British Journal of Medicine & Medical Research
20(3): 1-5, 2017; Article no.BJMMR.31747
ISSN: 2231-0614, NLM ID: 101570965

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Acute Psychosis as Clinical Manifestation of Tuberculous Meningitis

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Authors' contributions

This work was carried out in collaboration between all authors. Author DP wrote the first draft of the manuscript. Authors MLP and RM prepared final version of case report. Author RS managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/BJMMR/2017/31747
Editor(s):
(1) Georgios A. Androutsopoulos, Department of Obstetrics-Gynecology, School of Medicine, University of Patras, Rion, Greece.
Reviewers:
(1) Gulnur Tarhan, Adıyaman University, Turkey.
(2) Kolade-Yunusa Hadijat Oluseyi, University of Abuja and University of Abuja Teaching Hospital, Nigeria.
(3) Guadalupe García-Elorriaga, Mexican Social Security Institute, Mexico.
Complete Peer review History: http://www.sciencedomain.org/review-history/18147

Case Report

Received 23rd January 2017
Accepted 2nd March 2017
Published 10th March 2017

ABSTRACT

A young female was admitted with complaint of low grade fever, loss of appetite and fluctuating behaviour from the past two week. There was no history of any seizures, loss of consciousness, tuberculosis or any psychiatric illness. On examination the patient was apathetic, drowsy and her Glasgow Coma Scale was 13/15(E4V3M6). The patient was bed ridden and most of the time she use to sleep with occasional interruptions by episodes of agitation. No bladder bowel involvement was present. Motor system examination revealed grade 4/5 power in all four limbs with normal muscle tone and bilateral plantar flexor response. There was no signs of meningeal irritation (neck rigidity, Kernig's and Brudzinski's sign).
All routine investigation including CSF and CT scan was normal. On clinical ground she was diagnosed as a case of acute psychosis and kept on olanzapine. Initially she responded to drug, but again she was admitted due to headache, nausea and instability of gait. Cerebrospinal fluid examination was suggestive of tuberculous meningitis. MRI of brain showed infarct in right cerebellar hemisphere. The patient was treated with anti-tubercular drugs and steroids. On follow-up visit one month later, her psychotic symptoms had fully resolved. She was able to ambulate and care for herself.

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Keywords: Acute psychosis; tuberculous meningitis.

1. CASE REPORT

A 25 year old married female was admitted in December 2016 with complaint of low grade fever, loss of appetite from 10-15 days and fluctuating behaviour from the past 8 to 10 days. The family members also reported that she had lethargy, irrelevant talking, forgetfulness and slowness in motor and verbal response. For the above mentioned problem, the patient consulted a nearby community health centre where she was treated on empirical basis as a case of enteric fever. The patient symptoms did not improve and she was brought to our hospital by the family members. There was no history of any seizures, loss of consciousness, psychiatric illness or past history of tuberculosis or any family member having history of tuberculosis.

On examination the patient was apathetic, drowsy and Glasgow Coma Scale was 13/15 (E4V3M6). The patient was bed ridden and most of the time sleeping or lay quiet with occasional interruptions by episodes of agitation and screaming for which patient had to be given sedatives. At times she would wake up by her own and could walk with the help of a family member's support. The patient had a voluntary control on urination and defecation. Power in all four limbs was 4/5. There was no clonus, muscle tone was normal and plantar response was bilateral flexor. The patient was afebrile, she had no signs of meningeal irritations (neck rigidity, Kernig's and Brudzinski's sign not present), no visual blurring or diplopia.

2. INVESTIGATIONS

Blood investigations revealed a normal haemogram, normal serum electrolytes, a normal liver function test (LFT), and kidney function test (KFT). Elisa test for HIV, HbsAg, HCV was negative. IgM test for dengue, typhoid and smear for malaria parasite was also negative. Initial CT scan of head was normal (Fig. 1). Cerebrospinal fluid (CSF) cytological examination revealed a total cell count of 10 with 90% lymphocytes, protein was 52 mg/dl, sugar was 40 mg/dl, AFB staining was negative and ADA was <10 u/L and CSF virology was negative for Herpes simplex, Japanese encephalitis and Enterovirus. PCR and culture for mycobacterium in CSF were not performed as clinical presentation and CSF cytology was not suggestive of TBM.

