Difficulties Associated with Diagnosing and Treating Late Onset Bipolar Disorder- A Case Study

Isioma Amayo[1], Vincent F. Capaldi[2]

[1]M.D, Resident, Department of Psychiatry, Walter Reed National Military Medical Center, Bethesda, MD

[2]M.D, Walter Reed Army Institute of Research, Bethesda, MD

Introduction

- The above 60 years of age is a growing population with estimates to grow more than 3 times the general population within the next few years[1]
- Growth amongst this population, would mean growth in prevalence of **older age bipolar disorder (OABD)[2]**
- Within the OABD population are the subset group of **late onset bipolar disorder (LOBD**). These are individuals whose first presentation of bipolar disorder occurs after the age of 50 years. [2]
- LOBD is associated with diagnostic and management complexity, due to competing brain pathology that can mimic mania and increased iatrogenic risk within these population[3]

Case Description

We present a case of a 73y/o highly functional retired Caucasian male with a past history of unipolar depression diagnosed in early adulthood (mid 20s) with characteristic symptoms of amotivation, low concentration and lethargy. His symptoms were managed periodically with fluoxetine with infrequent bupropion adjuncts. Family members started to notice hypomanic like behaviors 2 months after a dose increase of bupropion XL from 150mg to 300mg daily.

References

1. Population Division. Department of Economic and Social Affairs; United Nations: World Population Ageing: 1950-2050.
2. Sajatovic M, Strejilevich SA, Gildengers AG, et al. A report on older-age bipolar disorder from the International Society for Bipolar Disorders Task Force. Bipolar Discord 2015;17(7):689–704. doi: 10.1111/bdi.12331.
3. Arnold, I., Dehning, J., Grunze, A., & Hausmann, A. (2021). Old Age Bipolar Disorder-Epidemiology, Aetiology and Treatment. Medicina (Kaunas, Lithuania), 57(6), 587. https://doi.org/10.3390/medicina57060587

Hospitalization

Chief complaints: Bizarre behaviors and getting into arguments with the preacher

HPI: History of one prior visit to the ED for symptoms of pressured speech, impulsivity, increased project initiation, and fractured sleep. His bupropion was recently discontinued by his primary care physician. He had been prescribed nightly 25mg quetiapine while in the ED, in addition to continued treatment with his long-term daily 40mg of fluoxetine.

MSE: Patient was intrusive, belligerent, and easily distracted. Speech was pressured with use of expansive vocabulary and metaphors. Circumferential method to answering open and closed ended questions with references to past exploits. Mood reported as “great” with expansive affect. No apparent delusions, mentioned exploits were true and no report or signs of perceptual disturbance.

Neurological exam unremarkable besides mania like behavior

Work-up: Vital signs, basic chemistry, thyroid, vitamin deficiency (B1, B9, B12, Vit D) and drug screen were unremarkable. Pt was however, positive for COVID-19 PCR, and was wholly asymptomatic

Brain MRI w/o contrast: showed no acute intracranial pathology. There was cerebral atrophy with a biparietal and bifrontal predominance. No punctate brain parenchymal susceptibility abnormalities was found and only minimal supratentorial small vessel ischemic disease was noted in the deep white matter.

Treatment

- Pt was quarantined in the COVID unit, where he remained asymptomatic with normal vital signs.
- Fluoxetine was discontinued
- Valproic acid started and eventually titrated up to 500mg twice daily
- Quetiapine increased to 100mg nightly, which lead to significant hypotension, and patient was switched to olanzapine 5mg nightly.
- After 10 days of quarantine, and 3 days in the inpatient psychiatric unit pt. impulsivity, pressured speech, and insight had improved. patient was ultimately discharged on 2.5mg of olanzapine in the morning and 5mg at night. He also was on 500mg of valproic acid twice daily.

Outpatient course

- Developed painful bilateral peripheral edema 2 weeks post discharge
  - Discontinued olanzapine believed to be the cause
  - Persistent even after 20mg of furosemide and post 4 weeks of discontinuing olanzapine
  - Valproic acid reduced to 125mg three times a day and started on aripiprazole 10mg daily
- Concerns of worsening shuffling gait, and hypophonic articulation, noticed while in the inpatient psychiatry unit
  - Discontinuation of aripiprazole
  - DAT scan showed-mildly asymmetric uptake in the bilateral caudate and putamen, comma-shaped appearance of bilateral striata which is not markedly abnormal. Early parkinsonian syndrome may have a similar appearance. Pt would require one year follow-up imaging for determination.
- New symptoms of depression, 2 months post-discharge. Characterized by hypersomnia, amotivation, indecisiveness, and anhedonia.
- Further work-up were unremarkable. Including:
  - lumber puncture with fungal, bacterial, and viral infectious cultures
  - Amyloid and TAU CSF testing

Take Home Messages

- Mania, the key syndrome for bipolar disorder can be a secondary manifestation or a primary illness, and treatment is generally based on this distinction[3]. In older age patient with increased risk for all cause dementia, cerebrovascular pathology, iatrogenic mediated effects, and infection this distinction becomes difficult, which complicates diagnostic clarity and treatment course.
- This patient experienced significant side-effects from most of our intervention. There is limited evidence based guidelines on how to treat OABD, let alone LOBD. It is worth considering in hindsight, the overall risk and benefits of aggressively managing mood disorders in a 73 year old.
- Lastly, although the patient was asymptomatically positive for the relatively novel COVID-19 virus. We are still yet to definitively rule it out as an important if not primary precipitating factor for mania.