According to the prevalence of the CT scan, it is easy to recognize cerebral hemorrhage. At present, the incidence of the recurrence in cerebral hemorrhage is reported to be 2 – 10% or 8.6% \(^1\). The frequency of multiple cerebral hemorrhage which occur simultaneously is about 2 – 2.6%, and mostly occur simultaneously in the bilateral cerebral hemisphere or in the supra- and infra-tentorial brain \(^2\text{--}^{18}\). We presented the second case in Japan of multiple intracerebral hemorrhage occurring in the ipsilateral basal ganglia.

**CASE REPORT**

A 75-year-old man visited our hospital because of the disturbance of consciousness and speech, and right hemiparesis on July 14, 1999. Forty-five minutes before the arrival, he dropped his teacup in his food and was unable to respond to his wife's talk.
The patient was diagnosed as a cerebral hemorrhage. The patient had been continued with antihypertensive therapy from the age of 57. He usually drank a moderate amount of alcohol but had no history of smoking. The pulse rate and blood pressure on admission were 45/min, and 208/92 mmHg, respectively. His body weight was 75.0 kg, and height 170 cm. Conjunctivas were intact. We could find no abnormality on chest and abdominal examination. On neurological examination, the patient was somnolent and barely opened his eyes when called. His speech was fluent, with paraphasia. The pupils were round and equal in size, and showed a sluggish light reflex. Dysarthria was evident and gag reflex was disturbed. Muscle strength was decreased to 2/5 on the right arm, and 3/5 on the right leg, at the manual muscle testing (MMT). The deep tendon reflexes were increased on the right upper and lower extremities, and right Wartenberg reflex and Babinski’s sign were present. Right hemihypesthesia was also recognized. No abnormality was found on blood examination at admission. As to the additional examination, Antithrombin III was 90%, TAT was under 2.0 ng/ml; protein C was 106%, and protein S was 80%; the all were within normal limits. Immunological examination, such as anti-nuclear antibody (ANA) and anti-DNA antibody showed no abnormality. No evidence suggesting about angitis or leukemia was obtained.

A computed tomographic (CT) scan of the brain (Fig. 1), obtained 45 minutes after the onset, revealed a large high-density area in the left putaminal region and a small high-density area in the left thalamic

Fig. 1 A computed tomographic scan of the brain, obtained 45 minutes after the onset.
Taking left posterior limb of internal capsule as a boundary, a high density area existed mainly in the left putamen, and another high density area mainly in the left thalamus.

Fig. 2 Magnetic resonance imaging of the brain, performed at the ninth day after the onset (T2-weighted images).
The high intensity area mainly on the left putamen (→).
The other high intensity area mainly on the left thalamus (⇒).
Magnetic resonance imaging (MRI) of the head, performed on the 9th day after the onset (Figs. 2, 3) revealed two high density lesions in the left putamen and in the left thalamus. These were independent and isolated and had appeared in the same region. Thin-slice MRI of the head on the 45th day after the onset, two hematomas were independent and isolated in the left putaminal region and in the left thalamic region. It appeared that these hemorrhage had occurred simultaneously on the same side of the supratentorial brain. Cerebral angiography on the 83rd day after the onset, no findings such as arteriovenous malformation (AVM), brain tumor, or aneurysm were indicated.

A gradient echo study using MRI showed no signs of cavernous hemangioma. Therefore, we diagnosed this case as having two ipsilateral hypertensive hemorrhage which had occurred simultaneously.

By the medical treatment and rehabilitation, his muscle strength improved to 3–4/5 in the right arm and to 5/5 in the right leg at the MMT. His aphasia was classified as Wernicke's aphasia because he had good comprehension of listening and reading, and he spoke fluently but with many lapses into nonsense, although the proportion of nonsense speech decreased in accordance with improvement in comprehension of short sentences. His was discharged on the 114th day after the onset.

Table 1  Location of multiple intracerebral hemorrhage

<table>
<thead>
<tr>
<th>Location</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Multiple simultaneous bilateral intracerebral hemorrhage</td>
<td></td>
</tr>
<tr>
<td>Thalamus / Putamen</td>
<td>7</td>
</tr>
<tr>
<td>Bilateral putamen</td>
<td>8</td>
</tr>
<tr>
<td>Bilateral thalamus</td>
<td>6</td>
</tr>
<tr>
<td>Putamen / Subcortical</td>
<td>2</td>
</tr>
<tr>
<td>Thalamus / Subcortical</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>25</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location</th>
<th>Age</th>
<th>Sex</th>
<th>Years</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left putamen / Left parietal lobe</td>
<td>67</td>
<td>M</td>
<td>1988</td>
<td>Nakamura et al</td>
</tr>
<tr>
<td>Left putamen / Left thalamus</td>
<td>75</td>
<td>M</td>
<td>1988</td>
<td>our case</td>
</tr>
</tbody>
</table>
DISCUSSION

Multiple simultaneous intracerebral hematomas are generally considered to occur at the same time. Matsuda et al. 19) defined multiple simultaneous intracerebral hematomas as hematomas which can be confirmed as two or more high-density areas on brain CT scan performed within 24 hours after the onset.

In the present case, CT scan performed 45 minutes after the onset showed the two hematomas: one was in the left putamen and the other was in the left thalamus. The putaminal hemorrhage appeared to be a hemorrhage from the lenticulostriate artery and the thalamic hemorrhage from a thalamus-penetrating branch of the vertebrobasilar artery. From the findings of angiography and MRI, we diagnosed considered this case as simultaneous multiple cerebral hemorrhage at putaminal and thalamic region.

