

# Pure Sensory Stroke Due to Bilateral Basal Ganglion Hemorrhage: A Case Report

## Bilateral Bazal Ganglion Hemorajisine Bağlı Pür Sensoryal Strok: Olgu Sunumu

### ABSTRACT

Bilateral simultaneous hypertensive intracerebral hemorrhages are extremely rare. The predisposing factors and pathophysiological mechanisms leading to the development of this picture are not well known. Possible mechanisms of simultaneous multiple hemorrhages include concomitant primary hemorrhages in two or more regions, or development of a second hemorrhage in another region shortly after the primary hemorrhage. The etiology of the cases presenting with bilateral simultaneous basal ganglion hemorrhage include migraine, lightning stroke, hyperglycemic hyperosmolar coma, hypertension and diabetic ketoacidosis coma. Bilateral simultaneous hemorrhage has a poor prognosis. The case of bilateral simultaneous intracerebral hemorrhage presented here had a good clinical course similar to a pure sensorial stroke.

**KEYWORDS:** Bilateral intracerebral hemorrhage, Sensorial stroke, Hypertension

### ÖZ

Bilateral simültane hipertansif intraserebral hemorajiler oldukça nadirdir. Bu tablonun oluşumundaki predispozan faktörler ve patofizyolojik mekanizmalar tam olarak bilinmemektedir. Aynı anda birden çok hemoraji gelişmesindeki olası mekanizmalar, iki veya daha fazla farklı bölgede aynı anda primer kanama olması veya primer kanamadan kısa bir süre sonra başka bir bölgede ikinci kanamanın olmasıdır. Bilateral simültane bazal ganglion hemorajisi görülen olguların etiyolojisinde migren, yıldırım çarpması, hiperglisemik hiperosmolar koma, hipertansiyon ve diyabetik ketoasidoz koması vardır. Bilateral simültane hemoraji kötü prognozlidir. Burada pür sensoryal strok kliniği gibi iyi seyirli olan, bilateral simültane intraserebral hemoraji olgusu sunulmaktadır.

**ANAHTAR SÖZCÜKLER:** Bilateral intraserebral hemoraji, Sensoryal strok, Hipertansiyon

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## INTRODUCTION

The pure sensory stroke (PSS) syndrome was first defined by Fisher in 1965, as a sudden unilateral sensory deficit, not accompanied by neurological signs and symptoms (3). The first pathophysiological study showed lacunar ischemia in the sensory nucleus of the contralateral thalamus to be the underlying cause of this syndrome. Ischemia in the mesencephalon and pons, the middle cerebral artery watershed area and the internal capsule posterior limb have been shown to be involved in PSS (1). In addition, hemorrhage, inflammation or tumors of these areas are also known to cause a clinical presentation of PSS (2).

Bilateral simultaneous hypertensive intracerebral hemorrhages (HICH) are extremely rare. Possible mechanisms of simultaneous multiple hemorrhages include concomitant primary hemorrhages in two or more regions, or development of a second hemorrhage in another region shortly after the primary hemorrhage (4).

PSS cases that developed due to hemorrhages in anatomic structures such as the thalamus, mesencephalon and pons have been previously presented in the literature; however, our report is the first to represent a case of PSS clinical presentation due to bilateral basal ganglion hemorrhage.

## CASE REPORT

A 63-year-old right-handed male patient was hospitalized in our clinic for advanced investigation, following referral from another centre where he was followed up for four days due to numbness of the left side. The patient had a history of hypertension for the last three years and was on irregular antihypertensive treatment. No other risk factors such as smoking, coagulopathy or vasculopathy could be established. A sudden numbness had developed on his left side four days ago during which his blood pressure was measured as 190/110 mmHg. Speech defects were observed during the incident, which lasted for two hours. No other complaint accompanied the numbness during the four-day period. Upon admission, his blood pressure was 140/80 mmHg and pulse rate was 98 beats/min and regular. Mental and cranial nerve examination was normal apart from the hemihypoesthesia, which involved the left inner side of his face, observed during the neurological

