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CASE REPORT

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Recurrent Psychotic Depression Associated with Chronic Subdural Haematoma: A Case Report

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ABSTRACT

Chronic subdural hematomas (CSDH) with isolated psychiatric presentation are rare. In this paper, we report a case of 77-year-old-female patient who had psychotic depression after repetitive head trauma without any neurological symptoms. The brain magnetic resonance imaging revealed an 20 mm subdural hematoma in the right frontoparietal region and a 7 mm subdural hematoma in the left frontal region. The psychiatric symptoms improved within the first week after evacuation but relapsed with the occurrence of right sided pneumocephalus. In the follow up, with the disappearance of the pneumocephalus, the psychiatric symptoms improved. It should be kept in mind that isolated psychiatric symptoms can be seen due to subdural hematoma and evacuation of the hematoma has an important role in improving the psychiatric symptoms.

Keywords: Depression, pneumocephalus, psychosis, subdural hematoma

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INTRODUCTION

Subdural haematoma (SDH) is the accumulation of blood in the subdural space between the dura mater and the arachnoid mater surrounding the brain (1). Depending on the time elapsed from the onset of bleeding, SDH is classified as acute, subacute and chronic. Chronic SDH (CSDH) lasts longer than three weeks (2). Depressive disorders (3–5) and psychotic disorders (2,5,6) have been reported to develop secondary to CSDH. Rarely, psychiatric symptoms may be the sole clinical manifestation of CSDH. A case of an 82-year-old male (5), who had depressive symptoms and paranoid delusions unaccompanied by a neurological disorder, due to CSDH in the left frontoparietal region, was reported in the literature. There was another case with bilateral SDH and psychotic disorder without any neurological symptoms (2). It may be more difficult to diagnose cases without neurological symptoms, and clinicians should be aware of the fact that CSDH may present with isolated psychiatric symptoms (7). There is no treatment recommendation with a high level of evidence or indication criteria for surgery in cases of CSDH with isolated psychiatric symptoms.

However, data can be provided to studies with a higher level of evidence thanks to the reporting of these rare cases. Thus, guidelines can be developed to determine the optimal management of these patients. In this article, we present a case of psychotic depression secondary to CSDH without neurological symptoms and unresponsive to pharmacotherapy.

Highlights

- **Recently emerging psychiatric symptoms have a challenging role in elderly patients.**
- **Psychiatric symptoms may be the sole manifestation of CSDH.**
- **Surgical treatment may be necessary even in CSDH with sole psychiatric symptoms.**

CASE

A 77-year-old female patient presented to our outpatient clinic with complaints of depressive mood, suicidal thoughts, insomnia, and behavioral changes. Her first psychiatric complaints started at the age of 70, as insomnia, fatigue and inability to run the household chores. She was diagnosed with major depressive disorder and treatment of sertraline 50 mg/d was initiated. Her complaints were completely resolved. She had a recurrence of depressive symptoms at the age of 72 while maintaining the drug, but no psychotic symptoms or impairment of cognitive functioning were detected. For the next three years, the symptoms continued with varying severity. Her family members reported that over time occasional agitative behaviors, such as hitting her head on the ground when her wishes were not fulfilled immediately, or attacking her husband with her cane were added to her complaints. She then developed persecutory delusions in the way of thinking that her kins were trying to harm her; hypochondriac and nihilistic delusions thinking that she could not have urinated and defecated

