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Chronic subdural haematoma mimicking extrapyramidal symptoms

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SUMMARY

A male patient in his 70s with chronic schizophrenia, who could previously walk independently, developed a gait disturbance without any significant neurological deficit. Initially, his short step length and unstable gait were thought to be related to extrapyramidal symptoms caused by medication side effects. We tapered his antipsychotic medication, but the unstable gait persisted. After 2 weeks of observation, we noted general weakness with left-side dominance, leading us to consider a focal brain lesion despite there being no recent history of falling or trauma. A CT scan of the brain showed chronic subdural haematoma and the patient underwent emergency surgery. After 14 days of treatment, he was discharged back to the chronic ward.

BACKGROUND

Extrapyramidal symptoms (EPS) are one of the most common adverse effects observed in patients taking antipsychotics. Also referred to as drug-induced movement disorders, the symptoms of EPS include dystonia, akathisia and Parkinsonism. In addition, prolonged use of antipsychotics can cause tardive dyskinesia. Drug-induced EPS results from antagonism of dopamine D2 receptors, one of the main actions of antipsychotics, especially in first-generation drugs such as haloperidol and fluphenazine. Although blocking of D2 receptors in the mesolimbic pathway can improve psychotic symptoms, blockage of the same receptors in the nigrostriatal pathway may result in the unwanted effects of EPS.¹ When the blockage becomes long term, overexpression of downstream D2 receptors may occur to compensate for the lack of dopamine, which manifests clinically as tardive dyskinesia.² Treatment usually includes discontinuing the current antipsychotics, or prescribing anticholinergics such as biperiden or trihexyphenidyl. EPS can be very damaging to the patient's quality of life and is one of the major reasons for patients dropping out of pharmacotherapy, which can directly increase the risk of relapse and rehospitalisation. Dealing with EPS is a significant part of clinical practice and a familiar scenario in the psychiatry field. Medication is not the only cause of EPS, however. Organic brain injuries,³ for instance, may mimic such symptoms. Because of the potentially lethal nature of brain injuries, timely and correct identification of the cause of symptoms is crucial. Reported here is a case of chronic subdural haematoma (SDH) that manifested as EPS, which was initially approached as drug-induced EPS.

CASE PRESENTATION

A male patient in his 70s with chronic schizophrenia, the onset of which occurred in his early 20s, had been in long-term hospital care in Taiwan for over 40 years. He had been taking olanzapine 5 mg and valproic acid 400 mg daily for about 3 years and was transferred to the acute ward due to violent behaviour towards another patient, which suggested exacerbation of symptoms. After his admission to the acute ward, we continued to administer his current psychiatric medication. He had a relatively stable mood, could cooperate with ward activity and could walk independently with no violent behaviour noted. However, he had a short step length with unstable gait—the typical manifestation of an extrapyramidal gait disturbance—and leaned forward while walking. He started using a wheelchair due to his gait disturbance about 1 week after his transfer to the ward. No seizures, dizziness, headache or recent falling episode was noted on the ward. Three weeks after being transferred, his laboratory results showed normal levels of serum sodium, potassium, chloride, calcium, creatinine, blood urea nitrogen, aspartate aminotransferase and alanine aminotransferase, as well as normal red and white cell counts. We considered psychiatric medication side effects related to EPS and thus tapered and then withheld his psychiatric medication. However, the patient still had general weakness after we withheld his psychiatric medication for 7 days. He became bedridden due to general weakness. We observed no improvement in EPS after olanzapine was completely discontinued, and so we consulted a neurologist for evaluation. Neurological examination showed decreased muscle power

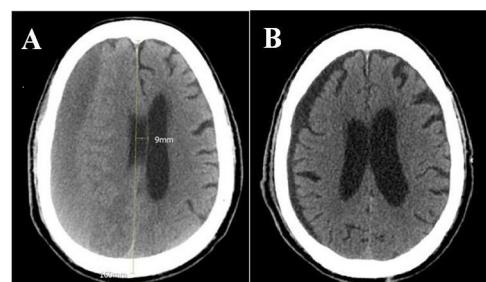


Figure 1 (A) Brain CT scan before surgical intervention. The initial CT before surgery revealed chronic subdural haematoma at right frontal, temporal and parietal region with midline shift about 9 mm. (B) Brain CT scan after surgical intervention. The postoperative brain CT revealed minimal subdural effusion without midline shift was noted. The trephination surgery relieved mass effect due to chronic subdural haematoma.



