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# Serotonin Syndrome Associated With Therapeutic Metaxalone Dosing in a Patient With Cirrhosis

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## Background

Metaxalone has recently been associated with serotonin syndrome (SS) in the setting of overdose. We report the first case of SS with therapeutic metaxalone dosing in a cirrhosis patient.

## Case Report

A 65-year-old male presented to the ED with altered mental status. He was recently prescribed metaxalone (800mg every 12 hours) for a back injury. Family reported within one hour of each ingestion the patient developed confusion, diaphoresis, facial flushing, and muscle stiffness. Symptoms would gradually improve over several hours but then worsen with each subsequent dose. Medical history included cirrhosis, depression, and thrombocytopenia. Home medications included venlafaxine, quetiapine, propranolol, and rifaximin.

Initial vital signs were BP of 154/62, HR of 78, RR of 20, temperature of 38.2 C, and oxygen saturation of 94%. The patient was confused, agitated, and profusely diaphoretic. Patient had ocular clonus, mydriasis, hyper-rigidity and sustained clonus of the lower extremities. Lab values showed platelets 114 thou/cmm, creatinine 1.68 mg/dl, lactate 3.2 mmol/L, ammonia 90 umol/L, normal aminotransferases, and a negative urine drug screen. EKG showed normal sinus rhythm. Computed tomography of the head showed no abnormality. Metaxalone level, drawn approximately 12 hours after last dose, was 11mcg/ml (peak plasma concentrations average 1.7mcg/mL three hours after 800mg dose).

The patient was intubated for airway protection using etomidate and rocuronium. Diagnosis of SS was suspected. Fentanyl was started for sedation, however, this was replaced

with midazolam. Hyperthermia resolved after intubation without antipyretics. Serotonergic symptoms gradually improved over several days. The patient had a prolonged hospital course complicated by GI bleeding and hepatorenal syndrome.

## Discussion:

Metaxalone, a 2-oxazolidinone, has been theorized to have reversible MAOI properties at elevated concentrations. Previously reported cases involve overdoses of metaxalone as a single drug or in combination with another pro-serotonergic agent. This is the first case of SS in therapeutic metaxalone dosing with a confirmed serum concentration.

Metaxalone primarily undergoes hepatic metabolism although the impact of hepatic disease on the pharmacokinetics of metaxalone has not been established. We propose that cirrhosis led to decreased elimination of metaxalone and an elevated serum concentration. Supratherapeutic metaxalone serum concentrations combined with a SNRI triggered SS.

## Conclusion

Hepatic insufficiency may lead to accumulation of serum metaxalone. At high concentrations or when combined with another pro-serotonergic agent this can lead to SS.

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