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for at least one year due to the fact that viruses completely destroyed her digestive system. She was admitted to our outpatient clinic, and risperidone 1 mg/d was added to treatments of escitalopram 10 mg/d and mirtazapine 30 mg/d which she had been using for the last year with the diagnosis of psychotic depression. Neuropsychological testing (NPT), magnetic resonance imaging (MRI) and computerized brain tomography (CT) were performed. Her minimental state examination (MMSE) test score was 19 (cutoff: 23) and modified mini mental state examination/3MS was 55 (z score=-0.33). Visual-verbal test score was 4, semantic fluency test score was 9 and alternating semantic test score was 8 (respectively, z score: 0.33; -3.34; -1.57). Number sequence-forward score was 4 (0–1 SD), total number sequence score was 4 (<-2 SD). Based on the cognitive test battery performed, her attention and memory were moderately, executive functions were mildly impaired, while other cognitive domains were normal. On MRI, chronic subdural hematoma up to 2 cm at its thickest part in the right frontoparietal region and 0.7 cm in the left frontal region was detected. The lateral ventricles were slightly enlarged, and there was a 5 mm midline shift. We did not observe significant volume loss in the hippocampus, in the pons and other brainstem regions (Figure 1). Due to the lack of increased intracranial pressure and the focal neurological signs, surgical intervention was not deemed to be necessary in neurosurgery consultation. Shortly after, she attempted suicide by hanging and was admitted to another clinic where quetiapine 75 mg/d was added to treatments of escitalopram and mirtazapine. After 3 months with escitalopram, mirtazapine and quetiapine, she was hospitalized due to her ongoing suicidal thoughts. At the time of admission, she was severely depressed and persecutory, somatic and nihilistic delusions were present. Quetiapine was increased to 100 mg/d. Repeated Brain MRI revealed that chronic subdural hematoma encircled the entire right cerebral hemisphere and was 1.8 cm (at its thickest part) and did not regress. During follow-up, risperidone 0.5 mg/day was started again and quetiapine was discontinued. Her ongoing agitation, depressive symptoms and psychotic symptoms were thought to be associated with the CSDH. The hematoma was evacuated by burr-hole operation. At the second day of surgery, her delusions completely disappeared as well as her affective complaints. On the third day, her somatic and nihilistic delusions recurred. In addition, a depersonalization delusion, in which she believed that someone else's limbs had been surgically replaced with her own, appeared. The CT scan revealed a pneumocephalus in the right frontal region, which caused a mild midline shift, with no signs of increased intracranial pressure or any other focal neurological deficit (Figure 2). With oxygen therapy and head elevation, pneumocephalus resolved on the seventh postoperative day. Simultaneously with the disappearance of pneumocephalus, psychiatric symptoms improved. The patient was discharged with mirtazapine 30 mg/d, escitalopram 10 mg/d and risperidone 0.5 mg/d without any prominent symptoms. While no recurrence was observed in the psychiatric symptoms during the first month after the patient's discharge, depressive symptoms and delusions reappeared on the 47th day of the operation. Re-accumulation of a 14 millimeters (mm) (at its thickest part) subdural hematoma as a result of leakage of neomembranes that could not be cleared was observed in the right frontal region on the new CT. She had no signs of neurological deficit or increased intracranial pressure. The hematoma in the right frontal region regressed to a thickness of 7 mm on CT in the 3rd month after the operation. Her psychotic and depressive symptoms also regressed (The relationship between patient's intracranial region events and Positive and Negative Syndrome Scale [PANSS] and Hamilton Depression Rating Scale [HAM-D] scores is shown in Figure 3). No change was detected in the NPT, which was repeated after approximately 1 year later. Treatment with escitalopram 10 mg/day, mirtazapine 30 mg/day and risperidone 1 mg/day was continued.

