Unilateral Basal Ganglia CT Abnormality in Hyperosmolar Hyperglycemic Nonketotic State

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Abstract

Hyperosmolar hyperglycemic nonketotic state (HHNS) is a complication in diabetes mellitus patient that may clinically presents as hemichorea-hemiballism especially in elderly Asian patient. Abnormalities of basal ganglia on CT were being associated with new onset chorea that was most often reported among diabetic patient with HHNS. CT exhibited high attenuation at basal ganglia which appeared similar to that of hemorrhage or calcifications. However CT findings in HHNS were hyperdense lesion at basal ganglia without surrounding mass effect or edema. Involuntary movement in patient of HHNS is treatable and has good prognosis if early diagnosis is established and glycemia is controlled. Imaging either CT or MRI is helpful for the diagnosis.

A 60-year-old Thai man with an underlying DM type II presented with an involuntary movement of the right upper extremity for 3 days. He was clinically diagnosed as having stroke. The NCCT showed homogeneous high attenuation at left caudate and lentiform nuclei. The laboratory tests revealed blood sugar 715 mg/dL, HbA1c 18.62 %, BUN 43 mg/dL, creatinine 1.7 mg/dL, Na 124 mmol/L. The serum osmolarity from calculation was 303.08 mosm/kg. The clinical presentation, CT findings and laboratory results were consistent with HHNS. He was treated with blood sugar control with intensive insulin treatment and hydration. One month later CT showed completely resolved high attenuation at left caudate and lentiform nuclei accompanied by the slow regression of the involuntary movement.

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บทคัดย่อ: Unilateral basal ganglia CT abnormality in hyperosmolar hyperglycemic nonketotic state: รายงานผู้ป่วย 1 ราย

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Hyperosmolar hyperglycemic nonketotic state (HHNS) เป็นภาวะแทรกซ้อนที่พบได้ในผู้ป่วยเบาหวาน อาการแสดงทางคลินิกอาจจะเป็นความเคลื่อนไหวผิดปกติที่ควบคุมไม่ได้ (involuntary movement) อาจพบได้ในผู้ป่วยสูงอายุ โดยเฉพาะในผู้ป่วยแถบเอเชีย ความผิดปกติที่พบในภาพเอกซเรย์คอมพิวเตอร์ของสมอง ในตำแหน่ง basal ganglia มีความสัมพันธ์กับการเคลื่อนไหวที่ผิดปกติซึ่งพบได้บ่อยในผู้ป่วยเบาหวานที่มีภาวะของ HHNS จะให้ลักษณะภาพในเอกซเรย์คอมพิวเตอร์ เป็น high attenuation ที่ basal ganglia ซึ่งอาจทำให้วินิจฉัยเป็น calcifications หรือ hemorrhage ได้ แต่ในภาวะของ HHNS ลักษณะผิดปกติที่เห็นในภาพเอกซเรย์คอมพิวเตอร์ จะไม่พบลักษณะของการกดเบียด หรือการบวมของบริเวณเนื้อสมองรอบ ๆ การเคลื่อนไหวผิดปกติในผู้ป่วย HHNS เป็นภาวะที่รักษาให้หายได้และมีพยากรณ์โรคที่ดี ดังนั้นการให้การวินิจฉัยที่รวดเร็วและควบคุมระดับน้ำตาลในเลือดได้จะส่งผลให้ผู้ป่วยหายได้ การส่งตรวจเอกซเรย์คอมพิวเตอร์จึงมีความสำคัญในการช่วยวินิจฉัยในระยะเริ่มแรกได้

