ABSTRACT

Dementia is not an uncommon presentation in psychiatric practice. Of the various causes of reversible dementia, subdural hygroma is a lesser-known potentially reversible cause. A case of dementia with Kluver-Bucy symptoms secondary to subdural hygroma is described and implications of Kluver-Bucy symptoms in dementia are discussed.

Key words: Subdural hygroma, dementia, Kluver-Bucy syndrome, carbamazepine

Subdural hygromas are fluid collections within the subdural space that are clear or only slightly xanthochromic or blood tinged. They can be caused by mechanical trauma, meningeal or parameningeal infection, or venous congestion. Kluver-Bucy syndrome (KBS) is a rare constellation of neurobehavioural signs and symptoms resulting uncommonly in humans following insult to bilateral temporal lobes. There are six core features of KBS (Poeck, 1985) - hyperorality, hypersexuality, changes in dietary habit, visual agnosia, placidity and hypermetamorphosis. We report a patient who developed dementia consequent to subdural hygroma and presented to us with Kluver-Bucy symptoms. Role of carbamazepine in the treatment of KBS is also discussed.

CASE REPORT

A 45-year-old man was admitted with an eight months history of forgetfulness, wandering tendency, inability to recognize familiar faces, aggressiveness, increased appetite and disturbed sleep. Over a time, his language had deteriorated and presently was limited to a few intelligible words. Moreover, patient now had to be helped with his daily personal activities. Preceding the emergence of these symptoms, patient had a febrile illness accompanied by altered sensorium that lasted five days. He recovered on parenteral and oral medications, the nature of which could not be discerned by the informants as they were uneducated rural people. A lumbar puncture was, however, not performed during that time. No history of trauma to head was reported. Past and family history were unremarkable.

On general examination, patient was fairly nourished but lacked in personal hygiene. Neurological examination revealed no long tract involvement or any other abnormality, other than a mixed expressive and receptive aphasia. Patient could recognize and name objects but would try to pick and use them or would put them into his mouth. Fundus examination was normal. Mental status examination revealed a confused and disoriented adult in partial touch with his surroundings. He was inattentive, distractible by visual or other stimuli and could comprehend only single-word pointing commands. His speech was mostly irrelevant and incomprehensible with marked perseveration. Moreover, he was unable to recognize familiar faces even after repeated exposure. His inattention and poor comprehension made him psychologically non-testable.

The routine blood investigations were normal. EEG showed non-specific abnormality.
in the form of poorly formed alpha (7.5-8 Hz, 15-30 microvolts) limited to posterior part of brain mixed with indeterminate activity, low amplitude fast activity and irregular theta activity. Occasionally, alpha activity showed hypersynchrony. CT scan of brain showed crescentric extraaxial areas of CSF density over fronto-temporo-parietal lobes bilaterally with mild dilatation of ventricular system. An impression of bilateral subdural hygroma was entertained.

The patient was treated on inpatient basis and commenced on thioriadazine (100 mg, once daily) and carbamazepine (gradually hiked to 600 mg/day, in divided doses). Following these medications, his increased oral tendency, wandering tendency and aggressiveness decreased. Patient was discharged and referred for neurosurgical intervention but unfortunately did not return for follow-up.

DISCUSSION

The diagnosis of subdural hygroma was based on radiological findings on CT scan and clinical picture. Contrast administration did not reveal any enhancing neomembranes which made chronic subdural haematoma less likely (Osborn, 1994). Moreover, a progressive downhill course without any fluctuations in consciousness is not typical of the latter condition. Subdural hygroma in this patient was most probably consequent to a parameningeal inflammatory pathology as suggested by the nature of the preceding illness. Symptoms of dementia (forgetfulness, impaired language, declining social behaviour and executive functioning) emerged after this illness and can be attributed to subdural hygroma.

The above presentation was accompanied by some striking symptoms reminiscent of the classic KBS. These symptoms included increased oral tendency (hyperphagia, oral exploration), prosopagnosia (a type of visual agnosia characterized by inability to recognize familiar faces), hypermetamorphosis (strong tendency to attend to and react to visual stimuli) and changes in dietary habits. Placidity and hypersexuality, the other features of KBS were absent which conforms with the earlier finding of overt hypersexuality being uncommon in human KBS (Lily et al., 1983). Furthermore, diffuse brain involvement in human KBS may cause features of dysfunction of other lobes to mask some of the Kluver-Bucy symptoms (Duggal et al., in press). Nevertheless, the patient had KBS considering the convention of labelling only those cases as complete or incomplete KBS which have the chief manifestation of increased oral tendency (Poeck et al., 1985). Another noticeable phenomenology was the patient's seizing and using things presented to him that was akin to 'utilization behaviour' seen in frontal lobe dysfunction (Lhermitte, 1983). This has been recognized as a sort of executive function defect (Tranel et al., 1994) and impairment in executive functioning is a feature of dementia. Although dementia has been associated with emergent Kluver-Bucy symptoms (Burns, 1990), subdural hygroma per se leading to dementia with KBS has not been documented in literature. A possible mechanism for such a presentation could be similar to chronic subdural haematoma presenting as dementia (Black, 1984). Human KBS has largely been described as irreversible but its partial response to carbamazepine has been cited in literature (Hooshmand et al., 1974). They ascribed this function to carbamazepine being a limbic anticonvulsant with a potent inhibitory action on amygdaloid kindling. This observation along with the evidence that KBS appears related to partial deafferentation of excitatory projections to amygdala (Kling et al., 1993) underscore the role of carbamazepine in treating KBS. This was true in this patient too whose behavioral problems did partly remit with carbamazepine. What remained to be seen was whether surgery would have ameliorated these and the other symptoms of dementia.

In conclusion, this case illustrates subdural hygroma presenting as dementia and the significance of Kluver-Bucy (KB) symptoms in
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Dementia. Patients with dementia usually seek psychiatric services and the clinician should explore for an underlying organic aetiology especially in the scenario of emergent KB symptoms and utilization behaviour. Prompt neuroimaging in such cases can benefit patients with a potentially reversible cause of dementia. Furthermore, a trial of carbamazepine may help patients of dementia with KBS.

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