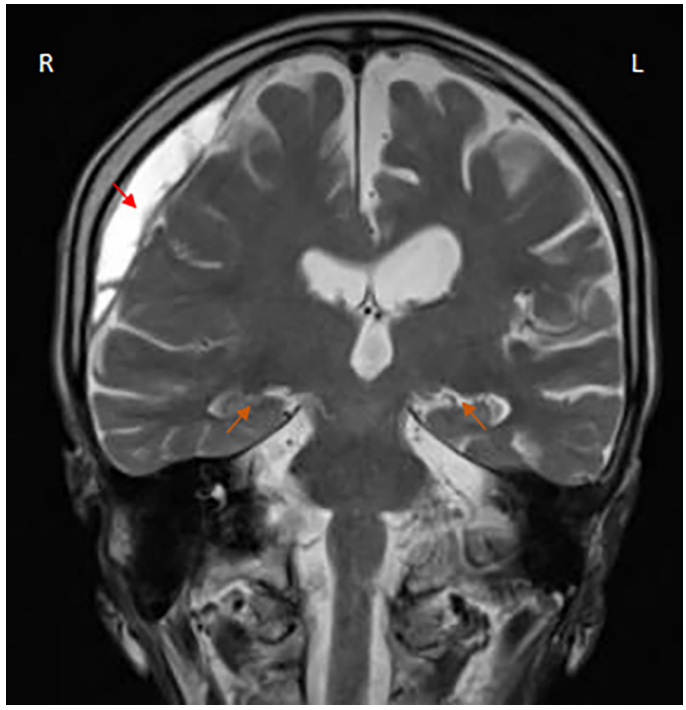

Figure 1. Brain MRI performed in November 2020 shows subdural hematoma (red arrow) in the right frontoparietal region and hippocampus was compatible with age (MTA score: 2) (orange arrows) on T2W-coronal section (13).

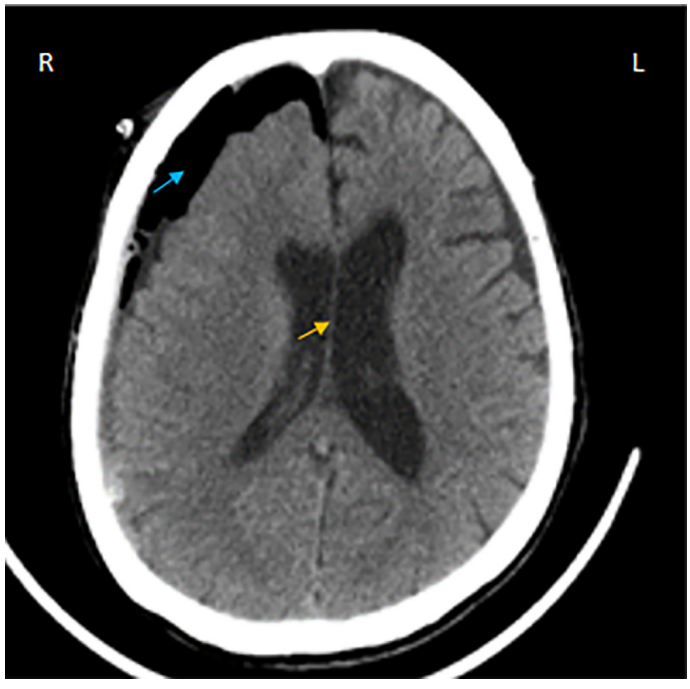

Figure 2. It is shown that pneumocephalus (blue arrow) in the right frontal region and mild midline shift (yellow arrow) is observed on the 2nd postoperative day.

DISCUSSION

In this article, we present a case in which depressive and psychotic symptoms were thought to be associated with CSDH and pneumocephalus. Symptoms improved with the operation but recurred with postoperative accumulation of blood and air.

There are very few case reports of psychosis secondary to chronic subdural haematoma (2,5,6). Elie et al. reported an 82-year-old male

