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Neuroleptic Malignant Syndrome: A Pathophysiological Dilemma

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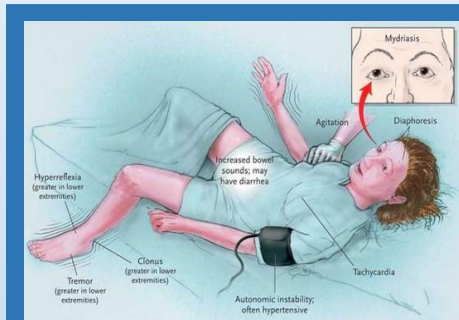
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What is NMS?

Neuroleptic malignant syndrome (NMS) is a rare disease occurring from an adverse reaction to anti-psychotic use. The diagnosis and predictability of the disease is extremely difficult as it mimics other syndromes (Margetić & Aukst-Margetić, 2010). The disease onset can occur when initiating medications, escalating doses, or adding an adjunctive anti-psychotic to the regimen. Although causing the unpredictability, the disease can occur at any dose (Paul, Michael, John, & Lenox, 2012). Further increasing the difficulty of diagnostics, signs and symptoms are very wide spread. The Diagnostic and Statistical Manual of Mental Disorders created a tool to assist in the clinical setting; it, "requires the presence of 2 core features of severe muscle rigidity and elevated temperature after recent initiation or change in dosage of an antipsychotic, along with 2 or more of the following symptoms: diaphoresis, dysphagia, tremor, incontinence, changes in level of consciousness, mutism, tachycardia, elevated or labile blood pressure, leukocytosis, and elevated creatine kinase (CK) levels" (Paul, Michael, John, & Lenox, 2012). The basis of diagnostics have been primarily through simple exclusion of other syndromes and diseases. With this being said the biggest factor in decreasing the severity of NMS is disease timely diagnosis and treatment.

Pathophysiology Dilemma

The pathophysiology of NMS is not completely understood, but there are theories and assumptions that have been found. The major dysfunction is thought to be due to sudden and profound decrease in dopamine D2 receptor blockade (Paul, Michael, John, & Lenox, 2012).



Depiction of a case with NMS

Retrieved from: http://epomedicine.com/wp-content/uploads/2013/12?Neuroleptic-malignant-syndrome_mini.jpg

Signs and Symptoms	Theory of pathophysiology
Muscle rigidity	Dopamine blockade in the nigrostriatal region
Elevated or labile blood pressures	Autonomic dysregulation of norepinephrine, serotonin, epinephrine; Dopamine is thought to play a role in interrupting the inhibitory pathways of the sympathetic nervous system (Margetić & Aukst-Margetić, 2010)
Elevated CPK	Muscle rigidity, rhabdomyolysis, agitation, physical muscle injury
Elevated temperature	Severe muscle rigidity, decreased dopamine in the hypothalamus (temperature control center of brain)

(Paul, Michael, John, & Lenox, 2012)

NMS: Devastating Effects

NMS although rare can be fatal. NMS has a spectrum of severity from minor to severe. It has been shown in evidence based practice that if NMS is diagnosed and treated early enough can drastically reduce the severity (McDermott, Noordsy, & Traum, 2013). It is important for clinicians and patients to watch for these signs and symptoms when escalating doses of antipsychotic medications or adding new or adjunctive antipsychotics. Those that live with the patients taking antipsychotics also need education to watch for these trigger signs.

Clinical Course	Effects
Altered level of consciousness	Requirements of mechanical ventilation and use of restraints (Hashim, Zeb-un-Nisa, Alrukn, & Al Madani, 2014)
Elevated CPK	Acute renal failure
Dysphagia	Risk for aspiration and long term NPO status leading to malnutrition
Diaphoresis	Skin breakdown
Labile blood pressure	Circulatory collapse and death
Muscle rigidity and bradykinesia	Risk for deep vein thrombosis, skin breakdown, rhabdomyolysis
Elevated temperatures	MRI image has shown damage to cerebellar and basal ganglia due to breakdown of membrane lipid, protein denaturation, and mitochondrial dysfunction (Lyons, & Cohen, 2013).

It is imperative for nursing to prevent secondary effects through appropriate positioning, hydration, frequent turning, close monitoring of vital signs and respiratory status, and daily skin assessments to name a few precautions.

The Dilemma In Conclusion

NMS has been a dilemma in the medical field. The pathophysiology has not been fully discovered. Diagnosis is rather difficult through mimicking of other syndromes and diseases. Types of treatments are controversial while standard medical practice has not been developed yet. The onset is unpredictable occurring at any dose. Standard practice for escalating doses of antipsychotic medication has only been made for a few such as quetiapine and clozapine (Langan, Martin, Shajahan, & Smith, 2012). After recovery re-challenging antipsychotic medication is still under debate. NMS is rare and typically self-limiting within 7-10 days if the antipsychotic medication is discontinued (Hashim, Zeb-un-Nisa, Alrukn, & Al Madani, 2014). Although there are those cases that are severe enough to cause respiratory and cardiovascular compromise. Nevertheless there is some information through evidence based practice that is certain. Those with history of NMS are to be treated with single oral antipsychotic medication with low doses and escalating as slow as possible (Ouyang & Chu, 2013). There is a much greater risk with using intravenous or intramuscular routes (Yanfen, Yahui, & Aiguo, 2014). Lastly and most important to note is that fast recognition and treatment have been proven to decrease the severity of symptoms.

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