ECT or Hospice: the Utility of ECT in End-of-Life Care

Winner

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Background

ECT is a safe and effective procedure used for several neuropsychiatric disorders including major depression and is the preferred treatment in cases of acute suicidality or catatonia. Despite established safety guidelines, literature on the use of ECT in the palliative care setting is sparse and poor coordination exists between palliative care and psychiatry when treating mental illness in this population.

Objective

To determine the safety and efficacy of electroconvulsive therapy (ECT) for the treatment of severe depression in a patient being considered for hospice care due to altered mental status and functional decline.

Design/Method

This is an observational descriptive case report of a 71-year-old male who presented for evaluation and treatment of altered mental status and functional decline in the setting of MDD, HTN, HLP, T2DM, OSA, CVA history, HFrEF, pAFIB, and ESRD on HD. At baseline, the patient exhibited anhedonia, affect flattening, psychomotor slowing with concern for catatonia, and decreased PO intake. An ECT consultation was requested.

Results

A baseline MMSE obtained 1-year prior scored 28 of 30 with reported clinical findings of mild cognitive impairment. While hospitalized, the patient was too altered to complete measurement-based evaluations. An extensive medical work-up, including brain imaging, was negative. Twice weekly ECT sessions were initiated with clinical improvement noted by the third treatment. Post-ECT, the patient was more alert and engaged with an improved mood and appetite. He received 10 treatments while hospitalized, dramatically decreasing hospital length of stay, and 5 monthly treatments as an outpatient with sustained clinical improvement. After 13 treatments, the effectiveness of ECT plateaued and was discontinued after 15 treatments. The patient entered hospice care within 1-month of discontinuing ECT and died shortly afterwards.

Conclusions

In an older patient with altered mental status, major depression, and functional decline being considered for hospice care, ECT was a safe and effective treatment that provided rapid relief of mood symptoms permitting discharge home and outpatient follow-up. ECT should not be a treatment of last resort and may be preferable to pharmacotherapy in the treatment of patients in the palliative setting.

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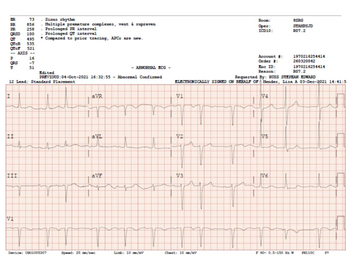
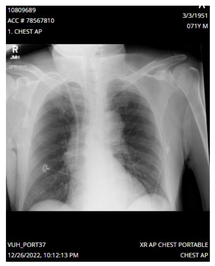
Case Presentation

Case: Pt is a 71-year-old male, married 40-years, retired computer technician, former preacher w/ master's in theology, w/ PMH significant for HLP, HTN, T2DM, HFrEF (EF 45-50%), paroxysmal AFIB, ESRD on HD, anemia of CKD, prior CVAs w/ mild residual cognitive impairment (MoCA 28/30), repaired AAA, OSA w/o CPAP, GERD, gout, BPH, and PPH significant for MDD, new-onset impulse control disorder, who presented to VUMC w/ 2-days of agitation and AMS. Did not recognize family members, odd behaviors (placing dentures in peanut butter in the fridge), and unable to follow commands. Concern for missed HD x 1. At baseline, able to perform ADLs and IADLs, adherent to medications, and engages w/ family and friends.
Home Rx: aspirin, atorvastatin, carvedilol, losartan, nifedipine, cinacalcet, Adderall, Lexapro, doxazosin, naltrexone, Lyrica, Compazine PRN, Peri-Colace PRN
Vitals: afebrile, BP 128/92, HR 132, RR 20, SpO2 95% on RA
Labs: BMP: K 2.8, HCO3 19, SCr 7.01 (on HD). Trop 0.18. BNP 378. CBC: WBC 5.0, Hgb 11.5, PLT 125. CRP 1. ESR 18. INR 1.3. Lactic acid 6.0 -> 2.8. UDS +BZDs (iatrogenic). UA >300 protein, large blood, Neg LE, Neg nitrites. VBG WNL.
Imaging: CXR w/o acute cardiopulmonary findings. CT head w/o acute IC findings but chronic small vessel disease. Unable to complete CTA due to agitation. MRI w/ chronic degenerative changes, moderate vol loss, and b R cerebellar infarct (old).
EKG: No acute ST-T changes.
EEG: Mild generalized non-specific cerebral dysfunction; irregular theta > delta activity and slow poorly sustained PDR.
Cultures: BCx NGTD. S/p Cefepime 2g, Vancomycin 20 mg/kg x1, LR 250 mL, Versed 1 mg x2, KCl 10 mEq

