Rehabilitation Process and Outcome



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CASE REPORT

Quetiapine Induced Acute Dystonia in a Patient with History of Severe Head Injury

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Abstract: A patient with a history of severe head injury 10 years ago regained ability to walk after years of being bound to a wheelchair. During the last psychiatric hospitalization, quetiapine was increased to therapeutic dose using a normal titration. As a result the patient developed dystonia of multiple muscle groups requiring 4 days of hospitalization for remittance of symptoms.

In this paper, we take a close look at the literature concerning extrapiramidal symptoms (EPS) in this context, and we suggest that in patients with a history of head injury, it is warranted to consider a slower titration of antipsychotic medications, including ones that are considered having a lower risk of EPS such as quetiapine.

Keywords: severe head injury, quetiapine, dystonia, extapiramidal symptoms

Rehabilitation Process and Outcome 2010:2 1-4

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Introduction

Dystonia is a common movement disorder with multiple etiologies.¹ It is characterized by involuntary and abnormal postures of the affected body part. The causes of dystonia are varied, and they can be classified as primary (idiopatic) and secondary movement disorders. The primary types are characterized by tremor and myoclonus in addition to dystonia. The secondary movement disorders could be metabolic, hereditary such as dopa-responsive dystonia, rapidonset dystonia-parkinson and myoclonus-dystonia,² traumatic or exogenous.

Drug induced dystonias are common, with certain populations at higher risk than others.³ Antipsychotic medications are often associated with extrapyramidal symptoms. Dystonia is usually an early side effect to antipsychotic medications, more common with high potency (on dopamine receptor) than with low potency antipsychotic medications. Rapid titration, being naïve to the medication, older females, and young men are few risk factors for its occurrence. Common presentation includes excessive contractions of various muscle groups. This could lead to abnormal postures which are often uncomfortable or painful.4 It could happen virtually in any striatum muscle groups. The causes of dystonia are not elucidated. Initially, dystonia was believed to happen as result of dysregulation in circuits in the basal ganglia. which are involved in initiation of muscle contraction. However, new evidence points out to areas outside of the basal ganglia, including cerebellotalamocortical circuitry and globus palidus internum.^{5,6}

Dystonia can be classified as acute and chronic. Acute dystonia usually occurs early in treatment, sometimes even after one dose of antipsychotic medication or with dose increase. Chronic dystonia is different from tardive dyskinezia which is a choreic disorder. However, both can occur after a period of time has elapsed since the initiation of antipsychotic treatment and usually are chronic.

Due to the fact that research protocols looking at response rate and side effects of antipsychotic medications do not include patients with significant co morbidities, both mental illnesses and physical conditions- the results (especially the incidence of side effects) cannot be generalized. Furthermore, having head injury alone is a risk for developing dystonia. 8–10 Also, patients with dementia such as

Lewy body dementia and frontotemporal dementia are more susceptible to extrapiramidal side effects.¹¹

Case Report

Ms. A, a 41 year old female, had a psychiatric hospitalization secondary to increased depression which eventually led to auditory hallucinations and increase in suicidal thoughts. The treatment provided in the hospital, which included individual therapy, groups, and pharmacological interventions, helped in decreasing her symptomatology.

The medications at the time of discharge are listed below. The only changes made were the discontinuation of fluoxetine 80 mg PO gam, an increase of desyrel from 300 mg PO qhs to 400 mg PO qhs and titration of quetiapine from 300 mg PO qhs to 300 mg PO bid over the first 4 days of hospitalization. While in the hospital, she started to have an increase in falls. After the hospitalization, she started to have episodes of dystonia, which due to sudden occurrence and pain associated with them caused her to fall. Ms. A reported that at times this happened in more than one extremity. Also, during the days after the psychiatric hospitalization, she had trouble swallowing. Eventually, she was taken to emergency department due to the pain which became more intense and inability to walk safely. She was hospitalized and a psychiatry consult was called due to history of depression and recent psychiatric hospitalization.

Ms. A had a long psychiatric history. She reported depression "all my life". She reported being raped by her father from ages four to 10. She has many losses throughout her life, usually triggering a major depressive episode. Though she often had suicidal ideation with a plan to harm self, only once did she attempt suicide by overdose, approximately 8 years ago when she started regaining her memory of trauma. Ms. A had a diagnosis of panic disorder without agoraphobia with current symptoms of one anxiety attack a day which was less than the five anxiety attacks a day she had prior. She also admitted to being attacked while at work 10 years ago and as result was in a wheel chair for few years due to brain injuries until she incrementally regained motor functioning. At the time of the psychiatric evaluations, she still complained of memory problems due to trauma, noted during the mental status exam. As result of that trauma, she admitted to avoidance, nightmares, sense of shortened future,



a sensation of emotional numbness, all of which were better controlled now than a few years ago.

Ms. A had also been treated for several medical conditions, including chronic pancreatitis, diabetes mellitus type 2, uncomplicated asthma, persistent controlled, hyperlipidemia and essential hypertension.

At the time of current hospitalization she was on the following medications: lamotrigine 50 mg PO qam, quatiapine 300 mg PO bid, desyrel 400 mg PO qhs prn for sleep, gabapentin 400 mg PO tid, duloxetine 40 mg PO qam, beclomethasone 80 mcg 2 puffs bid, lisinopril 10 mg PO qam, cyclobenzaprine 10 mg PO bid, esomeprazole 40 mg PO qam, warfarin 5 mg PO q5pm, metformin 1000 mg PO bid and fenofibrate 145 mg po with breakfast.