รายงานนี้เป็นผู้ป่วยชายไทย อายุ 60 ปี มีโรคประจำตัวเป็นเบาหวานมาด้วยอาการเคลื่อนไหวผิดปกติของแขนขวามาประมาณ 3 วัน แพทย์สงสัย stroke จึงได้ส่งทำเอกซเรย์คอมพิวเตอร์สมองพบเป็นลักษณะ homogeneous high attenuation at left caudate and lentiform nuclei การตรวจทางห้องปฏิบัติการพบ ระดับน้ำตาลในเลือดสูง 715 mg/dL, HbA1c 18.6, BUN 43 md/dL, creatinine 1.7 mg/dL, Na 124 mmol/L, serum osmolarity จากการคำนวณ คือ 303.1 mosm/kg จากอาการผลตรวจทางห้องปฏิบัติการ และความผิดปกติที่เห็นจากภาพเอกซเรย์คอมพิวเตอร์ น่าจะเข้าได้กับ HHNS ผู้ป่วยรายนี้ได้รับการรักษาด้วยการให้ intensive insulin, สารน้ำ ติดตามอาการและประเมินเอกซเรย์คอมพิวเตอร์ซ้ำอีก 1 เดือนต่อมา พบว่าลักษณะที่เห็นเป็น high attenuation ที่ basal ganglia หายไป และอาการเคลื่อนไหวผิดปกติลดลง

Introduction

Hyperosmolar hyperglycemic nonketotic state (HHNS) is one of serious complications of diabetes mellitus patients that is more commonly found in type 2. The important clinical manifestations include dehydration and loss of consciousness. The localized neurological deficit may also be found such as hemichorea-hemiballism, the spontaneous, involuntary, random, non-rhythmic, uncoordinated and fast jerking motions in distal parts of the limbs which may disappear during sleep, especially in the elderly Asian patients. The patients with sudden onset of chorea may be misdiagnosed as stroke that is much more common than HHNS. In this situation, the abnormalities of basal ganglia on the CT may be helpful to differentiate the diagnosis. The CT in HHNS with chorea usually exhibits the high attenuation at basal ganglia which appears similar to that of hemorrhage or calcifications. However CT finding in HHNS is the hyperdense lesion at basal ganglia without surrounding

mass effect or edema. Involuntary movement in patient of HHNS is treatable and has good prognosis if early diagnosis is established and hyperglycemia is controlled. Herein, a man with acute chorea due to HHNS and the CT findings is studied.

Case report

A 60-year-old male with an underlying diabetes mellitus type II presented with the involuntary movement of the right upper extremity for 3 days. The principal diagnosis of stroke was initially provided by the clinician and the NCCT was performed. The NCCT showed the homogeneous high attenuation at left caudate and lentiform nuclei without the surrounding pressure effect or brain edema. His laboratory tests were: plasma glucose 715 mg/dL, HbA1c 18.6%, BUN 43 mg/dL, creatinine 1.7 mg/dL, Na 124 mmol/L. Because the serum osmolarity and ketone tests were not available in my hospital, the serum osmolarity was calculated from the formula, Osm = (Na x 2)+(Glucose/18)+(BUN/2.8), and found to be 303.08. With the evidence of involun-tary movement, high serum glucose, high serum osmolarity and the NCCT findings of high attenuation of left caudate and lentiform nuclei, it should be properly diagnosed as HHNS. His serum glucose was gradually well controlled by intensive insulin and adequate hydration and then later controlled with oral medication. The CT was repeated a month later and it showed the complete resolution of the high attenuation at left caudate and lentiform nuclei and the involuntary movement was gradually improved.

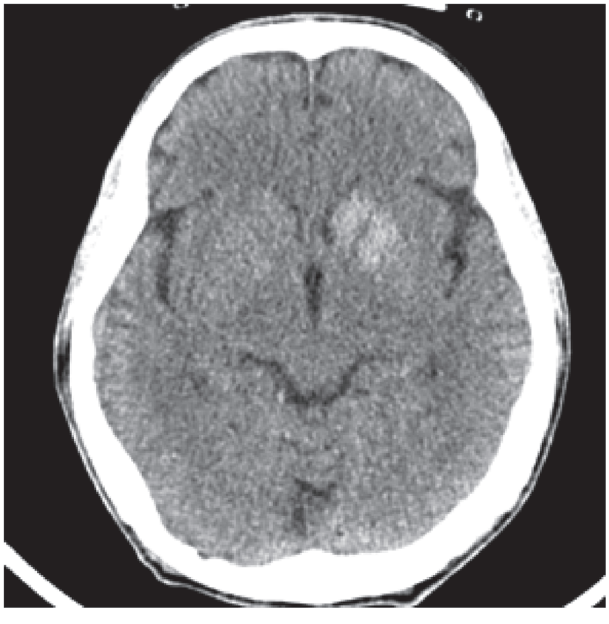

Figure 1. Non contrast MDCT of brain showed homogeneous hyperattenuation at left caudate and lentiform nuclei without perilesional edema or pressure effect to adjacent structures.

