Kluver–Bucy Syndrome in an Adolescent Girl: A Sequel of Encephalitis

Dear Editor,

Kluver–Bucy syndrome is a relatively rare phenomenon, which results from the involvement of bilateral temporal lobe, especially hippocampus and amygdala.[1] It is characterized by hyperorality, hypermetamorphosis (excessive exploration of the environment), altered sexual, feeding, and emotional behavior, and visual agnosia.[2] During childhood and adolescent, the manifestations differ. Many patients may have only few features of the syndrome and are described as “partial Kluver–Bucy syndrome.”[2] In pediatric population, it is infrequent and mostly results as a complication of herpes simplex encephalitis.[3] We present here the case of an adolescent female, who developed features of Kluver–Bucy syndrome following encephalitis.

An 11-year-old girl was referred for psychiatric consultation with a 2-year history of hyperactivity, impulsivity, hyperorality, excessive exploration of the environment (hypermetamorphosis), lack of fear inhibition, difficulty in speech (mostly in expression of speech), episodes of rage, inattentiveness, forgetfulness, and increased sexuality. Her sleep and appetite were normal. She had history of hospitalization for fever and altered sensorium, urinary and fecal incontinence, and rigidity in all limbs 6 months before the onset of these symptoms. But, no history of seizure was reported. Magnetic resonance imaging (MRI) of the brain during hospitalization revealed T2 signal hyperintensity in bilateral basal ganglia, thalamus, brain stem, and adjacent periventricular areas [Figure 1A–C]. Cerebrospinal fluid (CSF) examination revealed pleocytosis and elevated protein. CSF examination for herpes encephalitis, Japanese encephalitis, cytomegalovirus, Epstein–Barr, and dengue virus was negative. Thyroid profile, parathyroid hormone, serum ammonia, and Thyroid peroxidase antibody levels were normal. Electroencephalogram was normal. However, considering the clinical presentation and brain areas affected, possibility of Japanese encephalitis and dengue encephalitis was considered. Negative CSF vireology was probably because of her presenting 3 weeks after the onset of illness to the hospital. She was managed conservatively. She had marked rigidity and dystonic posturing of limbs and trunk, leading to a bed-bound stage. Her current behavioral symptoms became apparent after the rigidity improved with clonazepam and anticholinergic medication (trihexyphenidyl and tetrabenazine) over 6 months. Given the patient’s history of encephalitis, the MRI findings, and the current psychiatric presentation, she was given a diagnosis of Kluver–Bucy syndrome (a sequel of encephalitis). The patient had received atomoxetine up to 40 mg/day and because of partial response of her hyperactivity symptoms, methylphenidate was added, which later was increased up to 30 mg/day (1 mg/kg body weight). Her hyperactivity and inattention improved significantly. Her episodic aggression (rage) and hypersexuality also reduced after addition of olanzapine (5 mg/day). After over 6 months of follow-up, a significant reduction in her hyperactivity, inattention, hyperorality, hypersexuality,

![Figure 1](https://example.com/figure1.jpg)  
Figure 1: (A–C): MRI of brain: T2-weighted images (indicated by black arrows in 1A: Sagittal section; 1B: Transverse section; 1C: Coronal section) showing signal hyperintensities in bilateral basal ganglia, thalamus, insular cortex and brain stem
and episodes of rage and hypermetamorphosis was observed.

Kluver–Bucy syndrome often results from the involvement of bilateral medial temporal lobe and is rarely encountered in children and adolescents or may be in attenuated form in this age group. Viruses (herpes simplex virus) that have specific propensity to involve the temporal lobes may produce symptoms of Kluver–Bucy syndrome.

Hyperactivity accompanied with inattention (features of attention deficit hyperkinetic disorder) are less common because of Kluver–Bucy syndrome. Our patient had the aforementioned features, which were inadequately controlled with atomoxetine. However, add on methylphenidate reduced the impulsive behavior as well as hyperactivity. Evidences suggest that involvement of ventromedian nucleus of thalamus and amygdala might result in generation of rage. The rage episodes in our patient can be explained on the basis of diffuse involvement of bilateral thalamus and medial temporal lobe. Addition of antipsychotic drug (olanzapine) was useful in reducing the rage behavior.

Mood stabilizers, antidepressants (selective serotonin reuptake inhibitors), and antipsychotic drugs are found to be effective in the management of Kluver–Bucy syndrome. The clinicians need to be familiar with such rare entities, which might be seen following encephalitis. The viruses (herpes simplex and Japanese encephalitis virus) that have specific propensity to involve the limbic system (especially medial temporal lobe and amygdala) are likely to develop features of Kluver–Bucy syndrome. Collaboration between the neurologist and psychiatrist is highly essential for effective management of such cases.

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Conflicts of interest
There are no conflicts of interest.

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