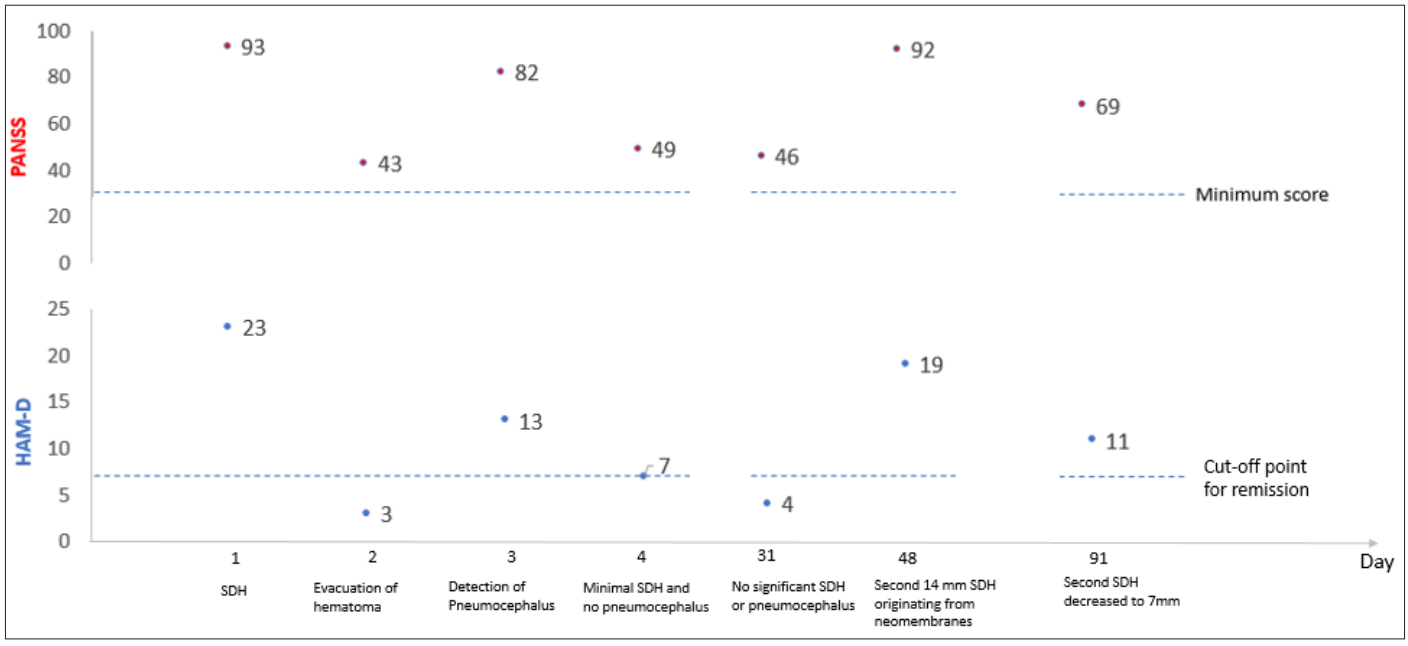

Figure 3. The relationship between patient's intracranial regional events and PANSS/HAM-D scores is shown.
HAM-D: Hamilton, PANSS: Positive and Negative Syndrome Scale

patient with left frontoparietal chronic subdural hematoma presenting with depression and paranoid delusions in the absence of any neurological deficit. In that case, psychotic symptoms slowly improved after evacuation of the hematoma, while pharmacological treatment was required for depressive symptoms (5). Kar et al. reported a 40-year-old male homeless patient with the bilateral subdural hematoma presenting with social withdrawal, disorganized behavior, apathy and poverty of speech without any neurological disorder (2). Despite the development of pneumocephalus, unlike our case, psychiatric symptoms did not worsen with pneumocephalus.

Her psychotic symptoms and the exacerbation of depression were temporally related to the subdural hematoma and pneumocephalus. Behavioral changes accompanying psychotic and depressive symptoms can be explained by the presence of a chronic subdural hematoma in the frontoparietal region. Persecutory, nihilistic and somatic delusions can be observed in frontal lobe lesions, particularly due to the lesions in the right dorsolateral prefrontal cortex (8). Delusions in psychoses secondary to neurodegenerative dementias are associated with corticostriatal network dysfunctions of frontal lobe origin. In our case, the right sided hematoma and pneumocephalus might have caused delusions by compressing the right frontoparietal cortex and also associated subcortical pathways. Chronic subdural hematomas in bilateral frontal regions were also associated with persistent depressive symptoms (9). In fact, confusion and delirium are the most frequent neuropsychiatric problems associated with CSDH (10). Nevertheless, we did not observe any changes in the mental status of the patient during the CSDH and the treatment period. The cognitive problems were limited to the attention, memory and executive functions and did not fluctuate during the course.