During the hospitalization, quatiapine and desyrel were stopped. Benztropine 2 mg PO bid was started as scheduled for the first days then on an as needed basis. Zolpidem 10 mg ½ to 1 tab PO qhs as needed for insomnia was also started.

As early as day two of the hospitalization, the patient reported less pain. On day 3, she was able to ambulate with assistance and on day 4 was discharged with treatment plan to follow up with psychiatry in 4 days. At the psychiatry follow up appointment, the patient continued to be stable without recurrence of dystonia or pain.

Discussion

The patient presented to the emergency department with pain in her extremities and an increase in falls. It is possible that the increased falls that Ms. A experienced could be explained as result of proprioception abnormalities that happen in the presence of dystonia and in turn led to kinetic abnormalities. Due to the time sequence of her developing acute dystonia as quetiapine was increased, we were able to establish causality. The diagnosis of dystonia was delayed by the fact that the patient was more focused on pain and falls. However, a more comprehensive history revealed the cause of pain and falls as result of dystonia.

It is important to always monitor the EPS in all patients taking antipsychotic medication. We suggest that even more attention should be employed in patients who are naïve to antipsychotic medication or in patients with other medical conditions or a complicated medical regimen. There are a few reasons

for this recommendation. One of the reasons is that the patients naïve to antipsychotics or patients that never experienced dystonia before have a difficult time describing it. When one tries to describe a new phenomenon, the normal process is either to ignore it (no relevance and is inhibited before we make a perception of it) or to make a perception of it by making mental approximations with known facts. This patient had been in pain already for many years due to her head injury and chronic pancreatitis in addition to her other medical conditions. Also, she had an extensive memory of her gait problem after the trauma. Therefore, it could be implied that her chief complaint of pain and falls could be understood from this perspective. Quetiapine has been considered, especially in certain services, to have lower EPS than other second-generation antipsychotics. This belief could have been generated by observation that is better tolerated in patients with Parkinson disease. However, in a large study, there were no significant differences, in patients with schizophrenia, between the rate of extrapiramidal symptoms between perpherazine and second-generation antipsychotics. However, patients on quetiapine seem to have a lower discontinuation rate due to parkinsonism.¹³

In view of this case report, the surgical and neurosurgical staff should exercise more caution when prescribing antipsychotic medications to brain injury patients. According to the literature cited in the manuscript, the history of head injury will predispose to dystonia. One can suggest that this information could be mentioned to the patient; however, after brain surgery there might be more pressing issues. Therefore, responsibility will lie on the treating physician with each office visit or hospital stay. History of brain injury is part of a complete history.

Antipsychotic medications are widely prescribed across disciplines, for a variety of FDA approved and unapproved indications, from psychotic conditions to sleep disturbances. In this manuscript we do not address the prescribing practices; we are merely suggesting that extra caution is warranted when we use antipsychotic medications for the patient population with a history of head injury. Nursing staff is instrumental in early identification of the extra-pyramidal symptoms. Therefore, the treating person should alert the team of possible side effects, and the measures that could be immediately taken if EPS are noted.



Conclusion

This case report suggests that in patients with a history of head injury, it is warranted to consider a slower titration of antipsychotic medications, including the ones that are considered having a lower risk of EPS such as quetiapine. Furthermore, detailed and frequent assessment of EPS is necessary.

Acknowledgements

Dr. R.G. Bota would like to thank Wendy T. Engkjer for scientific editing.

Disclosures

This manuscript has been read and approved by all authors. This paper is unique and is not under consideration by any other publication and has not been published elsewhere. The authors report no conflicts of interest

References

- Edwards MJ. Dystonia: a clinical approach. Acta Neurol Taiwan. 2008;17:219–27.
- Bressman SB. Genetics of dystonia: an overview. Parkinsonism Relat Disord. 2007;13 Suppl 3:S347–55.

- Orti-Pareja M, Jimenez-Jimenez FJ, Vazquez A, et al. Drug-induced tardive syndromes. *Parkinsonism Relat Disord*. 1999;5:59–65.
- Fahn S, Eldridge R. Definition of dystonia and classification of the dystonic states. Adv Neurol. 1976;14:1–5.
- 5. Argyelan M, Carbon M, Niethammer M, et al. Cerebellothalamocortical connectivity regulates penetrance in dystonia. *J Neurosci.* 2009;29:9740–7.
- Shanker V, Bressman SB. What's new in dystonia? Curr Neurol Neurosci Rep. 2009;9:278–84.
- Burke RE, Fahn S, Jankovic J, et al. Tardive dystonia: late-onset and persistent dystonia caused by antipsychotic drugs. *Neurology*. 1982;32:1335–46.
- Lee MS, Rinne JO, Ceballos-Baumann A, et al. Dystonia after head trauma. Neurology. 1994;44:1374

 –8.
- Loher TJ, Krauss JK. Dystonia associated with pontomesencephalic lesions. *Mov Disord*. 2009;24:157–67.
- O'Suilleabhain P, Dewey RB Jr. Movement disorders after head injury: diagnosis and management. J Head Trauma Rehabil. 2004;19:305–13.
- Czarnecki K, Kumar N, Josephs KA. Parkinsonism and tardive antecollis in frontotemporal dementia—increased sensitivity to newer antipsychotics? *Eur J Neurol*. 2008;15:199–201.
- 12. Pelosin E, Bove M, Marinelli L, et al. Cervical dystonia affects aimed movements of nondystonic segments. *Mov Disord*. 2009;24:1955–61.
- 13. Miller del D, Caroff SN, Davis SM, et al. Extrapyramidal side-effects of antipsychotics in a randomised trial. *Br J Psychiatry*. 2008;193:279–88.

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