by hematoma is the most likely cause of abducens nerve palsy.

The findings in the present case series demonstrate that hematoma clots within the prepontine cistern were significantly thicker in cases of VA dissecting aneurysm than in cases of supratentorial aneurysm. In cases of VA dissecting aneurysm, no statistically significant difference...
in the thickness of prepontine cistern clots was observed; however, there was a trend toward an increased frequency of abducens nerve palsies. These results indicate that patients presenting with bilateral abducens nerve palsies and prepontine cistern thick hematoma are more likely to have a ruptured VA dissecting aneurysm, even in the absence of findings on initial 3DCT or angiography.

There were some limitations in our study. The thickness of hematoma within the prepontine cistern was evaluated using axial CT imaging only. It is not possible to reproduce similar CT imaging conditions for individual patients because even subtle angle changes can dramatically alter the resultant findings. However, we believe the measurement of local hematoma volume to be a key strength of the present study. Further studies with a larger patient number are required to validate the findings of the present study.

Conclusions

The cause of abducens nerve palsy associated with SAH is direct mechanical pressure by a hematoma against the brainstem, thereby stretching the adjacent nerves lying between the brain and the skull. Clots within the prepontine cistern were significantly thicker in case of VA dissecting aneurysm than in the case of supratentorial aneurysm. Therefore, patients presenting with abducens nerve palsy and prepontine cistern thick hematoma are more likely to have a ruptured VA dissecting aneurysm.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References