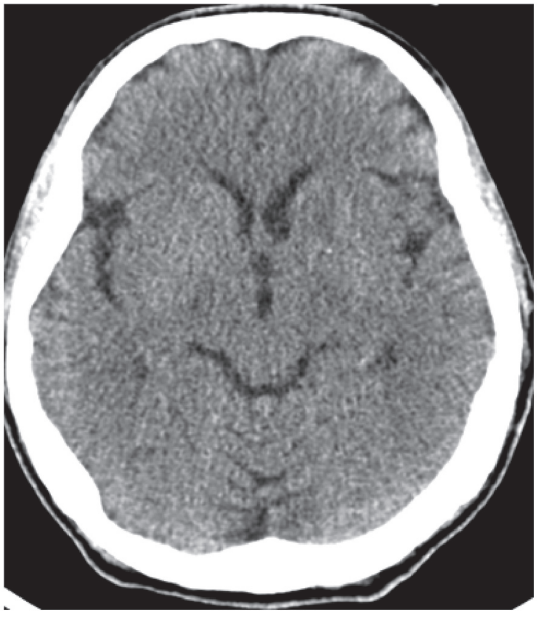

Figure 2. Follow up non contrast CT study a month later showed the resolved area of hyperattenuation on left caudate and lentiform nuclei.

Discussion

HHNS is one of serum metabolic derangements that occur in the patients with DM. It is life threatening emergency but less common than DKA. HHNS most commonly occurs in patients with DM type II, especially in the elderly, and carries a higher mortality rate than DKA. Acute onset of involuntary movement is one of presentations of the hyperglycemic state that may be mimicking stroke. It is important to rapidly make the proper diagnosis for reducing the morbidity and mortality. Laboratory and imaging findings are helpful for its diagnosis.

In emergency setting, HHNS is commonly misdiagnosed as stroke. Imaging findings are helpful by suggesting the correct diagnosis and ensuring the proper treatment. NCCT reveals the hyperattenuation predominantly at the putamen, and less commonly at the caudate nucleus and globus pallidus, contralaterally to the symptomatic side without surrounding edema or mass effect. It may be confused with the calcification or focal hemorrhage. However on follow up NCCT in HHNS, it should reveal the resolution of hyperattenuation lesion. The pathogenesis of imaging findings is still inconclusive, some authors have reported the presence of gliotic tissue with abundant gemistocytes, a form of reactive astrocytes, which can accumulate manganese. The accumulation of manganese in the brain results in the movement disorder and CT imaging findings of hyperattenuation in the basal ganglia.

My case presented with acute onset of the involuntary movement as the localizing neurological deficit so the principle diagnosis was stroke. But the NCCT exhibited the homogeneous attenuation at left caudate and lentiform nuclei without surrounding edema or mass effect. By the evidences of clinical presentation correlating with the NCCT findings suggest HHNS in the differential diagnosis of calcifications.

The laboratory criteria for diagnosis of HHNS may include the serum glucose of 600 mg/dL or more (715 mg/dL in my case), the effective serum osmolarity $\geq$ 320 mosm/kg. However in many studies it can be isoosmolar state in HHS as in our case whose serum osmolarity is 303. Bicarbonate level in HHNS is higher than 15 mmol/L whereas in my case, it is 21.9 mmol/L.

Overall, the clinical setting, laboratory and NCCT findings could be consistent with HHNS. Intensive insulin with adequate hydration was administered immediately and later the serum glucose was controlled with oral medication. Follow up NCCT a month later found the resolution of hyperattenuation at left caudate and lentiform nuclei.

The radiologist should recognize HHNS in the diabetic patients in the setting of acute involuntary movement and unilateral CT abnormality at the basal ganglia because it is treatable as soon as serum glucose is well controlled.

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