The patient's psychiatric symptoms initially appeared as a depressive episode after family related stressfull life events. Later, anxiety and agitation followed depressive symptoms which resulted in repeated self-harm behaviors. As a result of the repetitive head trauma, a subdural hematoma in right frontoparietal as well as left frontal regions occurred. Subsequently, psychotic symptoms developed. These psychotic symptoms, behavioural changes and exacerbation of depression, which developed as a result of compression of the right hemisphere by blood or air in the subdural space, have a temporally dynamic relationship. As can be seen in Figure 3, PANSS score decreased by more than 50% within 24 hours after evacuation of the haematoma, which corresponds to remission for psychotic disorders. Besides, the HAM-D score decreased below 7, which is the cut-off point for remission in depression. After surgical evacuation of the haematoma, psychotic and depressive symptoms and behavioural problems reappeared within 24 hours due to the air accumulation and recompression in the frontal region as a complication of the burr-hole procedure (PANSS: 82, HAM-D: 13). A 1.5 month period was achieved by completely removing the blood and air compressing the frontal region. However, fibrin and capillary-rich neomembranes, that are not completely cleared by craniectomy, have the potential to rebleed (11). In addition, enlarged subdural distance due to chronic haematoma, presence of septated neomembranes, and shift in midline structures were predisposing factors for recurrence of hematoma (11). Due to the advanced age and general health status of the patient, craniectomy procedure, which can provide clearance of neomembranes, was considered risky by the neurosurgery department. Approximately 1.5 months after the burr-hole operation, there was an accumulation of 14 mm haematoma after haemorrhage in the form of leakage of the existing neomembranes and the symptoms recured rapidly (PANSS: 92, HAM-D: 19). The patient was cautiously followed up with psychopharmacotherapy. Since the hematoma and symptoms regressed spontaneously in the course (PANSS: 69, HAM-D: 11) and there was no risk of suicide, craniectomy was not performed and follow-up with pharmacotherapy was continued. No specific treatment is recommended for CSDH patients presenting with psychiatric symptoms in the absence of overt neurological symptoms. Surgical treatments are generally performed in patients with marked neurological symptoms (12). In patients with minimal symptoms, nonoperative treatments may be preferred or spontaneous regression may occur (13). However, considering the fact that our patient had a history of suicide attempt during the course of CSDH, no change in symptoms despite pharmacotherapy and the temporal relationship between psychiatric symptoms and CSDH, burr-hole method was performed surgically. The dramatic improvement of psychiatric symptoms with evacuation of the haematoma, sudden relapse with the development of pneumocephalus, and the restoration of remission with the disappearance of pneumocephalus support our hypothesis. The case also coincides with previous case reports indicating that psychiatric symptoms in CSDH improved shortly after evacuation of

the haematoma (2–7). Similarly, in an article dated 1945, it was reported that psychotic depression developed twice in the same patient due to recurrence of SDH and haematoma had to be evacuated twice (6). In our case, the blood accumulated in the subdural space for the second time and spontaneously regressed to a level that did not put on risk the patient's health condition.

This case with psychotic depression required the evaluation of dementia independent of CSDH due to some features. The first psychiatric symptom reported in this patient was at 70 years of age. Although late life depression may be a prodrome or an associated symptom in neurodegenerative dementias, we were not able to definitively diagnose dementia disease in our patient. On one hand, there are deficits in memory and executive functioning. In addition, the patient never returned to her baseline functioning even with a significant improvement in her psychiatric symptoms. On the other hand, the course of the cognitive symptoms was stable or very slowly progressing as we did not observe a significant cognitive decline in our follow-up for 18 months. In addition, the current MRI at the age of 77 showed an MTA score of 2 (which may be compatible with her age) (14) and only a mild atrophy at the parietal regions (Figure-1). Furthermore, even though depression is relatively common in the mild stages of Alzheimer's Disease (AD), psychosis is more frequently observed in moderate to moderately severe patients (15). Also, the nature of the delusions is not compatible with psychosis related to AD. The delusions were nihilistic and persecutory in nature in our patient congruent to her mood as opposed to the delusions associated with the idea of theft and misidentification commonly observed in AD (16). There was no hallucination of any sensory modality in our patient. All the findings suggest that the psychiatric symptoms of the case may be better explained by CSDH rather than a neurodementing process, although the age, gender, education and neurocognitive status of the patient are particularly favourable for Alzheimer's disease.