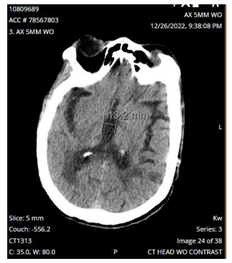
Figure 1. EKG and Imaging Obtained During Inpatient Work-up



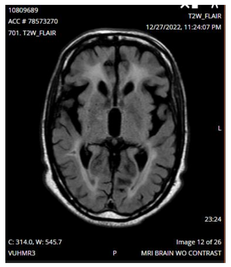
EKG 12-lead



X-ray AP chest



CT head non-contrast



MRI brain non-contrast

ECT Consultation: After 2-weeks in the hospital, the pt began to refuse routine medical care and PO intake. HD and antihypertensives were stopped and psychiatry was consulted. Severe MDD w/ concern for catatonia was diagnosed w/ initial recommendation to resume home stimulant. Palliative care was also consulted and hospice care was chosen by patient's family should his mental status not improve in response to treatment. ECT was consulted and twice weekly treatment begun as a last resort after 1-mo hospitalization. Patient's catatonia and severe depression significantly improved throughout the course of receiving ECT as an inpatient, permitting D/c home after 8-weeks.

ECT Details

Type: Low 0.5, Bilateral
Energy (%): 100
Charge (mC): 503.5
Current (A): 0.9
Stimulus Duration (s): 8
Frequency (Hz): 70
Pulse Width (ms): 0.5
Static Impedance (Ohm): 1280
Dynamic Impedance (Ohm): 200

Motoric Seizure Duration (s): 8
EEG Seizure Duration (s): 27

Thymatron® System IV

Contact: julian.raffoul@vumc.org

When Sleep Apnea Looks Like Depression: *A Case Study*

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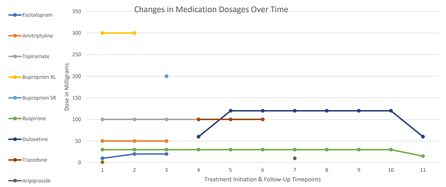
INTRODUCTION

Obstructive sleep apnea is the most common form of sleep-disordered breathing (prevalence = 22% males; 17% females)[4] characterized by repetitive episodes of airflow cessation or reduction, due to upper airway collapse. Symptoms of mood disorders can overlap with symptoms of obstructive sleep apnea (OSA), and may include fatigue and lethargy, daytime sleepiness, poor concentration, poor cognitive and executive function, inattentiveness, and reduced psychomotor speed. For example, in a study of newly diagnosed OSA patients, depression (35%) was comorbid [1] and, in an international study, patients with breathing disorders, including OSA, often reported major depressive disorder (MDD; 17.6%).[2] Lifestyle modifications (e.g., nutrition, smoking cessation, sleep hygiene) and the use of C-PAP or bi-PAP devices, can improve mood and quality of life, reducing psychiatric follow-ups, medication burden, and hospitalizations.

