Case: 21 yo AA male CC — Bizarre behavior & thoughts

TEAM 1: ADAM PENDLETON, NATALY SUMARRIVA, STEPHANIE DUDZINSKI, & MARIA LADINO

HPI

4/1/2015

- Referred OSH by school, and then to VPH from OSH
- Reported bizarre delusions, referential delusions, grandiosity, & tangential speech, flight of ideas, & being difficult to redirect
- "I am the closest person to God that exists"
- Reports anxiety, mind racing, & can't fall asleep because he's afraid he'll lose his train of thought
- Admits he hasn't been taking his meds & wants "real" ones
- Overwhelmed by tuition costs, anxious about finances
- Admits to having suicidal thoughts
- -Presents with some catatonic like symptoms

Past Psychiatric History

August 2012:

- First break psychosis @ VPH→Tx with Olanzapine 25 mg qday

2012-2015

-Not compliant with meds (S/E: excessive weight gain)

2014

Hospitalization @ OSH

2/9/2015

- -Presented to Dr. Stovall with similar symptoms as today, but less severe
- -Prescribed Remeron for anxiety & insomnia

Substance Use & Social History

Substance Use:

2013

UDS: +cannabis

4/1/15

UDS: + benzos, + cannabis

Social:

- Lives with twin brother who has also had hospitalizations for psychosis
- -Chemical engineering Student at Tennessee Tech
- -Mother (RN), 2 sisters at home (16 yo, 19 yo)

This Admission

4/1/15

- Trialed Risperidone 1 mg PO BID, increased up to 1 mg po QDay & 2 mg po QHS

4/6/15

- -Failed Risperidone
- Start Olanzapine 10 mg QHS, increased up to Failed Olanzapine, 15 mg QAM & 10 mg QHS
- Start Ativan 1 mg TID for possible catatonia, increased to 3 mg BID

4/8/15

- Involuntary admission court order

4/9/15

- TRC order obtained, allowed meds & collateral from family, school, & OSH

4/13/15

- Start Chlorpromazine 100 mg BID
- Started on Lithium 600 mg po QPM for mood stabilization

This Admission Continued

4/14/15

- Code Green called → PRT
- Given chlorpromazine 200 mg PO, Ativan 3 mg, & Lithium 300 mg PO
- Later, became diaphoretic, hypertensive & tachycardic
- Monitored vitals q1h
- HR continued to increase up to 147
- Highest BP 151/98

What else should we be checking? What are we worried about?

Psychiatric emergencies

PART I

TEAM 1: ADAM PENDLETON, NATALY SUMARRIVA, STEPHANIE DUDZINSKI, & MARIA LADINO

NMS: Neuroleptic Malignant Syndrome

- ➤ What is it?
 - >A distinctive clinical syndrome of mental status change, rigidity, fever, and dysautonomia
- Cause: Neuroleptic drugs AKA anti-psychotics AKA major tranquilizers
 - Caused by both typical and atypical antipsychotics, as well as anti-emetics
 - > Chlorpromazine, Pherphenazine, Haloperidol, Fluphenazine, Clozapine, Risperidone, Quetiapine, Olanzapine, Paliperidone...

Biggest risks:

- > recent or rapid dose escalation
- ➤ a switch from one agent to another
- > parenteral administration (IV, SQ, & IM)
- ➤ Males outnumber females twofold! All ages affected.
- > Pathogenesis unknown...hypothalamic dopamine block, nigrostriatal dopamine interference?

SS: Serotonin Syndrome

- ➤ What is it?
 - >Classically, a triad of AMS, autonomic hyperactivity, and neuromuscular abnormalities.
 - > Potentially life-threatening; associated with increased serotonergic activity in the CNS.
- ➤ Cause: Serotonergic agents
 - MANY drug classes...! Antidepressants, AEDs, pain-control agents, mood stabilizers, illicits, stimulants, herbs...
 - > SSRIs, SNRIs, bupropion, TCAs*, valproate, lithium, fentanyl, St. John's Wort, MAOIs, LSD, amphetamines...and many more.
- ➤ Risk is increased w/ admin of 2 or more serotonergic agents simultaneously.
- ➤ No gender preference; all ages affected
- > Pathogenesis:
 - In the CNS, serotonin modulates attention, behavior, and thermoregulation. In the peripheral NS, serotonin helps to regulate GI motility, vasoconstriction, and bronchoconstriction. Serotonin also promotes platelet aggregation.
 - ➢ No single serotonin receptor is solely responsible! (Post-synaptic 5-HT1A and 5-HT2A implicated.)

MH: Malignant Hyperthermia

- >What is it?
 - >A hypermetabolic crisis when an MH-susceptible individual is exposed to certain anesthetic agents
- Cause: Volatile anesthetics
 - > halothane, isoflurane, enflurane, sevoflurane, desflurane, or succinylcholine
- > Can occur minutes to hours after administration of offending agent.
- ➤ Key clinical sign is generalized muscle rigidity in the presence of neuromuscular blockade, particularly MMR (masseter muscle rigidity).
- \triangleright Males outnumber females twofold, and children < 19 y/o make up ~50% of cases.
- > Pathogenesis:
 - Excessive Ca²⁺ accumulation in people with genetic skeletal muscle receptor abnormalities.
 - Clinical manifestations are due to cellular hypermetabolism, leading to rhabdomyolysis, anaerobic metabolism, acidosis, and their sequelae.

NMS vs. SS vs. MH

Table 2. Comparison of features and management of neuroleptic malignant syndrome, serotonergic syndrome and malignant hyperthermia.

	NMS	SS	MH
Age/gender	Young males	No predisposition	Children, young adults
Triggering factor	Idiosyncratic	Dose related	Genetic
Fever	++	++	+++
Confusion	+++	+++	+
Dysautonomia	+++	+++	+++
Motor features	Tremor, rigidity	Myoclonus, stereotypies, hyperreflexia	14
Diaphoresis	++	++	+++
Elevated CK	+++	++	+++
Metabolic acidosis	+	+	++
Causative agents	Classic and newer antipsychotics, antiemetics, levodopa withdrawal	SSRIs, SNRIs, TAs, MAOIs	Volatile anesthetics, succinylcholine
Pharmacologic treatment*	Bromocriptine, amantadine, dantrolene, ECT	Cyproheptadine, Methylsergide	Dantrolene, Azumolene
Criteria	Levenson JL, 1985	Dunkley EJ, 2003	Larach MG, 1994

NMS: neuroleptic malignant syndrome; SS: serotonergic syndrome; MH: malignant hyperthermia; CK: serum creatine kinase levels; SSRIs: selective serotonin reuptake inhibitors; SNRIs: serotonin-norepinephrine inhibitors; TAs: tricyclic antidepressants; MAOIs: monoamine oxidase inhibitors; ECT: electroconvulsive therapy.

*Prior to any form of pharmacological treatment, patients should be stabilized and withdrawn from causative agent.