In conclusion, psychiatric conditions secondary to CSDH without any obvious neurological symptoms may not be as rare as it seems. Neuroimaging should be used in elderly patients with a history of mild head injury and psychiatric symptoms, even if the clinical condition is not accompanied by neurological symptoms. The presence of another underlying diseases should be kept in mind especially in elderly individuals with psychiatric symptoms that change their characteristics. Subdural hematoma, which may develop secondary to head trauma as well as spontaneously, should be considered in the differential diagnosis and it should be investigated with neuroimaging methods. Isolated CSDH patients without overt neurological symptoms should be followed cautiously, and surgical options should be considered without wasting time, without seeking any other criteria, in case of non-response to pharmacotherapy, in the presence of suicide risk, or when there is a risk of harming himself or someone else due to psychotic symptoms. The further studies with higher levels of evidence, that will guide psychiatrists who treat mental symptoms and neurosurgeons who undertake surgical treatment in the ideal management and follow-up of CSDH cases with isolated psychiatric symptoms, are needed.

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REFERENCES

1. Sahyouni R, Goshtasbi K, Mahmoodi A, Tran DK, Chen JW. Chronic subdural hematoma: a historical and clinical perspective. World Neurosurg. 2017;108:948–953. [Crossref]
2. Kar SK, Kumar D, Singh P, Upadhyay PK. Psychiatric manifestation of chronic subdural hematoma: the unfolding of mystery in a homeless patient. Indian J Psychol Med. 2015;37(2):239–242. [Crossref]
3. Alarcon RD, Thweatt RW. A case of subdural hematoma mimicking severe depression with conversion-like symptoms. Am J Psychiatry. 1983;140(10):1360–1361. [Crossref]
4. Nagatomo I, Ueyama K, Fukuzako H, Matsumoto K. Three cases of chronic subdural hematoma with depressive state. Jpn J Psychiatry Neurol. 1990;44(4):703–707. [Crossref]
5. Elie M, Primeau F, Cole MG. Chronic subdural hematoma in the elderly: a case report. J Geriatr Psychiatry Neurol. 1996;9(2):100–101. [Crossref]
6. Fleiss, NA. Psychiatric manifestations of chronic subdural hematoma, 1945. Available at: https://link springer.com/content/pdf/10.1007/BF01562207.pdf (18 Haziran 2023).
7. Sato T, Takeichi M. A case of chronic subdural hematoma with anxiety states and concomitant regression-like symptoms. Jpn J Psychiatry Neurol. 1987;41(4):663–667. [Crossref]
8. Devinsky O. Delusional misidentifications and duplications: right brain lesions, left brain delusions. Neurology. 2009;72(1):80–87. [Crossref]
9. Reeves SJ, Gould RL, Powell JF, Howard RJ. Origins of delusions in Alzheimer's disease. Neurosci Biobehav Rev. 2012;36(10):2274–2287. [Crossref]
10. Black DW. Mental changes resulting from subdural haematoma. Br J Psychiatry. 1984;145:200–203. [Crossref]
11. Kung W-M, Lin M-S. CT-based quantitative analysis for pathological features associated with postoperative recurrence and potential application upon artificial intelligence: a narrative review with a focus on chronic subdural hematomas. Mol Imaging. 2020;19:1536012120914773. [Crossref]
12. Soleman J, Nocera F, Mariani L. The conservative and pharmacological management of chronic subdural haematoma. Swiss Med Wkly. 2017;147:w14398. [Crossref]
13. Mehta V, Harward SC, Sankey EW, Nayar G, Codd PJ. Evidence based diagnosis and management of chronic subdural hematoma: a review of the literature. J Clin Neurosci. 2018;50:7–15. [Crossref]
14. Loreto F, Gontsarova A, Scott G, Patel N, Win Z, Carswell C, et al. Visual atrophy rating scales and amyloid PET status in an Alzheimer's disease clinical cohort. Ann Clin Transl Neurol. 2023;10(4):619–631. [Crossref]
15. Aarsland D. Epidemiology and pathophysiology of dementia-related psychosis. J Clin Psychiatry. 2020;81(5). [Crossref]
16. Pearce D, Gould RL, Roughley M, Reynolds G, Ward EV, Bhome R, et al. Paranoid and misidentification subtypes of psychosis in dementia. Neurosci Biobehav Rev. 2022;134:104529. [Crossref]