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CASE REPORT

Basal Ganglia Hyperdensity on Computerised Tomography of Brain: A Rare Finding of Non-Ketotic Hyperglycemia

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ABSTRAK

Hiperdensiti di ganglia basal dalam pengimejan tomografi (CT) otak selalunya dianggap sebagai strok hemoragik atau kalsifikasi dalam otak. Tanda-tanda hiperglisemia seperti kelemahan otot, hypotonia, gejala trak piramida dan hemichorea-hemiballismus (HC-HB), menyerupai gejala strok. Hiperdensiti di ganglia basal telah dilaporkan dalam pesakit dengan hiperglisemia non-ketotik (NKH). Ketidakupayaan untuk mengenalpasti hiperdensiti ini dalam CT otak sebagai tanda NKH boleh mengakibatkan kegagalan rawatan. Diagnosa awal dan rawatan hiperglisemia yang optimum akan mempercepatkan kadar pemulihan pesakit. Ini adalah kes atipikal gejala menyerupai strok dengan ciri-ciri neuroimaging yang menunjukkan hiperdensiti asimetri ganglia basal. Pemeriksaan neurologi tidak menunjukkan sebarang gejala HC-HB. Ujian makmal menunjukkan hiperglisemia tanpa asidosis dan ketonaemia. Pesakit disyaki mengalami pendarahan ganglia basal dan dirujuk kepada pakar pembedahan saraf dan otak. Walau bagaimanapun, gejala menyerupai strok yang dihidapi pesakit pulih sepenuhnya berikutan pembetulan tahap paras glukosa. Kes NKH ini merupakan kes kedua yang dilaporkan mempunyai ciri-ciri neuroimaging tipikal yang tidak dikaitkan dengan HC-HB. Kesedaran perlu ada terhadap kemungkinan NKH dalam ketiadaan gangguan pergerakan dengan hiperdensiti basal ganglia asimetrik di otak CT. Kesalahtafsiran sebagai pendarahan intrakranial akut boleh mengakibatkan pengurusan suboptimal.

Kata kunci: hiperglisemia, kencing manis, neuroimaging, strok

ABSTRACT

Hyperdensity of basal ganglia in computed tomography (CT) of brain is always

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recognised as hemorrhagic stroke or calcification. Features of hyperglycemia include muscle weakness, hypotonia, pyramidal tract signs and hemichorea-hemiballismus (HC-HB), which mimic the symptoms of stroke. Hyperdensity of the basal ganglia was reported in patient with non-ketotic hyperglycemia (NKH). Inability to recognize the hyperdensity in CT brain as a feature of NKH may lead to failure of treatment. Early recognition and reversal of hyperglycemia will improve the outcome. This was a case of an atypical presentation of stroke-like symptoms with the neuroimaging finding showing asymmetric hyperdensity of basal ganglia. Neurological examination failed to elicit any sign of HC-HB. Laboratory test showed hyperglycemia with absence of acidosis and ketonaemia. The patient was diagnosed having basal ganglia hemorrhage and referred to the Neurosurgical team. However, the stroke-like symptoms completely resolved following the normalization of glucose level. This is the only other reported case of NKH with typical neuroimaging features not associated with HC-HB. One should be aware of the possibility of NKH in the absence of movement disorder with asymmetric basal ganglia hyperdensity in CT brain. Misinterpretation as acute intracranial bleeding may result in suboptimal management of the true underlying cause.