She remained afebrile during hospital stay, hence a psychiatry opinion was taken in which she was suspected to be a case of acute psychosis. She was prescribed tablet olanzapine 5mg at bedtime. On conservative management the patient's symptoms improved and she was discharged after 6 days on tablet olanzapine 5 mg at bedtime and was advised for follow up.

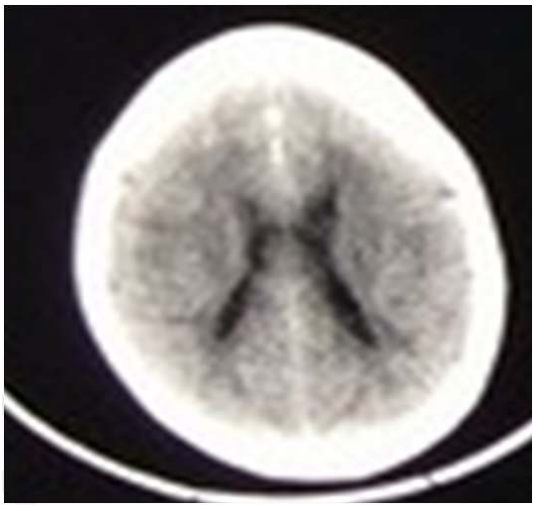

Fig. 1. CT scan of head of the patient at first visit showing a normal CT

3. FOLLOW UP

After 10 days follow up, she visited in outpatient department with complaints of severe headache, on-off altered behaviour, difficulty in walking and vomiting from past 3-5 days. She was looking ill, emaciated and lethargic. On examination the patient was afebrile, drowsy with positive neck rigidity and Kernig's sign. The patient was admitted again and CT head was performed. CT report was suggestive of prominent lateral ventricles with opened temporal horns (Fig. 2). A guarded lumbar puncture was performed for CSF analysis which revealed lymphyocytic pleocytosis (TLC-80/mm^3, DLC-Lymphocyte 90%, Neutrophil 10%) with elevated protein (177 mg%), decreased sugar(19 mg%) and Adenosine deaminase value was 13.7u/L. India ink for Cryptococcus neoformans was negative. Cartridge Based Nucleic Acid Amplification Test (CBNAAT) in CSF was positive. CSF culture for mycobacterium was done on Löwenstein Jensen media, which came out positive after 4 weeks.

Routine blood investigations revealed a normal hemogram, normal KFT and LFT. In electrolytes low level of sodium (128 mmol/L) was present and ESR was 30 mm/hr.

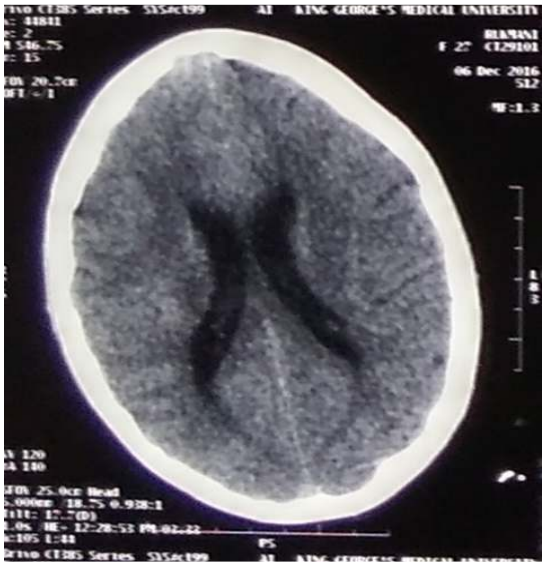

Fig. 2. CT scan head of the patient on first follow up visit showing prominent lateral ventricles with opened temporal horns

MRI brain was done on 6th day of admission. It revealed showing hyperintensity in right cerebellar hemisphere suggestive of acute infarct (Figs. 3, 4a, 4b and 4c). The patient was started treatment with intravenous dexamethasone 0.4 mg/kg/day, mannitol 1.5 gram/kg stat followed by 500 mg/kg 8 hourly and anti-tubercular drugs (Isoniazid 5mg/kg/day, Rifampicin 10mg/kg/day, Pyrazinamide 25 mg/kg/day, Ethambutol 15 mg/kg/day) according to her body weight. The patient's symptoms resolved gradually over 3-4 weeks after starting of treatment. She is now on continuous follow up in the OPD.CSF culture on Löwenstein Jensen media for mycobacterium came out to be positive after 4 weeks.

