

# Bilateral Putaminal Hemorrhage with Cerebral Edema in Hyperglycemic Hyperosmolar Syndrome

Soo-Jin Cho<sup>1</sup>, Tae Kyoung Won<sup>1</sup>, Seung-ju Hwang<sup>2</sup>, and Joong Hyuck Kwon<sup>1</sup>

<sup>1</sup>Department of Neurology & Internal Medicine, Sejong General Hospital, Kyonggi-do, Korea;

<sup>2</sup>Department of Family Medicine, Shincheon Union Hospital, Kyonggi-do, Korea.

Bilateral putaminal hemorrhages rarely occur simultaneously in hypertensive patients. The association of intracerebral hemorrhage with cerebral edema (CE) has been rarely reported in diabetic patients. We present a patient with bilateral putaminal hemorrhage (BPH) and CE during the course of hyperglycemic hyperosmolar syndrome (HHS). A 40-year-old man with a history of diabetes mellitus and chronic alcoholism was admitted with acute impaired mentality. His blood pressure was within the normal range on admission. Laboratory results revealed hyperglycemia and severe metabolic acidosis without ketonuria. After aggressive treatment, plasma sugar fell to 217 mg/dl, but brain CT showed BPH and diffuse CE. Our case demonstrated that HHS should be considered as a cause of BPH with CE. Initial brain imaging study may be recommended for patients with diabetic coma.

**Key Words:** Diabetic coma, hyperglycemic hyperosmolar syndrome, cerebral edema, putaminal hemorrhage, intracerebral hemorrhage

## INTRODUCTION

The putamen is the most common site of spontaneous intracerebral hemorrhage (ICH) that is usually related to hypertension.<sup>1</sup> Bilateral, simultaneous ICH is a rare occurrence in patients with hypertension, but other etiologies have been reported.<sup>2-5</sup>

Cerebral edema (CE) is one of the serious complications of diabetic ketoacidosis (DKA) and is more common in diabetic children or patients

with types I diabetes mellitus.<sup>6</sup> The association of CE with bilateral putaminal hemorrhages (BPH) has not been fully investigated and has been reported only in patients with DKA.<sup>7-9</sup>

We present a patient with BPH and CE during the course of hyperglycemic hyperosmolar syndrome (HHS)

## CASE REPORT

A 40-year-old man with a history of type II diabetes mellitus was admitted to the emergency department of an outside hospital with acute impaired mentality. Diabetes mellitus had been diagnosed 6 years previously, and treated with subcutaneous insulin. Just prior to the recent admission, he had drunk for 2 days without injection of insulin and his wife discovered him unresponsive that morning.

He was unresponsive to pain and had shallow respiration. His pupils were fixed and dilated. Laboratory results were as follows, blood glucose 615 mg/dl, blood urea 25 mg/dl, estimated osmolarity 328 mOsm/L, blood pH 6.86, PCO<sub>2</sub> 34.1 mmHg, and glycosuria without ketonuria. After two-hours of intravenous hydration with normal saline, insulin and bicarbonate, he was referred to our hospital.

His blood glucose was 404 mg/dl, blood pH 7.046, and PCO<sub>2</sub> 15 mmHg on admission. Plasma glucose fell to 213 mg/dl 4 hours after admission and renal and hepatic function tests remained normal throughout admission. Blood cultures revealed bacteremia with staphylococcus epidermidis on admission, but there were no signs of

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Reprint address: requests to Dr. Soo-Jin Cho, Department of Neurology, Sejong General Hospital, 91-121, Sosa Bon 2-Dong, Sosa-Gu, Bucheon-si, Kyonggi-do 422-232, Korea. Tel: 82-32-340-1128, Fax: 82-32-349-3005, E-mail: dowonc@naver.com

any regurgitation murmur or vascular phenomena suggesting endocarditis. Brain CT performed 14 hours after admission showed BPH and CE (Fig. 1), intravenous 10% glycerin (cerostril<sup>®</sup> 500 - 1000 cc/day) was infused for treatment of CE.

Neurological examination performed one day after admission detected normal brainstem reflexes, minimal withdrawal responses to noxious stimuli, and decreased deep tendon reflexes without ankle jerk. He was mute and uncooperative. Neurological examination on hospital day 18 revealed spontaneous roving movement of his eyes, an increased generalized rigidity, and hyperreflexia. Brain CT performed 2 years after his insult showed extensive ventriculomegaly, cerebromalacia on the bilateral striatum, and low density on the bilateral subcortex (Fig. 2). During the two years he had never gained any consciousness nor shown any meaningful movement.

## DISCUSSION

HHS and DKA are severe states of acute decompensated diabetic mellitus, sometimes associated with life-threatening neurologic complications.<sup>10</sup> Seizures and impaired mental status occur more frequently in patients with HHS, while CE occurs in approximately 1 percent of children with DKA.<sup>11,12</sup> CE is far less common in patients with HHS, but our patient was classified as HHS according to the American Diabetes Association's nomenclature.<sup>13</sup>

Bilateral simultaneous ICH occurs in 2.6% of patients with hypertensive ICH and is rarely associated with migraine.<sup>1-2</sup> Hemorrhagic transformation after ischemia was considered as a possible mechanism of bilateral hemorrhage in neonates with perinatal asphyxia or in patients with methanol intoxication or lightning strike.<sup>3-5</sup> Some investigators reported recently that ICH was one of the neurological complications in patients with HHS or DKA.<sup>7,9,14</sup> The association of ICH with CE was suspected, and ischemic insults or loss of autoregulation related to hyperglycemia was considered as a possible mechanism for both pathologies.<sup>7-9</sup> Our patient had neither a history of hypertension nor any of the possible causes of bilateral ICH listed above. In addition, bilateral



Fig 1. Brain CT 14 hours after admission showing bilateral putaminal hematoma (BPH) and diffuse cerebral edema (CE).



Fig 2. Brain CT 2 years after his insult showing extensive ventriculomegaly, cerebromalacia on the bilateral putamen, and low density on the bilateral subcortex.

ICH in this case appeared temporally related to HHS.

The depressed mental status from initial presentation was not fully explained by the patient's metabolic derangement or sepsis. His blood osmolality was not high enough to cause coma,<sup>10</sup> and

he did not improve after the correction of his metabolic abnormalities. In addition, CE was not compatible with typical neuroimaging findings of septic encephalopathy.<sup>15</sup> Consequently, bilateral ICH and CE might have depressed the initial mental status in this case.

Rapid correction of blood sugar has been considered to be a precipitant for CE, but CE was reported before the initiation of treatment of diabetic coma.<sup>16,17</sup> Recently, lower initial partial pressures of arterial carbon dioxide (PCO<sub>2</sub>) and higher initial serum urea nitrogen were reported as significant risk factors for CE in children with DKA.<sup>12</sup> Cerebral ischemia secondary to the vasoconstriction related to low PCO<sub>2</sub> might be a plausible mechanism of CE.<sup>12</sup> The PCO<sub>2</sub> level of our patient was low enough to cause vasoconstriction, so there is a possibility that rapid correction of hyperglycemia and low PCO<sub>2</sub> might have aggravated CE in our case.

In conclusion, BPH with CE can occur in patients with HHS. Brain imaging study may be recommended for HHS patients with depressed mentality, especially those who osmolarity is not high enough to cause mental change.

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