Putaminal petechial haemorrhage as the cause of chorea: a neuroimaging study

Ming-Hong Chang, Hung-Ting Chiang, Ping-Hong Lai, Chern-Guey Sy, Susan Shin-Jung Lee, Yeung-Yuk Lo

Abstract
Objectives—A hyperintense putamen on either CT or MRI as a finding associated with chorea has occasionally been described and is almost always associated with non-ketotic hyperglycaemia. The cause of the hyperintensity of the striatum in these images is still controversial. Some reports have found that calcification was responsible whereas others have advocated petechial haemorrhage as the cause. The purpose of this study was to determine whether hyperintense striata are caused by petechial haemorrhage or calcification, with the sequential imaging changes.

Subjects and methods—Five patients presenting with an acute onset of either hemichorea or generalised chorea and showed either unilateral or bilateral hyperdense striatum on the initial CT were assessed. Neuroimaging studies including sequential CT and MRI examinations and detailed biochemical tests were performed.

Results—Three patients had pronounced hyperglycaemia and the other two patients had no biochemical abnormalities. In all patients, the first CT scans, performed within two weeks of the onset of chorea, showed a high density over the striatum contralateral to the chorea, which diminished or disappeared two months later. T1 weighted imaging disclosed hyperintense signals over the striatum contralateral to the chorea on admission which diminished two months later. T2 weighted imaging at two months showed hyposignal intensity changes corresponding to the area with hypersignal changes on T1 weighted images, implying haemosiderin deposition.

Conclusion—Based on the evolution of clinical manifestations and the findings of neuroimaging, putaminal petechial haemorrhage might be a new entity causing either hemichorea or generalised chorea.

Keywords: petechial haemorrhage, striatum, CT, MRI

Chorea may be the manifestation of a wide variety of degenerative, vascular, metabolic, or toxic disorders involving the CNS, in which dysfunction of the basal ganglia, particularly of the striatum, is generally assumed to be responsible. Various structural lesions in the contralateral subthalamic nucleus and striatum seem to play a critical part in the development of chorea, with acute vascular accident being the most common pathological process. Since the advent of CT, there have been occasional reports of chorea development secondary to striatal pathology, which is commonly seen as a lesion of increased density. There is much controversy regarding the cause of the striatal hyperdensity on CT; some advocate calcification, and others have reported petechial haemorrhage to be the cause. It has also been questioned whether non-ketotic hyperglycaemia is the only systemic disease associated with a hyperdense putamen manifesting as chorea. Previous reports of imaging findings were only single case reports. We collected five patients with chorea whose CT disclosed a hyperdense striatum. A series of sequential neuroimaging examinations, including CT and MRI, were performed within two weeks of the onset of chorea and two months later. Based on these imaging studies, we attempted to differentiate whether the hyperdensity of the striatum on CT was caused by calcification or haemorrhage.

Materials and methods
Within a period of two years, we encountered five choreic patients (four men, one woman) whose brain CT on admission showed a hyperdensity over the basal ganglion contralateral to the choreic side. Their age ranged from 60 to 73 years. Those who clearly had underlying diseases causing the chorea, such as hyperthyroidism, hereditary diseases, or drugs history, were excluded. Biochemical tests, including Hba1c and osmolality, and coagulation function were obtained. Brain CT and MRI were performed within two weeks after the onset of chorea and repeated two months later. Brain CT was performed with a Somatom HiQ scanner (Siemens) in the axial plane with an 8 mm thickness without contrast enhancement. Images were obtained on admission and two months later. Brain MRI was performed on a 1.5 T superconducting system (Sigma; GE Medical Systems). Spin echo T1 weighted images with 600/20/2 (repetition time/echo time/excitations) and T2 weighted images with 2500/90/1 were obtained in the axial plane. Images were 5 mm thick with a 2.5 mm gap between sections. The acquisition matrix was
Table 1 Summary of clinical data