Keyword: diabetes mellitus, hyperglycemia, neuroimaging, stroke

INTRODUCTION

Hyperglycemia is a common clinical presentation in Emergency Department (ED) with features of muscle weakness, hypotonia, pyramidal tract signs, and hemichorea-hemiballismus (HC-HB) (Abe et al. 2009; Bandyopadhyay & Dutta 2005; Choi et al. 2016; Liang & Lin 2013; Lin & Chang 1994; Lin et al. 2001; Mihai et al. 2008; Oh et al. 2002; Patni & Bagga 2017). These clinical features resemble signs and symptoms of stroke and patients would invariably undergo non-contrasted computed tomography (NCCT) of brain to look for the evidence of hemorrhagic or ischemic changes. The most commonly reported and best recognized neuroimaging finding in HC-HB with non-ketotic hyperglycemia (NKH) are asymmetric hyperattenuation involving the lentiform and caudate nuclei (Bhattarai 2016; Heo & Jeong 2017; Lai et al. 1996; Patni & Bagga 2017; Zaitout 2012). These are also well-established neuroimaging findings in patients with NKH including both simple hyperglycemia and hyperglycemia hyperosmolar non-ketotic syndrome (HHNS). The hyperdensity changes in computed tomography (CT) of brain can mimic other pathology including hypertensive hemorrhage (Wilson et al. 2011).

CASE REPORT

A 45-year-old male presented to the Emergency Department (ED) complaining of generalised body weakness, more over the right upper

and lower limbs for the past two days. He was not certain of the exact time of symptoms onset. He complained of instability and frequent falls at home during this period. He also complained of difficulty swallowing with choking episodes while having meals. He denied any fever, blurring of vision, dizziness, palpitation, chest pain or back pain. He had past medical history of diabetes mellitus and hypertension, and was on regular medication for the past five years. Ten weeks prior to the current complaint, he presented with reduced consciousness and chest pain. He was found to have acute ST elevation myocardial infarction (STEMI) Killips IV. He was intubated, ventilated and sent for CT brain which did not report any abnormality (Figure 1a). Patient subsequently was transferred to a cardiac centre for coronary angiography that showed coronary arteries obstruction in three vessels. However, only one stent was placed to relieve the symptoms. Patient was subsequently discharged with ramipril, ticagrelor, cardiprine, atorvastatin, pantoprazole, subcutaneous (SC) actrapid 6 units TDS, and SC insulatard 18 units ON. He claimed that he has been compliant to recent medications.

On examination, Glasgow Coma Scale was 14/15 with E3V5M6 and pupil was equal, round and reactive bilaterally. His initial vital signs were blood pressure of 127/72 mmHg, pulse rate of 86 beat/minute, respiratory rate of 16 breath/minute and temperature of 37°C. The initial random blood glucose level was 31 mmol/L. Neurological examination revealed a reduction of power with grade 4/5 over both upper limbs and lower limbs bilaterally, otherwise normal in tone and reflex. Plantar reflex were equivocal bilaterally. Cranial nerve examination showed loss of right nasolabial fold and weakness of the gag reflex. Cerebellar examination was normal. Other

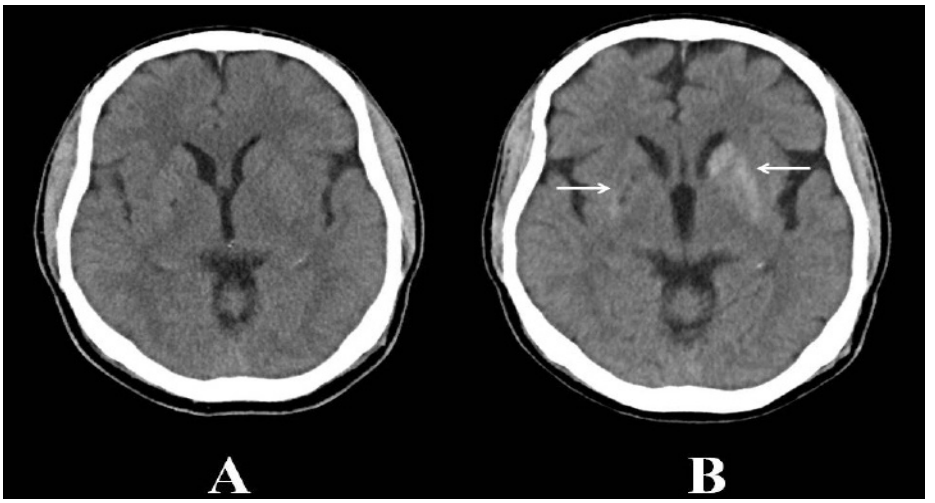

Figure 1: NCCT brain. A) NCCT brain 10 weeks prior to current admission showed no hyperdensity over basal ganglia and head of caudate nucleus. B) NCCT brain at current admission showed hyperdensity of the bilateral head of caudate nucleus and basal ganglia, more over the left side.