CASE PRESENTATION

A 38-year-old female patient presented at an outpatient clinic with a longstanding history of treatment-resistant symptoms including depression, anxiety, mood fluctuations, irritability, fatigue, inattentiveness and poor concentration. She stated that she felt “moody” and that depression followed her “like a shadow” for many years. She suffered from fibromyalgia and wondered if she may also have ADHD due to her poor focus and mental fatigue. She scored 16/21 on the GAD-7 anxiety scale, 100/144 on the Leibowitz social anxiety scale and 55 on the social phobia scale. She endorsed a long history of sleep difficulties including sinus congestion and drainage, morning headaches, and poor, broken, unrestful sleep; yet, she had never been referred or assessed for sleep apnea. Further, she had a history of failed or poor response to several medications targeting these symptoms including paroxetine, sertraline, Trazodone, amitriptyline, escitalopram, topiramate, aripiprazole, buproprion, and duloxetine. She was self-medicating her anxiety and inducing sleep with delta-8 gummies and honey. In addition to medication adjustments, a sleep study referral was placed, which indicated OSA and facilitated treatment with a CPAP device. At subsequent follow-up appointments, patient noted drastically improved mood, energy, and sleep and that she had tapered her medications (i.e., duloxetine & buspirone) with her primary care’s supervision. Her medication management appointments became less frequent. Her performance at work and overall quality of life improved.



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ETSU

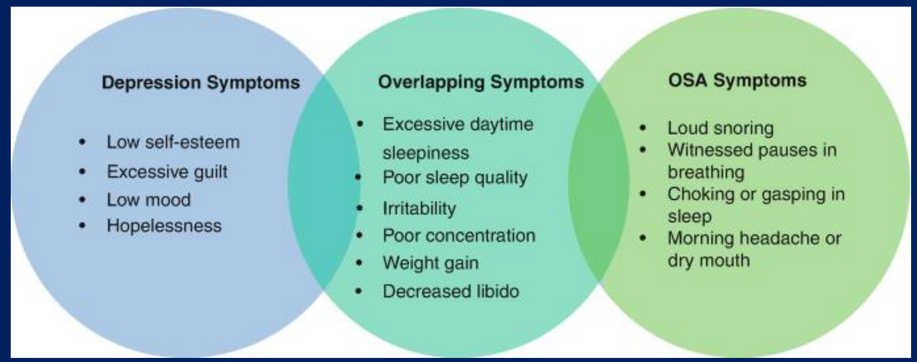
BACKGROUND

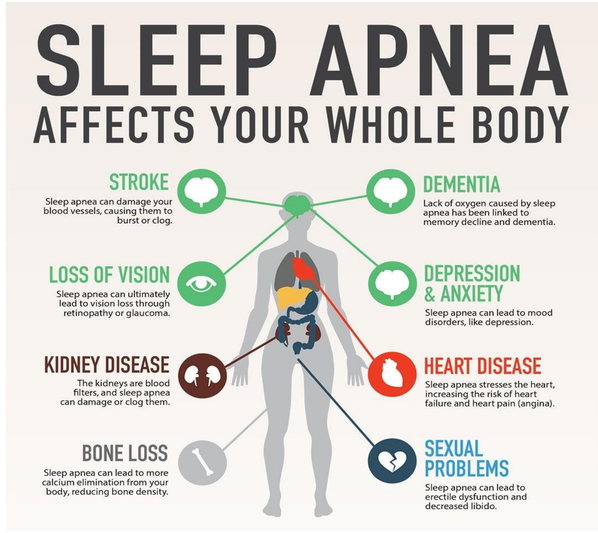
Symptoms of sleep apnea and depression overlap considerably, and the two disorders are often comorbid. It is important that clinicians screen for sleep apnea as part of their routine assessment of mood disorders. Detecting and treating sleep apnea can lead to improvement in mood, executive functioning, and quality of life, and can reduce patient’s health care burden, including risk of stroke and myocardial infarction.

Obstructive Sleep Apnea (OSA): The most-common breathing-related sleep disorder; Characterized by multiple episodes of cessation of breathing, lasting at least 10 seconds with desaturations and arousals; Involves episodes of upper (pharyngeal) airway obstruction (apneas and hypopneas) during sleep. *Apnea* is the complete obstruction of airflow, and *hypopnea* is a reduction in airflow.