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of the left limbs, a positive left Babinski sign and hyperactive knee jerk, triceps reflex and brachioradialis reflex on the left side. Thus, a focal brain lesion in the right hemisphere was suspected. The patient was transferred to a general hospital for further evaluation. A CT scan of his brain showed chronic SDH in the right frontal, temporal and parietal lobes (figure 1A). He was then transferred to a medical centre for surgical intervention by means of trephination of the chronic SDH in the right parietal region. A follow-up CT scan of the brain revealed only minimal subdural effusion without a midline shift (figure 1B). The drainage tube was removed, following which the patient's limb weakness improved. After his surgical stitches were removed, the patient was discharged and transferred back to the hospital in stable condition. He is currently in the chronic ward with clear consciousness and improved muscle power.

INVESTIGATIONS

In order to rule out intracranial haemorrhage, infarction or brain tumour, we arranged for the patient to undergo a CT scan of the brain.

TREATMENT

Surgical indications for SDH include neurological deficit and massive mass effect with a midline shift of more than 5 mm. We arranged emergency trephination of the chronic SDH in the right parietal region, with a finding of >150 mL dark-brown blood (motor oil like). The right parietal Jackson-Pratt drainage tube was removed at postoperative day 2.

OUTCOME AND FOLLOW-UP

The patient was transferred back to the hospital after being discharged from the medical centre and underwent regular follow-up. He regained clear consciousness and can walk and eat independently.

DISCUSSION

The presentation of chronic SDH can vary from no symptoms to decreased memory, confusion, weakness or numbness of the arms, legs and face and difficulty in speech and walking. Movement disorders such as Parkinsonism can also be associated with SDH. Elderly people with subacute progressive Parkinsonism should therefore undergo CT studies to differentiate it from primary Parkinsonism.

The aetiology of chronic SDH includes head injury, subdural effusion, cerebrospinal fluid leakage and spontaneous intracranial hypotension, especially in young-aged patients to middle-aged patients, even without preceding trauma or haematological disorders.

Surgery is the best treatment option for chronic SDH involving moderate-to-large haematoma with accompanying neurological deficits. The ideal treatment strategy should be tailored to the individual patient's condition. Careful supportive treatment can be considered if it is well tolerated.⁴

In the present case, SDH was not diagnosed at our initial impression due to the lack of neurological deficits and trauma history. A patient with SDH may also initially present with withdrawn behaviour, the absence of speech, apathy, blunted affect and poor self-care, which may be thought to be manifestations of psychosis.⁵ Moreover, in many cases of chronic SDH, reversible Parkinson-like symptoms are the acute presenting feature.⁶

These symptoms may be misleading in patients with underlying psychiatric disorders who are taking antipsychotic medication.

Because such movement disturbances are a common concern in patients who use antipsychotics, as psychiatrists, we initially considered the disturbance in the present case to be olanzapine-related EPS. We did not consider the possibility of a focal neurological lesion until we consulted the neurologist. The neurological examination showed decreased muscle power of the left limbs, a positive left Babinski sign and hyperactive knee jerk, triceps reflex, and brachioradialis reflex on the left side, which implied reflex asymmetry, alerting us to the possibility of a brain lesion.

This case highlights that medications such as antipsychotics are not the only cause of EPS. An organic brain injury, such as chronic SDH, may mimic these symptoms. Because chronic SDH can be lethal, its timely detection and treatment is crucial.

Learning points

- In patients receiving antipsychotic treatment, medication side effects are often overemphasised when symptoms related to gait, weakness or alertness are observed.
- Such symptoms may instead be caused by a new medical condition such as an organic brain injury.
- With a progressing brain injury, timely and correct identification of the cause of the symptoms is crucial.
- The presence of a focal brain lesion may initially present as a feature mimicking extrapyramidal symptoms.

Contributors This case was cared by Po Wen Cheng and Rue An Tsai under the supervision of Wen-Ching Chen. Surgical intervention was done by Chieh-Hui Lee. The report was written and discussed by all co-authors.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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