CASE REPORT

CHRONIC SUBDURAL HAEMATOMA PRESENTING AS LATE ONSET PSYCHOSIS

Thingbaijam Bihari Singh*, Athokpam Ranita Devi*, Senjam Gojendra Singh*, Mhetre Bhushan Bhagwan*

***Department of Psychiatry, Regional Institute of Medical Sciences (RIMS) Imphal Manipur, PIN- 795004, India.**

Abstract

Objective: **This case report highlights a case of late onset psychosis which was an uncommon occurrence to psychiatric services in developing countries. Medical causes of late onset psychosis, though known, are often missed. Chronic subdural haematoma (CSDH) is predominantly a disease of the elderly. A history of direct trauma to the head is usually absent.** ***Methods:*** **A previously healthy 80-year-old male presented with 4 months duration of late onset psychosis.** ***Result:*** **Neurological examination was normal. Routine investigations were within normal limits and MRI brain revealed a chronic subdural haematoma. Owing to his age and small size of the haematoma, patient was not operated on and showed improvement with the pharmacologic treatment for psychosis.** ***Conclusion:*** **Detailed longitudinal history, clinical examination, investigations along with high index of suspicion is necessary to effectively manage this condition. As CSDH is known as a great imitator and is usually a disease of the elderly, it should be kept in mind while dealing with cases of late onset psychosis.** ***ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 175-178.***

Keywords: Late Onset Psychosis, Chronic Subdural Haematoma

Introduction

Chronic subdural haematoma(CSDH) is predominantly a disease of the elderly. It is an encapsulated collection of old blood, mostly or totally liquefied, and located between the dura and arachnoid mater [1]. Generalized cerebral atrophy and increased venous fragility associated with aging are the major predisposing factors. With aging, the mass of the brain decreases leading to an increase in the space between the brain and the skull from 6% to 11% of the total intracranial space. This causes stretching of the bridging veins and the greater movement of the brain within the cranium makes these veins vulnerable to trauma [2, 3].

Interestingly, falls have been reported to be a very common presenting symptom (74%) in a recent prospective study involving 43 elderly patients [4]. Also in a retrospective study of a series of patients with subdural haematoma, fall was the most frequently encountered (57%) risk factor. It is noteworthy that in 10% of the cases, no risk factors could be identified [5]. However, a history of head injury (direct trauma) is absent in about 30%–50% of the cases; indirect trauma seems to be more important. About half the patients have a history of fall but without hitting their head on the ground [6, 7]. In many situations, the trauma is so trivial that it is forgotten. Other predisposing factors include anticoagulation, alcoholism, epilepsy, bleeding diathesis, low intracranial pressure secondary to

dehydration or after the removal of cerebrospinal fluid, and receiving renal dialysis, presumably due to platelet dysfunction [3]. As many as 24% of patients with CSDH are on warfarin or an antiplatelet drug, [4] 5%–10% have a history of alcoholism and epilepsy [1].

There are limited data for CSDH presenting as late onset psychosis. The interest in this case lies in the manifestations of late onset psychotic behaviour in a patient with an underlying chronic subdural haematoma.

Case report

An 80-year-old male from a rural background was treated by a private psychiatrist for seven days of irrelevant talk, suspiciousness, smiling and muttering to himself, irritability, wandering tendency, sleep disturbances, and decreased appetite in the month of October 2011. He was treated with neuroleptic medications details of which were not available. However, medications were discontinued since January 2012 as he was feeling well.

Again in the second week of February 2012, he gradually developed sleep disturbance and frequently remained awake at night. He started talking irrelevantly and excessively than before. He was fearful of everyone near him and became anxious, apprehensive and resistive. Later, he started abusing people and accusing them of doing black magic on him. He tried to go out of the house many times and family members had to stop him. His agitated behaviour remained the same and wandering tendency could not be controlled. His oral intake was irregular and reduced than usual. In view of all these, he was admitted in the Psychiatric ward, Regional Institute of Medical Sciences (RIMS) Hospital and was thoroughly evaluated.

Patient had history of alcohol dependence during his young age but had completely abstained from alcohol for more than 2 years. There was no past history of major head trauma, diabetes, hypertension, or use of anticoagulant or antiplatelet drugs.

Mental status examination revealed a thin old man in appropriate attire, was irritable and uncooperative, answering irrelevantly most of the time with poor rapport and eye contact. His attention could be easily distracted and he was very restless to go out of the room. He was oriented to time, place, and person. His short term memory was impaired as shown by digit span test, and his judgment and insight was also poor. Systemic examination was normal. Neurological examination showed no evidence of lateralizing signs. Muscle tone, power and tendon reflexes were normal.

During the initial few days, patient was irritable and agitated and had wandering tendency so he was given injectable olanzapine (10 mg i.m.) once at bedtime and sodium valproate 250 mg at bedtime along with clonazepam 0.5 mg once at bedtime. On day five of admission, parenteral drugs were stopped as irritability and psychomotor agitation completely subsided, but oral olanzapine (5 mg), sodium valproate (500 mg in divided doses), and clonazepam (0.5 mg) were continued. ECG, thyroid profile, and laboratory tests, including blood chemistry (electrolytes and urea) and urinalysis were within normal limits. A MRI Brain was requested on day seven of admission in view of the unusual presentation and revealed thin fluid signal intensity, overlying the left fronto-parietal and occipital convexity suggestive of chronic subdural haematoma. (See Figure 1)