Apnea-Hypopnea Index (AHI)

AHI (events/hour)	**Severity**
0-5	Normal
5 - 15	Mild sleep apnea
15 - 30	Moderate sleep apnea
>30	Severe sleep apnea





RESULTS

In previous research and clinical work, symptoms of obstructive sleep apnea overlap with those of mood disorders, including cognitive dysfunction, irritability, weight gain, and lethargy and daytime sleepiness. These diagnostic linkages may be exacerbated by other physiological conditions, such as fibromyalgia, and are bidirectional, such that sleep disturbances deleteriously impact psychiatric functioning and, reciprocally, psychiatric distress negatively influences sleep health.

Therefore, approaches to psychiatric treatment that address OSA and psychopathology simultaneously would be most beneficial. In previous research, CPAP-based treatments have shown great promise in reducing OSA and comorbid psychopathology and remain the gold standard in management. For example, in a two-month study of CPAP therapy, patients with treatment-resistant depression and comorbid OSA reported reduced depressive symptoms, as assessed by the Beck Depression Inventory and Hamilton Depression Rating Scale. [2] Similarly, across 19 randomized control trials, CPAP therapy resulted in improvements in depression, compared to control groups.[1] Although mechanisms underlying the association between OSA and psychopathology are not fully known, it is postulated that poor sleep quality and intermittent hypoxia can deleteriously influence mood, whereas CPAP therapy and oxygen supplementation may decrease these symptoms.

CONCLUSIONS

Given the extant literature, and improvement of the patient described in this case study, it is recommended that clinicians assess for sleep disturbances, including sleep apnea, as a routine component of psychiatric care. As needed, referring patients for polysomnographic assessments and sleep hygiene interventions is a gold standard approach to managing comorbid mood and sleep disorders and should be considered early in the treatment course.

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Diagnostic Challenges in a Case of Acute Catatonia and the Use of a Comprehensive Approach with Neuromodulation Interventions

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Introduction

This case study presents a 33-year-old AA male with a manifestation of acute catatonia, highlighting the critical need for a comprehensive medical evaluation in AMS, avoiding premature attributions to a psychiatric etiology.

Case Presentation

Mr. T, was admitted from jail with **AMS, rhabdomyolysis, acute kidney injury,** and **elevated liver enzymes.** Pt. had **no history of psychiatric illness or substance use** prior to a **1-month decompensation** involving paranoid delusions and disorganized behavior.

Vitals:
- T: 37.9 °C HR: 120 RR: 16 BP: 137/87

Physical Exam:
- No lead pipe rigidity

Initial management in the ED involved sedation:
- IV Fluids & CIWA protocol
- IV Haloperidol 5 mg
- IV Droperidol 2.5 mg
- IV Diphenhydramine 50 mg
- IV Lorazepam 2 mg

Laboratory results included:
- WBC = 25.3K
- CPK = 4021
- Creatinine = 2.1
- Head CT w/out contrast was unremarkable

Initial workup yielded negative results:
- Toxicology screens (UDS, Salicylate, Acetaminophen, EtOH)
- Ammonia levels
- HIV/RPR testing

Patient scored **14 at initial Bush-Francis Catatonia Rating Scale** assessment. Psychiatry, Neurology and Rheumatology were consulted.

Past Psychiatric History:
- Unspecified psychotic disorder, diagnosed in the community two weeks prior to admission.
- He was decompensating in mental status for 1 month involving increasing levels of paranoia leading to delusions that individuals were attempting to harm his daughter.
- This led him to assault his significant other and his daughter leading to his incarceration.
- No history of psychotropic medications as an outpatient.
- Patient did not have scheduled psychotropic medications nor PRN medications while incarcerated.