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age/sex</th>
<th>Limbs with chorea</th>
<th>Serum osmolality</th>
<th>HbA1C</th>
<th>Use of haloperidol</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>71/M</td>
<td>RU, LUI, LLol</td>
<td>102</td>
<td>5.1</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>68/M</td>
<td>RU</td>
<td>94</td>
<td>5.6</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>73/M</td>
<td>LUI, LLI, RUI</td>
<td>413</td>
<td>10.7</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>60/F</td>
<td>RUI, RLol</td>
<td>364</td>
<td>11.3</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>68/M</td>
<td>RUI</td>
<td>476</td>
<td>10.6</td>
<td>No</td>
</tr>
</tbody>
</table>

R = right; L = left; U = upper; Lo = lower; l = limb.

 victorious in hypertensive haemorrhages. Subsequently the striatum should be ascribed to petechial haemorrhage or calcification remains unsettled. The CT findings in our five patients were similar to those described by Sanfield et al and Nakagawa et al. Brain MRI was obtained within two weeks after the onset of chorea. The T1 weighted images clearly showed a hypersignal intensity over the basal ganglia which may be explained by the presence of methaemoglobin resulting from petechial haemorrhages. Subsequent resolution of this hypersignal intensity on T1 weighted images with increased hyposignal intensity on T2 weighted images over the basal ganglia, and the continual diminution or disappearance of high densities on CT imaging on follow up, all suggest that the process may be due to the evolution of petechial haemorrhage with haemosiderin deposition.

The differential diagnosis of a hyperdense putamen on CT includes calcification and petechial haemorrhage. A definite distinction can be made by noting the time course of the clinical and signal changes and by direct measurement of tissue density in Hounsfield units. Although it is not possible to entirely exclude calcification based on imaging abnormalities alone, such rapid resolution of the hyperdensity lesions over a few months it is certainly more likely to be caused by haemorrhage than calcification. In addition, the clinical presentation of our patients with acute onset of chorea also favoured haemorrhage rather than a chronic process such as calcification as the cause of image hyperdensity. Lastly, the tissue density values in the hyperdense putamen were in the range of 40 to 50 Hounsfield units, a finding similar to that for petechial haemorrhage, whereas measurement of the calcified choroid plexus and pineal glands in the same patients gave values of around 80 Hounsfield units. All the above findings supported petechial haemorrhage as a cause of the hyperdense putamen. Therefore, we propose that putaminal petechial haemorrhage is a new entity, that may be an uncommon but not rare, causing chorea.

Altafullah et al and Nakagawa et al first reported two patients with non-ketotic hyperglycaemic chorea whose neuroimages had faintly increased densities over the contralateral basal ganglion, suggesting petechial haemorrhage. Therefore, they proposed that non-ketotic hyperglycaemia complicated by multiple putaminal petechial haemorrhage was the cause of chorea. Since then, it has been generally accepted that non-ketotic hyperglycaemia, whose CT showed unilateral or bilateral faint hyperdensity over the putamen. They concluded that these lesions were best ascribed to calcification. Altafullah et al and Nakagawa et al also reported two patients with non-ketotic hyperglycaemic chorea whose CTs disclosed faintly increased densities over the contralateral basal ganglion. Brain MRI of these patients suggested that the hypersignal intensity was the result of petechial haemorrhage. Because the chorea subsided either spontaneously or after medication, necropsy is not readily available. Therefore, the issue of whether the faintly increased densities over the striatum should be ascribed to petechial haemorrhage or calcification remains unsettled. The CT findings in our five patients were similar to those described by Sanfield et al and Nakagawa et al. Brain MRI was obtained within two weeks after the onset of chorea. The T1 weighted images clearly showed a hypersignal intensity over the basal ganglia which may be explained by the presence of methaemoglobin resulting from petechial haemorrhages. Subsequent resolution of this hypersignal intensity on T1 weighted images with increased hyposignal intensity on T2 weighted images over the basal ganglia, and the continual diminution or disappearance of high densities on CT imaging on follow up, all suggest that the process may be due to the evolution of petechial haemorrhage with haemosiderin deposition.