4. DISCUSSION

Tuberculosis continues to be a major global as well as a national health concern. According to WHO Tuberculosis was the fifth major cause of death worldwide in 2012. In the WHO 2016 world TB report the estimated TB incidence rate in India is between 200-300/lacs with mortality rates between 20-39/lac population [1]. Extra pulmonary Tuberculosis(EPTB) is crucial to diagnose and treat promptly as if left untreated it can result in significant morbidity and mortality. CNS manifestations of TB include meningitis (TBM), cerebral and spinal tuberculoma, myelitis and arachnoiditis. Tuberculous meningitis can occur in isolation or along with pulmonary tuberculosis.

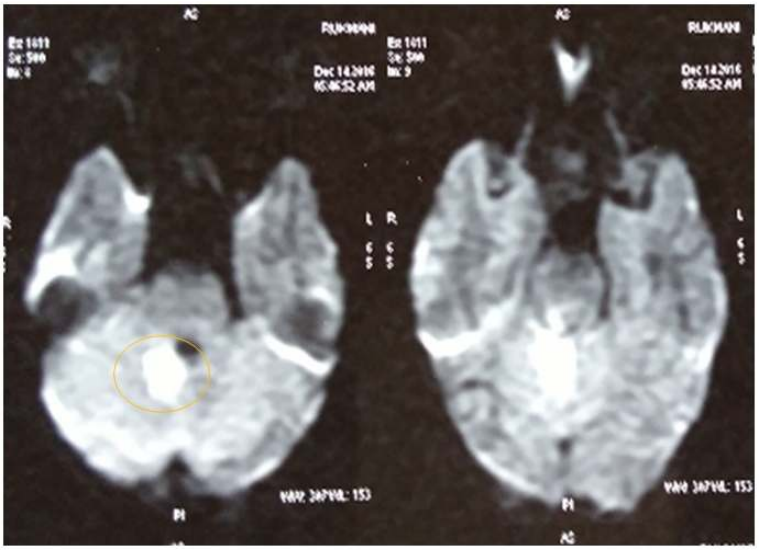

Fig. 3. Diffusion weighted image showing hyperintensity in right cerebellar hemisphere suggestive of acute infarct