systemic examinations were within normal range. CT brain was reported to have new findings of hyperdensity at bilateral basal ganglia and head of caudate nucleus, the left more than the right (Figure 1b). Venous blood gas showed no metabolic acidosis with pH 7.40, HCO_3 22 mmHg, BE 1.1 and the serum osmolality was 304.4 mOsm/kg. Serum ketone was negative. HbA1c was 10.6%. His full blood count, renal profile and C-reactive protein were normal. Chest X-ray was unremarkable. Electrocardiography (ECG) showed sinus rhythm.

The patient was diagnosed with basal ganglia haemorrhage and referred to neurosurgical and medical team. The patient was admitted to neurosurgical ward for further work-up and aggressive glucose control. His symptoms improved with insulin administration and normalization of the glucose level. There was complete resolution of the neurological deficit within 24 hours. The final diagnosis was non-ketotic hyperglycemia secondary to poor compliance to medication. He was discharged well after five days of admission and instructed to follow up at endocrine clinic as outpatient.

DISCUSSION

NKH includes both simple hyperglycemia and HHNS. HC-HB can be a symptom of NKH in primary diabetes mellitus with a classical CT neuroimaging finding of hyperattenuation over unilateral or bilateral of the putamen and/or caudate nucleus (Bhattarai 2016; Heo & Jeong 2017; Lai et al. 1996; Oh et al. 2002; Patni & Bagga 2017; Zaitout 2012). It is a rare condition with only 53 patients reported in the largest published case series between 1985 until 2001. The majority were females and the mean age of onset was 71.1. This is a benign disorder with clinical improvement correlated with reversible basal ganglia lesion (Oh et al. 2002). To date, there are only three published case reports with similar presentations of NKH without HC-HB (Choi et al. 2016; Hansford et al. 2013; Liang & Lin 2013). The typical neuroimaging finding of NKH is hyperattenuation over putamen and/or caudate nucleus with sparing of internal capsule without mass effect or perilesional oedema (Johari et al. 2014; Lin et al. 2001; Wintermark et al. 2004). However, the hyperdensity changes in CT brain can represent other pathologies including acute intracranial bleeding (ICB) (Wilson et al. 2011). Acute ICB appears as hyperdensity on CT scan and the most common cause of the non-traumatic ICB is hypertensive hemorrhage which occurred most commonly in the basal ganglia, pons, thalamus, and cerebellum (Ojemann & Heros 1983). Other differential diagnoses of hyperdensity lesion in basal ganglia include basal ganglia calcification, foreign body, Tay Sachs disease, Wilson's disease, Hallevorden-Spatz disease, tuberous sclerosis, and Sturge-Weber syndrome (Adams 1980; Basak 2009; Kim et al. 2006). When a typical neuroimaging finding of asymmetrical hyperdensity is present, NKH should be the top of the differential diagnoses despite absence of typical presentation of HC-HB. Clinical outcome of NKH is

excellent with institution of appropriate insulin therapy and symptoms would rapidly resolve (Mohd Fazrul & Tan 2018).

CONCLUSION

Patient with NKH may present with stroke-like symptoms at ED. Hyperdensity of caudate nucleus and putamen in CT brain mimic haemorrhagic stroke in NKH. Early recognition of the clinical and radiological features of NKH is important. Complete reversal of hyperglycemia in NKH is able to rapidly resolve symptoms and improve outcome.

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