Social History:
- No history of substance use

Differential considerations included:
- Neuroleptic malignant syndrome (NMS),
- Brief psychotic disorder with catatonia
- Autoimmune encephalitis
- Meningoencephalitis
- Seizure (convulsive) NOS

Hospital Course

Initial Interview:
Patient was looking straight ahead, not agitated, but rather very still. He appeared to be able to track with his eyes, attending to my presence upon entry. He would not speak or follow commands. His mouth was held slightly open, and his tongue was observed to be making small movements. No apparent limb rigidity was appreciated. He has not eaten or had anything to drink since admission.

Hospital Days (HD) 0-5:
- His white count and CPK trended back to normal
- Though his mental status remained altered.
- He was often somnolent, unresponsive, and nonverbal, though he occasionally required 0.5 mg Ativan for agitation.
- He remained intermittently tachycardic, with heart rate sometimes escalating to the 160s.

On HD 5:
- Brain MRI w/wo contrast was unremarkable.

On HD 6 :
- EEG demonstrated poorly reactive, diffuse, rhythmic delta slowing implying a moderate to severe encephalopathy of nonspecific etiology.

Neurology hesitated to further explore organic causes via a lumbar puncture (LP), influenced by the patient's "psychotic" label and disorganized behavior. Neurology attributed the patient's presentation to NMS and signed off the case on HD 7.

IR performed an **LP on HD 9**:
CSF Results listed below **were all negative**:
- Cell count
- Gram stain
- Fungal culture

Further Work up yielded non-diagnostic results:
- ANA reflex
- Lupus reflex
- Lupus anticoagulant
- Hexagonal phase phospholipid
- B12 level
- HSV PCR
- Encephalitis Ab
- NMO Ab
- Anti-MOG
- ACE

Anti-NMDA and **paraneoplastic panels** were not sent as Neuro and Rheum deemed the tests of low utility given the patient being **afebrile, normal CSF**, and had **absent evidence** consistent with **anti-NMDA on MRI**.

Psychopharmacology

Treatment approaches, provided limited relief:
- Lorazepam (max: 30 mg/day)
- Memantine (max: 20 mg/day)
- Valproic acid (max: 3 g/day)
  - Avg level: 63.8 mcg/mL
- Antipsychotic trials with Aripiprazole 20 mg daily, Olanzapine 20 mg daily, Risperidone 4 mg BID
  - Medications were trialed individually and cross tapered for new medication trials.

Neuromodulation

Limitations in Treatment:
Electroconvulsive therapy was unavailable locally and legal charges impeded transfer, restricting treatment options.

Electroconvulsive Therapy in Catatonia:
- Electroconvulsive Therapy (ECT) is the definitive treatment for cases where catatonia **persists** for more than **2 to 3 days** or if there are **malignant** features.
- ECT works synergistically with benzodiazepines, typical **response rates reach 80%.**
- **Bitemporal (PT) ECT** is recommended**, 3 times per week** for at least a total of 6 sessions.[2]

Mechanism of Action:
- ECT is thought to treat catatonia by **increasing cerebral blood flow** to the **orbitofrontal** and **parietal cortex**, increasing GABA activity and GABA receptor expression.
- The **increased** release of **dopamine** and modulation of **dopamine receptors** is another proposed mechanism.[1]

Discussion & Conclusion

This case underscores **the diagnostic complexity of acute catatonia** and the importance of transcending assumptions based on psychiatric history. Comprehensive medical assessments are vital in elucidating the underlying causes of catatonic states and in ruling out treatable causes, even in patients with psychiatric comorbidities. As with this patient only with thorough diagnostic work up could the team more confidently attribute his symptoms to a psychiatric etiology. This poster presentation address why clinicians should maintain vigilance and intellectual rigor in the pursuit of accurate diagnosis in order to select effective therapeutic interventions and enable optimal patient outcomes.