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NEUROIMAGING FINDINGS

Brain CT of patients 1 and 3 showed bilateral, faintly increased densities over the putamen or caudate nucleus. Patients 2, 4, and 5 had similar hyperdense lesions over the striatum contralateral to the chorea, which had diminished or disappeared on follow up examination. T1 weighted imaging of these lesions initially disclosed a hypersignal intensity, which corresponded to the high density lesions found on CT, over the striatum contralateral to the chorea, which also diminished two months later (figure). T2 weighted imaging showed slightly increased or isodense changes in the striatum at the first examination. The increased hyposignal intensity observed on T2 weighted images on the follow up examination corresponded to the area with hypersignal intensity on T1 weighted imaging (table 2).

Discussion

Jones et al first described a patient with hypertensive putaminal haemorrhage presenting with hemichorea. Brain CT showed a dense and homogeneous haematoma, a common finding in hypertensive haemorrhages. Subsequently, Sanfield et al and Inbody et al described two choreic patients, with and with-
caemia is the sole cause of putaminal petechial haemorrhages manifesting with chorea. However, our series of patients included two with non-diabetic chorea whose brain CTs indicated the presence of petechial haemorrhage. Furthermore, a series of examinations in these patients excluded coagulation diseases or other systemic disorders, which can contribute to the haemorrhage. Therefore, our findings in these patients showed that non-ketotic hyperglycaemia is not the only possible cause of petechial haemorrhage over the basal ganglia in patients presenting with chorea.

The clinical range of putaminal haemorrhage is one of great variability, directly reflecting the size of the haematoma and its pattern of extension. The classic manifestations of massive putaminal haemorrhage are relatively abrupt onset of unilateral motor and sensory deficits with accompanying visual abnormalities and change of consciousness. Since the advent of CT, precise determination of the size and location of hyperdense and homogeneous haematoma is possible. Recently, a syndrome of the small haematoma have been reported, and chorea is one of its clinical manifestations. Although the haematoma is small, it is very dense and homogenous and not restricted to the putamen and head of the caudate nucleus.
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date nucleus. These findings are different from the imaging studies of our patients, which showed sharply defined lesions of faintly increased density without a mass effect, which were located strictly within the striatum. These findings are also very different from those of common hypertensive intracranial haemorrhages. To date, our series is the largest to describe the CT and MRI findings of petechial haemorrhage in patients with or without diabetes, manifesting with chorea.

Most patients with chorea showed partial or complete resolution of symptoms after treatment with neuroleptic drugs, but this treatment usually had to be continued for a long period. However, when the cause of the choreas was a stroke, the chorea usually remitted spontaneously.1 Choreas associated with non-ketotic hyperglycaemic chorea has also been reported to show dramatic improvement after correction of hyperglycaemia.14 All of our patients had a good prognosis and the hyperkinesia disappeared within three weeks, implying that chorea secondary to putaminal petechial haemorrhage is a benign and limited disease.

Non-ketotic hyperglycaemia has occasionally been associated with various neurological abnormalities15 16 in which choreoathetosis is a rare manifestation.17 18 Recently, we19 and Lin et al20 described several patients who presented with non-ketotic hyperglycaemic chorea. Neuroradiological studies including CT and MRI do not show the changes we have found. Therefore, we can clearly separate non-ketotic hyperglycaemic choreic patients into two groups, one with petechial haemorrhage and the other without definite structural lesions. At present, we are unable to predict which patients will develop putaminal haemorrhage. This implies that a critical unidentified factor plays an important part in the production of this pattern of haemorrhage and further investigation is required to determine this factor.

1 Martin TP, Alosh N. Hemichorea associated with a lesion of corpus lysis. Brain 1934;57:504–5.