examination performed on the 4th day. Strength was preserved in all four extremities. Deep tendon reflexes and cerebellar system examination were normal. Plantar responses were bilateral flexor. Cranial computerized tomography (CT) (without contrast) obtained two hours after the onset of numbness displayed a hyperdense appearance of bilateral basal ganglions, which was more evident on the right side (Figure 1), and cranial magnetic resonance imaging (MRI) obtained four hours later showed an appearance (Figure 2) consistent with hypointense hyperacute hemorrhage in T1 and hyperintense hyperacute hemorrhage in T2 at the same locations. Cerebral magnetic resonance angiography was normal. Total blood count, biochemical analysis, erythrocyte sedimentation rate, CRP, complement levels, rheumatoid factor, fibrinogen, antithrombin III, INR, partial thromboplastin time and prothrombin time values were normal. No causative factors such as blood dyscrasias, AVM, angioma, septicemia, malignancies or sinus thrombosis were identified. Antiedema therapy was not initiated. Follow up cranial CT showed the hematomas to gradually resolve in several weeks. The patient's complaints improved significantly while her neurological examination became normal on the 45th day. No further complaints were observed for two years following arterial blood pressure regulation.

## DISCUSSION

Development of simultaneous intracerebral hemorrhage in different arterial regions is a very rare clinical picture. The predisposing factors and pathophysiological mechanisms leading to the development of this picture are not well known. Symmetric rupture of cerebral microaneurysms is suggested to explain the development of bilateral hemorrhages. Another opinion involves the development of contralateral hemorrhage shortly after the development of unilateral hematoma due to rupture of microaneurysms (4).

Cases with simultaneous bilateral cerebral hemorrhage have been reported in the literature. Some of these cases have presented with bilateral basal ganglion hemorrhage, while hypertension was found to be a responsible factor in the etiology of six of the cases. The etiology of other cases presenting with bilateral simultaneous basal ganglion hemorrhage included migraine, lightning stroke,

hyperglycemic hyperosmolar coma and diabetic ketoacidosis coma (5, 7, 8, 9). On the other hand, multiple cerebral hemorrhages due to factors such as blood dyscrasia, angioma, septicemia, malignancies, sinus thrombosis and amyloid angiopathy may be observed (4, 7).

Bilateral intracerebral hemorrhages of even a small volume can end with deleterious clinical pictures such as loss of consciousness, tetraparesis, pseudobulbar paralysis and death. Poor cerebral blood flow and the diaschisis phenomenon can have possible roles in the poor prognosis observed in these patients (4, 6). Due to this poor prognosis, the common opinion concerning hypertensive bilateral intracerebral hemorrhage is the ease regarding consideration of surgical endication (6). PSS is characterized by hemisensory symptoms without other major neurological signs. It was initially attributed to thalamic lacunar infarction, but several reports have shown the PSS can be due to small infarcts involving the posterior part of the internal capsula, the cerebral cortex and the brainstem (1, 2, 3). PSS has not previously been reported with bilateral basal ganglion hemorrhage. Although bilateral basal ganglion hemorrhage was present in our case, observation of only left hemihypoesthesia during neurological examination suggested a possible damage in the right spinothalamic tract. The spinothalamic tract is a bundle of sensory axons ascending through the white matter of the spinal cord, carrying sensory information to the brain (3). It carries pain and temperature sensory information to the thalamus of the brain. From there, signals go to the cingulate cortex, the primary somatosensory cortex, and insular cortex respectively. A unilateral lesion usually causes contralateral anesthesia or hypoesthesia (loss of pain and temperature) similar to our patient's clinical picture. Anesthesia will normally begin 1-2 segments below the level of lesion, affecting all caudal body areas (1, 3). This is clinically tested by using pinpricks.

Cranial CT has an important role in the imaging of bilateral cerebral hemorrhages, which are fairly rare. In the presented case, hemorrhage was observed in the cranial CT, which was obtained two hours after the appearance of the clinical picture. Although CT displayed an image consistent with HICH in bilateral basal ganglion regions, the clinical course of the patient was good as in PSS. Based on this clinical presentation, we obtained a cranial MRI at the 4th hour in order to help in eliminating other diagnoses such as calcification. MRI revealed an image consistent with hemorrhage during the hyperacute period.

Despite previous cases of bilateral simultaneous hemorrhage that had a poor prognosis, the case presented here with bilateral simultaneous basal ganglion hemorrhage had a good course similar to pure sensorial stroke.

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