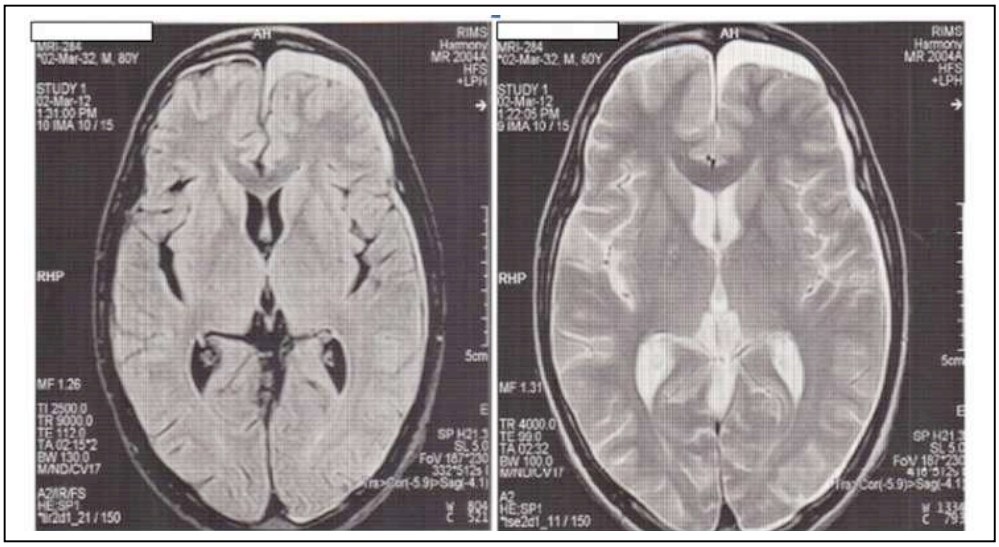

Figure 1. Axial T1 (left) and T2 (right) weighted images from an MRI scan of the brain showing CSDH over the left fronto-parietal-occipital region

Following the outcome of the MRI, a neurosurgical consultation was requested. As there was no indication for surgery, conservative management was continued. Moreover the absence of any lateralizing signs suggested that intracranial compensatory mechanisms were likely occurring. He was regularly followed up for six months and was symptom-free. Subsequent MRI brain was normal.

Discussion

CSDH should be differentiated from acute subdural haematoma. Acute subdural haematomas generally occur in younger adults, after a major trauma, often associated with structural brain injury, and present within 72 hours. In contrast, CSDHs often occur in the elderly after a trivial injury without any damage to the underlying brain, and usually there is a period of weeks to months before it becomes clinically evident. It has a peak incidence in the sixth decade of life. Fogelholm and Waltimo estimated an incidence of 1.72/100 000 per year, the incidence increasing steeply with advancing age up to 7.35/ 100 000 per year in the age group 70–79[8]. This incidence is expected to rise further due to the continuing growth of the older population [1].

Chronic subdural haematoma has been called the great imitator [9] and the initial head trauma may go unnoticed in more than one-third of patients [10]. When the presenting features are psychotic symptoms without any neurological deficits, a chronic subdural haematoma is likely to escape detection. In this elderly patient with no prior history of psychiatric illness, an organic basis of his psychotic symptoms was considered.

CT brain was not done owing to patient's age, non-acute mental status changes and specificity of MRI over CT in chronic brain pathology. We suspect that this patient might have sustained a head injury at the time of one of the trivial falls which might have passed unnoticed and led to development of CSDH.

Conclusion

Psychotic symptoms in the elderly patient may be a manifestation of psychiatric, medical or medication-induced illness. This case highlights that CSDH may cause late onset psychosis. Hence, clinicians should maintain a heightened vigilance in assessment of psychosis in the elderly in view of multiple interacting factors between underlying organicity and psychiatric symptoms.

References

1. Adhiyaman V, Asghar M, Ganeshram KN, Bhowmick BK. Chronic subdural haematoma in the elderly. Postgrad Med J 2002; 78:71–75.

2. Ellis GL. Subdural haematoma in the elderly. Emerg Med Clin North Am 1990; 8:281–94.

3. Traynelis VC. Chronic subdural haematoma in the elderly. Clin Geriatr Med 1991; 7:583–98.

4. Jones S, Kafetz K. A prospective study of chronic subdural haematomas in elderly patients. Age Ageing 1999; 28:519-21.

5. Asghar M, Adhiyaman V, Greenway M W. Chronic subdural haematoma in the elderly –a North Wales experience. J R Soc Med 2002; 95:290-292.

6. Feldman RG, Pincus JH, McEntee WJ. Cerebrovascular accident or subdural fluid collection? Arch Intern Med 1963; 112:966–76.

7. Rozzelle CJ, Wofford JL, Branch CL. Predictors of hospital mortality in older patients with subdural haematoma. J Am Geriatr Soc 1995; 43:240–4.

8. Fogelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. Acta Neurochir 1975; 32:247–50.

9. Potter JF, Fruin AH. Chronic subdural haematoma-"the great imitator". Geriatrics 1977; 32:61-6.

10. Cameron MM. Chronic subdural haematoma a review of 114 cases. J Neurol Neurosurg psychiatry 1978; 41:834-9.

Corresponding author: ***Athokpam Ranita Devi, Department of Psychiatry, Regional Institute of Medical Sciences (RIMS), Imphal*** **PIN- 795004** ***India.***

Email: ranitaathokpam@gmail.com

Received: 1 February 2013 Accepted: 14 March 2013