At present, most multiple simultaneous intracerebral hematomas are thought to occur in the supratentorial brain. As shown in Table 1, 27 cases of multiple simultaneous intracerebral hematomas in the supratentorial brain are reported in Japan. In twenty-five of all cases, the hematomas occurred in the bilateral cerebral hemisphere. 80 percent of hematomas are in the bilateral basal ganglia, with almost same rate seen in the thalamus/putamen, bilateral putamen, bilateral thalamus and basal ganglia. However, only two of 27 cases were with ipsilateral intracranial hemorrhage; one is reported by Nakamura et al. 20) in 1988 (left putamen and left parietal cortex) and the other is the present (left putamen and left thalamus).

According to Charcot (1868) et al., there are two possible mechanisms in the pathogenesis of multiple simultaneous hypertensive intracerebral hemorrhage. The first is the rupture of two cerebral microaneurysms due to necrosis of blood vessels, rapidly followed by plasmatic necrosis of the blood vessels, and the second is a change in hemodynamics which secondarily causes the failure of other arteriovenous and small penetrating vessels 12)14)19). From the cases previously reported, two individual cerebral hemorrhage were recognized at 1 to 3 hours after the first attack 8)12)14)20). In this case, we recognized two high density areas in 45 minutes after the onset of his complains. Thus, the mechanism of this case may be related to two microaneurysmal ruptures. However, this is merely speculation, since the period and range over which the changes in hemodynamics take place are not known.

The number of the cases of simultaneous intracerebral hematomas in the cerebral hemisphere is extremely rare than that in the bilateral cerebral hemisphere. From our experience, the case of ipsilateral simultaneous intracerebral hematomas could exist more often than not. The reason why ipsilateral simultaneous intracerebral hematomas is rare is probably the existence of mixed type of intracerebral hemorrhage, some of which are multiple ipsilateral simultaneous intracerebral hematomas that have two sources of hemorrhage, but can not be confirmed the sources. It could apply to especially large lesion with evolution.

As two lesions of cerebral hemorrhage were recognized in ipsilateral (left side) cerebral hemisphere, we examined left internal carotid artery and left vertebrobasilar artery about the existence of vascular abnormality including arteriovenous malformation and could find no abnormality. From these results, we suspected that the attack of cerebral hemorrhage that recognized in ipsilateral hemisphere may occurred incidentally.

Some recent reports claim that leukoaraiosis is related to cerebrovascular disorders 21). There are also reports maintaining that leukoaraiosis can become a risk factor for cerebral hemorrhage as well as for hypertension 22). Matsuda et al. report that leukoaraiosis is frequently observed at a high rate in cases of multiple simultaneous hypertensive hemorrhage 19). The present case had no evidence of leukoaraiosis besides multiple lacunar infarctions in the bilateral basal ganglia combined
with putaminal lesions. We presume that the present patient had hypertensive changes in the penetrating vessels.

The prognosis of the patients with simultaneous multiple bilateral intracerebral hemorrhage is generally considered to be poor \(^{19}\). But, Nakamura's case \(^{20}\) (1988) recovered completely with medication and was neurologically normal at the time of discharge. In our case, hemiparalysis recovered to the extent for him to be able to transfer himself to a wheelchair from the bed with little assistance. These two cases did much better than others did.

Putaminal hemorrhage is classified into five types according to CT \(^{23}\). This classification focuses on showed the invasion of hematoma around putamen, especially this is correlated with neurological grading. Firstly the putaminal hemorrhage was identified as 1 type localized outside of capsula interna and the thalamic hemorrhage was identified as 1a type localized in the thalamus, both of lesions did not reach capsula interna. Secondary most acute enlargement cases of hypertensive intracerebral hemorrhage are attributed to the management failure of blood pressure in 12 to 24 hours after onset. The present case, however, could be avoid acute enlargement of hematoma by intensive management of blood pressure from the early stage and obtain favorite prognosis including survival and physiological function.

Besides, In the cases of bilateral intracerebral hemorrhage, we occasionally experience the symptom of pseudobulbar palsy. But in this case, this symptom that sometimes induces severe conditions such as aspiration pneumonia or respiratory obstruction, was not recognized. This may be the another reason for good clinical course in this case.

**References**


抄 録
同時発症した高血圧性同側被殻，視床出血の 1 例

本間 裕子 堀田 宗文 加茂 力
白石 幹 杉原 浚 高橋 洋一

症例は 75 歳男性，既往に高血圧を認める。突然，失語を発症し，その後意識障害にいたり入院となる。来院直後の頭部 CT 上，左被殻と左視床に高吸収域を認め，フォローの MRI 上では，2 つの独立した病変を認め，同時に出発した高血圧性脳出血と診断した。保存的治療により，症状の改善を認め，CT 上高吸収域は吸収され，発症 114 病日に退院となった。著者が調べた限り，本症例を含め，本邦報告例の同時多発脳出血は 27 例であり，うち 25 例は両側大脳半球に病変を認め，同側に同時多発病変を認めた脳出血は 2 例目であった。一般的に，同時多発性の高血圧性脳出血は予後不良であるが，本症例の予後はきわめて良好であり，示唆に富む症例であった。

（聖マリアンナ医大誌 30: 487-492, 2002）