CASE REPORT

Partial Kluver-Bucy syndrome secondary to tubercular meningitis

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SUMMARY

Tubercular meningitis (TBM) is a devastating extra pulmonary manifestation of tuberculosis and demonstrates a high neurological morbidity. A rare complication of this condition is Kluver-Bucy syndrome (KBS), which is a neurobehavioral disorder characterised by hyper-sexuality, visual agnosia, bulimia, placidity, hyperorality and memory deficits caused by lesions to the amygdala. The amygdala lesions can be due to many causes, including traumatic brain injury, systemic conditions and infections such as tuberculosis. Here, we present a case of partial KBS in a patient undergoing treatment for TBM.

BACKGROUND

Tuberculosis (TB) is a major public health issue with global distribution. The disease progression is insidious in nature and, if not caught and treated early enough, can lead to extra-pulmonary manifestations. One such manifestation is tubercular meningitis (TBM), which accounts for nearly 1% of all cases of TB.¹

The neurological morbidity and mortality that TBM bears are at a disturbingly elevated level.¹ Epidemiological studies on extra pulmonary TB in the USA have demonstrated central nervous system (CNS) involvement in up to 10% of cases.² The 2011 CDC data indicate that 5.7% of extra pulmonary TB cases have TB involving the meninges.³ Studies demonstrate various neurological complications that develop from advanced progression of TBM, such as hemiparesis, aphasia, cranial nerve palsies, visual abnormalities, seizures, gait ataxia, hearing abnormalities and others.^{1-2 4}

One rare complication of TBM is Kluver-Bucy syndrome (KBS), a neurobehavioral disorder resulting from lesions to the amygdala. This syndrome complex consists of docility, dietary changes, placidity, visual agnosia, hyper sexuality, hyperorality.⁵⁻⁸ There may often be aggressive behaviour.⁵ KBS is less frequently reported as a sequel to TBM and is a rare form of CNS involvement in TB.⁹⁻¹¹

We present a case of partial KBS in a patient undergoing treatment for TBM.

CASE PRESENTATION

A 22-year-old male of Indian ethnicity was brought to the hospital after he was assaulted by a mob for following a female to her home without her consent. His parents reported that they noticed some behavioural changes in him for past 2 months, including hyperphagia, increased inclination for the opposite sex, stalking females,

following females in his neighbourhood to their homes, frequent fiddling with his genitals, biting young children aged 2-3 years at home, aggressive behaviour, memory changes and docility when confronted regarding the incident.

His parents further reported that, 13 months ago, the patient had a symptom of low-grade fever for 1 month. They went to multiple physicians but treatment did not result in any relief to the patient's symptoms. After 4 months of various investigations and management plans, a doctor finally prescribed prophylactic antitubercular drugs. This led to a significant improvement in symptoms over the next 15 days and medication was then stopped. After 1 month of treatment discontinuation, the patient reported low-grade fever, vomiting and altered consciousness, and afterward admitted to the intensive care unit. The investigation report stated that the complete blood count (CBC), liver function tests (LFT), renal function tests (RFT) and HIV test were negative for any abnormalities. The cerebrospinal fluid (CSF) showed a protein level of 200 mg/dL, a glucose level of 44 mg/dL, a cell count of 270/cm³ with 98% lymphocytes and an adenosine deaminase level of 19 units/L. MRI studies demonstrated ventricular enlargement, mild decompensated hydrocephalus and basal ganglia and left medial temporal lobe infarcts along with areas of haemorrhage. On the basis of his MRI and CSF analysis reports, he was diagnosed with TBM. Several medications, including antitubercular drugs, intravenous steroids and mannitol, were started. By the fourth week of admission, the patient demonstrated significant clinical improvement and was finally discharged. On discharge he was placed on a maintenance dose of antitubercular drugs (rifampicin and isoniazid). However, for the past 2 months, his parents noted the behavioural changes described above.

INVESTIGATIONS

Current CBC, Vitamin B12 and folic acid, LFT, RFT, HIV and venereal disease research laboratory test results are within normal limits. A recent MRI with contrast demonstrates gliosis involving bilateral basal ganglia, with more on the left and adjacent basi-fronto-temporal lobes (figure 1).

TREATMENT

Apart from his antitubercular drugs he was started on risperidone 1 mg, and this dose was gradually titrated to 2 mg over the span of a 5-day period. This led to a significant improvement in the patient's aggressive behaviour and sexual symptoms

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Figure 1 Gliosis involving bilateral basal ganglia, with more on the left and adjacent basi-fronto-temporal lobes.

within 2 months and gradually risperidone was stopped within 3 months. He is currently in regular follow-up in our outpatient clinic for the past 2 months.

