CASE REPORT

Akathisia: a life-threatening side effect of a common medication

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SUMMARY

The authors describe the case of a 38-year-old man with a history of schizoaffective disorder, who attempted suicide following the recent starting of a neuroleptic agent that resulted in the development of intolerable akathisia. He survived the attempt, and following changes in his medications the akathisia resolved with no further suicidal ideation.

BACKGROUND

Akathisia is an under-recognised side effect of antipsychotic neuroleptic agents that can lead to fatal outcomes when missed. The amount of distress that it can cause is often overlooked and clinicians should be active in eliciting this symptom especially when adjusting medication regimes. This case highlights a potential life-threatening complication of a neuroleptic agent.

CASE PRESENTATION

A 38-year-old man with schizoaffective disorder was brought to an inner-city hospital after a self-inflicted gunshot wound into his oral cavity.

This man was usually well supported in the community by his family, a counsellor and community psychiatrist. He achieved average grades at school and had worked in several semiskilled and unskilled jobs after graduating from high school. He had never been in a relationship nor convicted of any crimes. His medical history was significant for a mild traumatic head injury from a motor vehicle accident at the age of 24 with no lasting complications and previous amphetamine use. Last amphetamine use was more than 6 months prior to presentation. He was diagnosed at the age of 25 with BAD, which was typically episodic with full inter-episodic recovery. At the age of 30, he was diagnosed with schizoaffective disorder with the development of negative and psychotic symptoms, most prominently paranoid delusions. He has not had any history of suicidal thoughts or behaviour.

His suicidal ideation began a few days prior to the attempt. On the day of the presentation, he left a note, went to a shooting gallery, hired a gun and shot himself in the mouth. The shot was not fatal and the gun jammed on his second attempt. He was then brought to the emergency department (ED) by ambulance.

He revealed that he had been frustrated with the new onset of an unpleasant sensation of restlessness after being put on depot pipothiazine. He describes low mood secondary to this, otherwise denied further symptoms of depression. He denied any persecutory and referential ideation or command hallucinations.

He stated that he attempted suicide to rid himself of the restlessness. Previously on chlorpromazine, olanzapine and sodium valproate, he was initiated on pipothiazine depot following a recent manic relapse with psychotic symptoms secondary to non-adherence. He had brought up his concern of the side effects of his medication to his psychiatrist but owing to his lack of insight and complex compliance issues, he was continued on the depot.

On presentation to ED, his Glass Coma Scale was 15. The primary survey did not show compromises in airway, breathing and circulation. The secondary survey demonstrated haemotympanum in the left ear and a House-Brackmann grade V palsy of the left facial nerve. There was no facial bone instability. The oral examination showed an intact postpharyngeal wall with visible entry point in the left retromolar trigone. The left parotid region was tender and full to palpate.

Mental state examination revealed a young man with visible psychomotor agitation. His mood was frustrated and his affect was restricted and angry, but stable. Thought process was grossly linear and thought content involved suicidal ideation and frustration at medication side effect. There were no perceptual disturbances. Insight and judgement were poor.

INVESTIGATIONS

Radiological investigations included CT head, face, temporal bones, cervical spine and CT angiogram of the head and neck. Debris and air locules were noted along the trajectory tract, extending from the left oropharynx to the left mastoid tip region, parapharyngeal space, masticator space and the parotid space. Shrapnel were seen within the mastoid portion of the left facial nerve with one shard <1 mm to the suboccipital portion of the left internal carotid artery. The anterior and posterior walls of the left external auditory canal were fractured with possible involvement of the tympanic membrane. Blood tests, including full blood picture, electrolytes, iron studies and thyroid function test, were unremarkable.

DIFFERENTIAL DIAGNOSIS

The presentation of akathisia and self-harm attracts several differential diagnoses.

Common causes of akathisia include diabetes, renal disease, Parkinson's disease and peripheral neuropathy, none of which this patient suffered from. Iron deficiency and hyperthyroidism were

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also ruled out on blood panels. Given the history of previous head trauma, neurological conditions would also have to be considered and a likely predisposition to akathisia should be noted. However, this patient did not have any other neurological signs and this was the first onset of akathisia.

With a previous diagnosis of schizoaffective disorder, this patient's self-harm presentation pointed towards a possible depressive relapse of schizoaffective disorder or an anxiety disorder. However, he did not meet the criteria for depression as he denied most symptoms of depression and did not present depressed. He displayed negative symptoms and the lack of psychotic symptoms may be owing to an early relapse.

Although patients with schizophrenia can sometimes experience significant anxiety, which can present as physical restlessness, this patient denied anxiety symptoms. Coupled with a lack of social stressors, stable living circumstances and good community support, an anxiety disorder was unlikely.

