

ORAL PRESENTATIONS

[ID-O#044] Risperidone Overdose: Unmasking Torsades de Pointes Risk with Comprehensive QT Assessment

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Background: Risperidone, an atypical antipsychotic, exhibits predominant antagonism at α_1 -adrenergic receptors with minimal effects on the human ether-à-go-go related gene (hERG) potassium channels. This differentiates it from other antipsychotics known to cause significant QT prolongation.

Case Presentation: A 52-year-old woman with schizophrenia ingested a presumed overdose of risperidone (600-800 mg). She presented with chest tightness and initial vital signs of tachycardia (102 bpm), hypotension (97/55 mmHg), tachypnea (24 breaths/minute), and oxygen saturation of 98% on room air. Physical examination revealed decreased alertness, but normal pupillary size, muscle strength, and reflexes. The initial electrocardiogram (ECG) demonstrated sinus tachycardia with a QT interval of 440 ms (QTc using Rautaharju correction: 540 ms). Management included intravenous (IV) crystalloid resuscitation, activated charcoal, IV potassium replacement, and admission for continuous ECG monitoring and supportive care. Approximately 19 hours post-ingestion, she experienced a syncopal episode followed by polymorphic ventricular tachycardia with Torsades de Pointes (TdP) morphology, which spontaneously reverted to sinus rhythm. Serum potassium measured after the event was 3.5 mEq/L. Our case highlights the potential for risperidone overdose to cause severe QT prolongation, which may be further aggravated by pre-existing hypokalemia (2.5 mEq/L) as seen in this patient. All QT correction formulas (Bazett, Fridericia, Framingham, Hodges, and Rautaharju) consistently indicated a marked QT prolongation.

Conclusion: Risperidone overdose can lead to significant QT prolongation and TdP, especially in the setting of hypokalemia. Clinicians should employ comprehensive QT assessment tools such as QT nomograms to evaluate and manage the risk of TdP in these situations, thereby optimizing patient safety.