DISCUSSION

TBM originates from a primary pulmonary TB infection, obtained either from inhalation or from the ingestion of infected dairy products.⁴ The infection spreads to the CNS via lymphatic and haematogenous routes and a Rich focus is formed in the meninges or brain parenchyma.⁴ This Rich focus remains dormant and reactivates after a variable period of time if the patient is immunocompromised or elderly.⁴ In early stages, TBM manifests constitutional symptoms such as fever, malaise, headache, anorexia and recent weight loss.^{2 4} As the disease progresses, the patient can present with altered mental status, meningeal signs and disseminated features such as hemiparesis, visual abnormalities, seizures, cranial nerve palsies and hearing abnormalities.^{1-2 4} There are some other unusual manifestations of TBM, such as generalised myoclonus, rigidity, gait ataxia and KBS, which may lead to misdiagnosis and therefore delayed detection, and cause significant morbidity and permanent impairment.^{1-2 4}

One of the unusual manifestations that can lead to misdiagnosis is KBS, which is a rare neurobehavioral syndrome. In 1984, Aichner, after studying 53 patients with KBS, described the following symptoms: increased oral activity, hypersexuality, memory deficits, bulimia, placidity, inability to recognise people, and hypermetamorphosis.² To diagnose a patient with partial KBS, a combination of three or more different elements in this list must be present.²

There are multiple non-specific aetiologies of KBS, including post-traumatic brain injury, encephalitis, meningitis and systemic disease.⁵⁻¹³ Unilateral temporal lobe injury has been found to also cause KBS.² Structural and functional dysfunction of the temporal lobe may cause KBS; it may also result from the disruption of any pathway connecting the dorsomedial thalamus to prefrontal cortex and/or other limbic areas.^{6 10}

Although there is no specific treatment for KBS, it has been found that antipsychotic medications can lead to a significant

improvement in abnormal sexual behaviour.² Other medications such as carbamazepine and leuprolide have also been used.^{10 12-13} We treated the patient with risperidone 2 mg, which demonstrated a marked improvement in behaviour on his second outpatient visit, 6 weeks after initiation of the treatment. He is currently still in follow-up.

Learning points

- ▶ Early diagnosis and opportune treatment of tubercular meningitis is of utmost importance in preventing the considerable mortality and morbidity associated with the disease.
- ▶ Kluver-Bucy syndrome (KBS) is one of the rare neurological complications of tuberculosis.
- ▶ Although the specific mechanism of action is unknown, antipsychotics and carbamazepine can be used to treat KBS with variable success.

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Contributors KKJ assembled the case history and investigations from hospital records, analysed the data and wrote the paper. SKS and PK selected the case, assessed the patient data and critically reviewed the paper. CDA helped with editing and reviewing the case report.

Competing interests None declared.

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REFERENCES

- 1 Cherian A, Thomas SV. Central nervous system tuberculosis. *Afr Health Sci* 2011;11:116–27.
- 2 Rieder HL, Snider DE Jr, Cauthen GM. Extrapulmonary tuberculosis in the United States. *Am Rev Respir Dis* 1990;141:347–51.
- 3 Centers for Disease Control and Prevention. Atlanta, GA, USA: Centers for Disease Control and Prevention, 2012 (cited 08 March 2016); [first screen]. <http://www.cdc.gov/tb/statistics/reports/2011/table27.htm>
- 4 Komolafe MA, Sunmonu TA, Esan OA. Tuberculous meningitis presenting with unusual clinical features in Nigerians: two case reports. *Cases J* 2008;1:180.
- 5 Bhat PS, Pardal PK, Das RC. Partial Kluver-Bucy syndrome as a delayed manifestation of head injury. *Ind Psychiatry J* 2009;18:117–8.
- 6 Marlowe WB, Mancall EL, Thomas JJ. Complete Klüver-Bucy syndrome in man. *Cortex* 1975;11:53–9.
- 7 Park HK, Kim KJ, Moon HJ, et al. Klüver-Bucy syndrome with isolated bilateral hippocampal atrophy following status epilepticus. *J Epilepsy Res* 2012;2:10–2.
- 8 Deginal A, Changty S. Post traumatic Klüver-Bucy syndrome: a case report. *Indian J Neurotrauma* 2011;8:41–2.
- 9 Thirunavukarasu S. Temporal and pontine involvement in a case of herpes simplex encephalitis, presenting as klüver bucy syndrome—a case report. *J Clin Imaging Sci* 2011;1:43.
- 10 Jha S, Patel R. Klüver-Bucy syndrome—an experience with six cases. *Neurol India* 2004;52:369–71.
- 11 Shraberg D, Weisberg L. Klüver-Bucy syndrome in man. *J Nerv Ment Dis* 1978;166:130–4.
- 12 Morcos N, Guirgis H. A case of acute-onset partial Kluver-Bucy syndrome in a patient with a history of traumatic brain injury. *J Neuropsychiatry Clin Neurosci* 2014;26:E10–1.
- 13 Samanta D. Transient Kluver Bucy syndrome from methamphetamine withdrawal. *Neurol India* 2015;63:267–8.

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