Case report

Indian Journal of Neurotrauma (IJNT)
2011, Vol. 8, No. 1, pp. 41-42

Post traumatic Klüver-Bucy syndrome : A case report

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Abstract: Klüver-Bucy syndrome [KBS] is a neurobehavioral disorder, less frequently reported in neurotrauma patients. We report a case of post-traumatic Klüver-Bucy syndrome in a young patient who had moderate head injury. Patient had symptoms of hyperorality, hypersexuality, placidity and dietary changes. Patient improved with conservative treatment. Only few cases of post-traumatic KBS are reported in literature. According to available reports this is first case of post-traumatic KBS being reported from India.

Keywords: Klüver-Bucy syndrome; temporal lobe injuries; encephalitis; hypoxic brain damage

INTRODUCTION

Klüver H and Paul Bucy first noted behavioural changes in Rhesus monkeys after analysing temporal lobectomy as early as 1937-39[1,2]. This syndrome complex consists of docility (decreased aggressive behaviour) dietary changes (eating inappropriate objects or over eating), placidity, visual agnosia (inability to recognise familiar objects), hypersexuality (heightened sexual drive), hyperorality (an oral tendency to examine objects by mouth). There may often be aggressive behaviour.

The first case of KBS was reported in human, after bilateral temporal lobe damage due to Herpes simplex meningoencephalitis[3]. Though the clinical manifestations of KBS are similar both in humans and rhesus monkey, few features like amnesia, dementia, dysphasia, seizures are limited to humans[4,5,6]. The syndrome has been described in association with other CNS disorders like Alzheimers disease, juvenile neuronal lipofusciosis, Huntington's disease, toxoplamosis, traumatic brain injury, hypoglycaemia, acute intermittent porphyria, TB meningitis, heat stroke and Shigellosis[7,8,9]. We report a case of KBS in traumatic brain injury patient who had bilateral temporal lobe injuries.

CASE REPORT

A -17-year-old male sustained head injury in a road traffic accident, and was admitted eight hours later. He was unconscious since the time of injury. On examination, he was irritable, with equal and reactive pupils. The Glasgow Coma Scale was E2V3M5 (10/15), and he was moving all limbs. There were no external injuries except abrasion over left temporal region.

CT brain (Fig 1) showed bilateral ill-defined temporal contusions with edema. No other significant lesions noted. He was managed with antibiotics, mannitol, anti-epileptics and other cerebro-protectors. As he recovered, he started exhibiting features of KBS from seventh day onwards in the form of hyperorality (increased oral exploration, tendency to touch objects by mouth], hypersexuality (sexual instincts in the form of stroking sex organs), placidity (becoming quiet when scolded for abnormal behaviour), hypermetamorphosis (strong tendency to react to visual stimulus). Visual agnosia (psychic blindness, inability to recognize familiar objects or known persons) aggressiveness and dietary changes (over eating or any form of objects are being swallowed) too were noted. MRI brain could not be done due to financial constraints. He was given carbamazepine 200mg BD. Three days later, his symptoms were decreased, and he was discharged after three weeks of

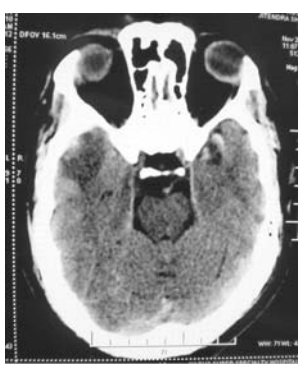

Fig 1: CT SCAN BRAIN; Showing bilateral temporal lobe contusions with surrounding oedema

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hospital stay. On follow up after eight weeks, there was thirty percent improvement in behavioural attitude and other KBS symptoms. Thereafter, he was lost to follow-up.

DISCUSSION

KBS was documented among humans after temporal lobectomy by H Terzian and G D ore in1955[5] and later by Marlowe WB and etal in 1975[3].

Anatomical basis of KBS is due to damage that occurs when both the right and left medial temporal lobe of the brain malfunction. The amygdala has been a particularly implicated brain region in the pathogenesis of this syndrome[4,5,6,9]. KBS also results from disruption of pathways connecting the dorsomedial thalami with pre frontal cortices and other limbic areas which essential for memory and regulation of impulses and emotions[3,6].

Single photon emission CT[SPECT] study by Ozawa etal[10] clearly showed hypoperfusion in the bilateral frontal, parietal and most remarkably, in temporal regions. In our case, patient had bilateral temporal lobe contusions that resolved with conservative treatment. Interestingly appearance of KBS in deeply unconscious patient is considered as good prognostic feature[11]. Asensio and Juan have described a case of KBS after a minor head injury whose CT brain was normal but MRI brain showed few demyelinating plaques in frontal, temporal lobes[12].

The natural history of KBS is not known, but evidence suggests that in trauma,its course is temporary, ranging from seven days to one year[12]. There is no specific treatment except oral Carbamazepine(CBZ)[13]. CBZ and leuprolides have been found to decrease the sexual behavioral abnormality in individuals with KBS. Other medications such as haloperidol and anti-cholinergics may also be useful in treating behavioural abnormalities associated with KBS[6].

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