Taking into account the temporal sequence of events leading up to his suicide attempt and his mood and affect, his presentation was more consistent with a drug-induced akathisia and dysphoria.

TREATMENT

The patient was admitted to the hospital under the care of the head and neck surgery team. His injuries including the facial nerve palsy was treated conservatively with intravenous steroids and prophylactic antibiotics. He was started on regular risperidone and PRN lorazepam for agitation and akathisia. His usual antipsychotic medications were withheld owing to the potential of exacerbating his dysphoria, akathisia and suicidality.

OUTCOME AND FOLLOW-UP

Three months after his hospitalisation, the patient continues to be managed in the community with stable control of his schizophrenia on risperidone. He denies any suicidal ideation and no longer suffers from akathisia.

His facial nerve palsy showed little improvement over the past 3 months and is still graded as House-Brackmann IV. He is scheduled for a facial reanimation surgery that will provide him with symptomatic and cosmetic improvement of his facial nerve palsy.

DISCUSSION

Diagnosis of neuroleptic-induced akathisia

Akathisia, in Greek, means 'not sitting still', and neuroleptic-induced acute akathisia is defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM) IV as (1)a subjective feeling of restlessness with (2) outward manifestation as a result of (3) initiating or increasing dose of an antipsychotic medication.[1] These can be associated with feelings of dysphoria, irritability, violence and self-harm.[1] The symptoms usually occur within 4 weeks of this medication change.[1] It occurs in up to 75% of patients using first generation antipsychotics, but is a less frequent side effect of [s] second generation antipsychotics.[1 2] Other medications, such as serotonin-specific reuptake inhibitor antidepressant medications, calcium-channel blockers and antiemetics, can also produce an identical phenomenon.[3]

Unlike other movement disorders, no single motor feature is pathognomonic for akathisia. Coupled with the difficulty in describing the subjective feelings, akathisia is frequently underdetected, and direct questioning by the clinician is often required to elicit this symptom.[3] The difficulty in distinguishing akathisia and agitation is well-recognised and as management differs between them, it is important to make the distinction. In general, the chronological sequence of events between change in medication/dose, the feeling of being driven to action to alleviate the sense of restlessness and particular localisation of site of restlessness (eg, to lower limbs) point towards akathisia.[3]

The Barnes Akathisia Rating Scale (BARS) is the most widely used rating scale for grading the severity of drug-induced akathisia.[4] It includes both components of subjective self-awareness of restlessness with objective observable movements by the subject.[4]

Neuroleptic-induced akathisia and self-harm

Akathisia is a common and distressing side effect of neuroleptic medications which produces profound and diverse physical and emotional discomfort. Its association with self-harm is not unsurprising and has been previously reported in the literature.[5–7]

Previous studies have attempted to investigate the correlation between akathisia and suicide.[8 9] In a study population of patients with schizophrenia and schizophreniform disorder on antipsychotics, those describing subjective feelings of akathisia were more likely to be suicidal than those who did not suffer from akathisia.[8] The same study also found that subjective restlessness had greater predictability for suicidality than objective symptoms.[8]

Management of neuroleptic-induced akathisia

The treatment of neuroleptic-induced akathisia begins with its prompt diagnosis. Clinicians should have this diagnosis as a consideration whenever changes are made to neuroleptic medications.

Akathisia as a side effect of neuroleptic medication should be forewarned; when diagnosed, patients should be reassured that it is an effect of medication rather than their illness.[3 7] The use of atypical, second generation antipsychotics over conventional antipsychotics should be considered, whenever possible.[3 7] When neuroleptic-induced akathisia is diagnosed, cautious reduction of dose of the medication should be attempted.[3] Substitution with a medication less likely to cause akathisia should then be carried out.

When medication substitution or dose reduction is not immediately available, one may consider β-blockers, of which propranolol is a common choice for its lipophilic properties allowing the penetration of the blood–brain barrier.[3 7] The starting dosage is variable, but a suggested dose is 20 mg three times a day .[3 7] Besides this, benzodiazepine is also a common first or second line treatment option. If these are ineffective, or if the patient exhibits Parkinsonism symptoms, anticholinergic medications can then be tried.

Recognition and treatment of drug-induced akathisia usually leads to favourable prognosis evidenced by previous case reports and the current case. It is a modifiable risk factor of suicidal behaviour in patients with schizophrenia and should be actively managed in this group of patients.

Learning points

- Akathisia is a common side effect of neuroleptic medication which is under-recognised and has been associated with suicide.
- Screening of neuroleptic-induced akathisia should be routine.
- Neuroleptic-induced akathisia is easily treatable and generally has good outcomes when treated.

Contributors The authors declare that they have all made substantial contributions to conception, design, drafting and final approval of this paper.

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