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A Case Study of Variegated Syndromic Sets: Catatonia and Medication-Induced Parkinsonism Confounding Diagnostics

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Objective

- The minutiae of psychomotor characteristics in mental disorders is an area less overtly "psychiatric." However, as revealed via the following case study, grasping these particulars can optimize diagnostic clarity when Catatonia or Medication-Induced Parkinsonism (MIP) develop.

Background

- Catatonia and MIP overlap in etiology and symptoms, and neuroimaging implies possible shared aspects of pathophysiology.
- Catatonia is diagnosed by at least 3 of 12 psychomotor disturbances, the most common of which are mutism, posturing, stupor, and staring – the last of which is not included in DSM-5-TR [1].
- Etiology of catatonia is broad and includes but is not limited to schizophrenia and mood disorders, autoimmune and inflammatory disorders, and medications (e.g., paliperidone & valproic acid).
- The clinical course of catatonia is complex. Symptoms typically present acutely, but can be insidious, transient, or chronic. Approximately 20% of patients do not respond to widely used first line treatment, IV lorazepam [2]. Other treatments include ECT and NMDA receptor antagonists, which are typically effective, but not all patients respond.
- MIP is characterized by the development of Parkinsonism in temporal association with medication impinging on dopaminergic neurotransmission.
- MIP does not have standardized diagnostic criteria. Cardinal features, by commonness of presentation, include bradykinesia, rigidity, tremor (rest/action), and postural instability, each of which has wide symptom expression [3].
- Clinical course varies; offending agent may produce symptoms in days, weeks, or months.
- Removal of receptor blockading agent is preferred but depends on clinical picture [4]. Additionally, antiparkinsonian or anticholinergic medications can be considered when dopamine receptor blockade cannot be removed [5].

Most Common Symptoms of Catatonia and Associated Features of MIP	
Catatonia	MIP
Mutism – limited to no speech	Bradykinesia – global slowing hampering expression
Staring – vacant gaze with reduced blink rate	Bradykinesia – masked facies, reduced blink rate
Stupor – slow to no movement, little to no responsiveness	Bradykinesia – decreased and slowed motor activity
Posturing / rigidity – maintenance of a position against gravity or efforts to be moved	Rigidity / Bradykinesia – "lead-pipe" tone and "freezing" may appear similar to mild posturing and rigidity

Design/Methods

- 56 yo WM with past psychiatric hx of bipolar 1 disorder MRE manic with catatonia presents with 1 week of altered mental status, "stiffness," imbalance and urinary incontinence. Psychotropic regimen includes valproic acid, paliperidone, and maintenance ECT. Initial Bush-Francis (BF) score of 11.
- MRI noted mildly enlarged ventricles, with out crowding at vertex or acute callosal angle.
- Initial lorazepam challenge resulted in mild improvement in BF score. Treatment was held due to sedation (score never below 4), with persistent mutism, staring, rigidity, and stupor.
- ECT was increased to 2x week, memantine was added and paliperidone monthly injection was held. Psychomotor symptoms began improving weeks after removal.

Results

- Etiology, symptoms, and interventions employed over the clinical course all served to confound diagnosis.
- Presenting medication regimen could contribute to catatonia or MIP, while ECT could treat catatonia. Poor response to benzodiazepines would not rule out catatonia and lack of response to ECT and memantine may occur, but probability of compounding events would raise suspicion of alternate or concurrent diagnoses.
- Clinical presentation of antipsychotic induced catatonia commonly features parkinsonism [6]. In a study of dopamine signaling in the context of catatonia, via dopamine transporter single photon emission computed tomography (DAT-SPECT) of the striatum, DAT dysfunction improvement correlated with symptomatic resolution [7].

Conclusions

- Associations between catatonia and MIP exceed overlapping symptomatology, complicating diagnosis and treatment efficiency.
- Further research on the mechanism underlying common characteristics is warranted, though dopaminergic neurotransmission is likely involved and already a call has been made for "Reconceptualizing Catatonia as Psychiatric Parkinsonism." [8]

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