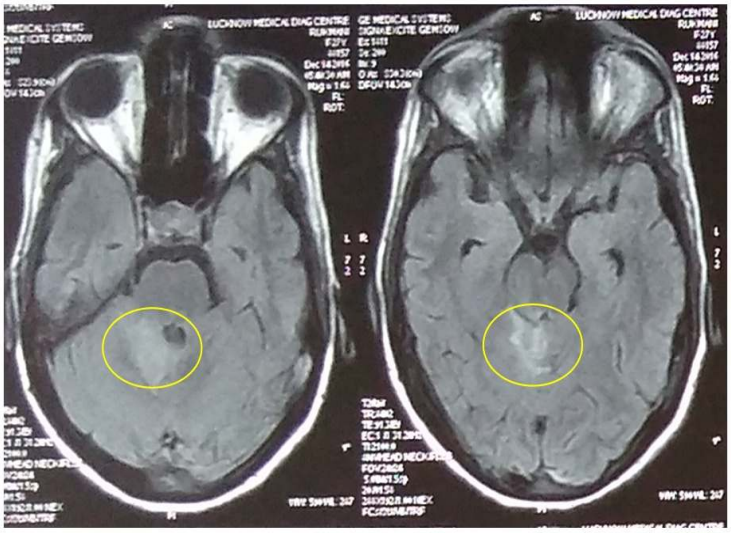

Fig. 4(a). Right cerebellum

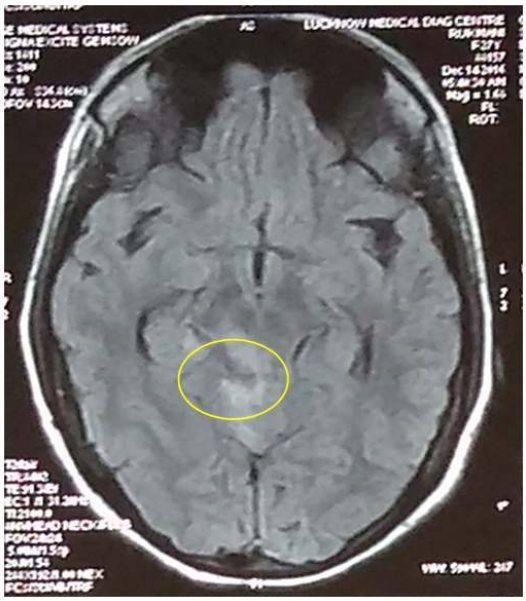

Fig. 4(b). Midbrain

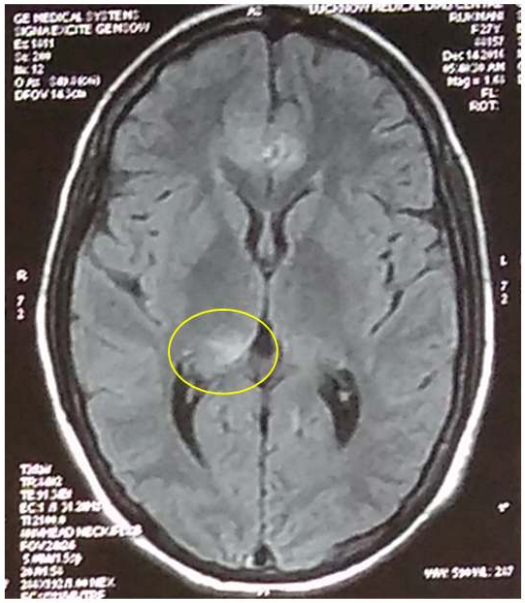

Fig. 4(c). Right thalamus

Fig. 4. (a, b, c). T2 flair MRI image showing signal intensities at different levels of brain

TBM is important as it has myriad of manifestations and any delay in diagnosis and treatment could lead to permanent neurological deficit and even death. Major risk factors being HIV positive status, diabetes mellitus, younger age and malnutrition [2]. TBM should be presumed in a patient with clinical features of meningitis in the form of fever, headache, neck rigidity and vomiting, with or without altered sensorium and associated focal neurological deficits for a period of 5 days or more [3]. Common clinical manifestations include fever, anorexia, headache and vomiting. Uncommon and rare features include cranial nerve palsy (3rd and 6thcommon), hemiparesis, coma, seizures, pyschosis, internuclear ophthalmoplegia and photophobia [3,4].

Psychosis has been defined as the inability or impaired ability to distinguish reality from

hallucinations and/or delusions. The symptoms may include a change in behaviour, mood, thinking and perception. Our patient's symptoms began in the form of psychosis with mixed positive and negative symptoms without any fever, headache or other evidence of meningitis. TBM was not suspected initially due to absence of fever and any signs of meningitis, supported by a normal CT scan of head and a normal CSF picture. The patient's general condition improved and discharged on antipsychotic medication. She did well for about 10-12 days. She again developed abnormal behaviour, headache and vomiting for which she was admitted again and was eventually diagnosed as a case of TBM based on clinical findings, CSF and imaging studies. Current guideline recommendation for diagnosis of TBM in India include CSF evidence of lymphocytic pleocytosis with elevated protein and low sugar, plus imaging with CT head/MRI brain and a clinical background suggestive of TBM. PCR is not routinely recommended but could be used as an adjunct [3].

Psychosis as an initial symptom of TBM was also reported by RMJ Che et al in a 19 year old female, in which she had symptoms of psychosis for about 1 year before she was diagnosed with TBM [5]. In another case report a 30 year old female was diagnosed with TBM and SIADH [6]. She also had psychotic symptoms since 2 months. In our patient the symptoms started about one month prior to diagnosis of TBM. These findings suggests that a varied time course of psychotic symptoms may be present in TBM.

5. CONCLUSION

In a country like India with an immense TB burden this case report highlights the importance of keeping TBM as a differential in a patient presenting with psychosis with no prior history of any psychiatric illness and also acknowledging that TBM can be present even when there is no fever and signs of meningitis. An early consultation, diagnosis and prompt initiation of treatment in our case led to recovery of symptoms and averted development of any complication.

CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case report and the images.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history:
The peer review history for this paper can be accessed here:
http://sciencedomain.org/review-history/18147