Neuroleptic malignant syndrome (NMS) is associated with antipsychotic medications and characterized by mental status changes, muscular rigidity, hyperpyrexia, and dysautonomia. Malignant catatonia (MC) is most often associated with schizophrenia and commonly presents with catalepsy, stupor, mutism, waxy flexibility, negativism, posturing, autonomic dysfunction, rigidity, fever, and muscle injury. The pathophysiology of NMS has been attributed to a reduction in dopaminergic activity, either due to withdrawal of dopaminergic medication or reduction in dose; or to dopamine receptor antagonism by antipsychotics. NMS symptoms typically develop over the course of one to three days. MC and NMS share many features including elevated creatinine phosphokinase (CPK), leukocytosis, altered mental status (AMS), tremors, fever, and autonomic instability; however, lead-pipe rigidity and recent antipsychotic use favor NMS. In one review of 292 cases of suspected NMS versus MC the two diagnosis were indistinguishable in 20% of cases. CPK elevation greater than 1000 international units/L (IU/L) is widely accepted as a cutoff for NMS to be considered, however in one study of thirty psychiatric inpatients with NMS 40% had CPK levels below 1000 IU/L. A retrospective analysis found that the mortality rate in NMS is 5-20% despite early diagnosis and treatment, this rises to 70% if complications are also present. MC has a mortality rate of 9-10% irrespective of effective treatment and earlier diagnosis in one retrospective study.

OBJECTIVES
Introduce a case of neuroleptic associated myoclonus without elevation in the CPK level.

Compare NMS with MC, as the presentations are very similar, and both are easily overlooked.

INTRODUCTION
Neuroleptic malignant syndrome (NMS) is associated with antipsychotic medications and characterized by mental status changes, muscular rigidity, hyperpyrexia, and dysautonomia. Malignant catatonia (MC) is most often associated with schizophrenia and commonly presents with catalepsy, stupor, mutism, waxy flexibility, negativism, posturing, autonomic dysfunction, rigidity, fever, and muscle injury. The pathophysiology of NMS has been attributed to a reduction in dopaminergic activity, either due to withdrawal of dopaminergic medication or reduction in dose; or to dopamine receptor antagonism by antipsychotics. NMS symptoms typically develop over the course of one to three days. MC and NMS share many features including elevated creatinine phosphokinase (CPK), leukocytosis, altered mental status (AMS), tremors, fever, and autonomic instability; however, lead-pipe rigidity and recent antipsychotic use favor NMS. In one review of 292 cases of suspected NMS versus MC the two diagnosis were indistinguishable in 20% of cases. CPK elevation greater than 1000 international units/L (IU/L) is widely accepted as a cutoff for NMS to be considered, however in one study of thirty psychiatric inpatients with NMS 40% had CPK levels below 1000 IU/L. A retrospective analysis found that the mortality rate in NMS is 5-20% despite early diagnosis and treatment, this rises to 70% if complications are also present. MC has a mortality rate of 9-10% irrespective of effective treatment and earlier diagnosis in one retrospective study.

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CONCLUSION
NMS and MC both require inpatient medical care and pose diagnostic challenges when complicated by co-occurring acute medical conditions. This poster highlights the importance of considering both NMS and MC despite non-elevated CPK in medically complex patients with psychiatric co-morbidities.

REFERENCES

Neuroleptic Associated Myoclonus Without Rhabdomyolysis

Maxwell Miller, DO; Jonathan Coakham, MD; Rachel Gooding, MD

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