

Background

Methadone, a synthetic opioid commonly used for opioid use disorder and chronic pain management, is known for its effectiveness but also carries significant risks, particularly regarding cardiac health. Methadone can prolong the QTc interval, increasing the likelihood of life-threatening arrhythmias, such as Torsades Pointes and ventricular tachycardia (VT). Patients with pre-existing cardiac conditions, such as atrial fibrillation (AF), are at an even higher risk when treated with methadone. This case report presents a 56-year-old male patient with a history of opioid use disorder and AF on Eliquis, who developed methadone-induced QTc prolongation, resulting in wide complex VT.

Objectives/Aim of the Study

The aim of this case report was to highlight the importance of methadone induced Qtc.

Case Presentation

A 56-year-old male with a history of opioid use disorder, had been on methadone therapy for several years and was concurrently on Eliquis for AF. He presented to the emergency department with complaints of palpitations, dizziness, and near-syncope. Upon arrival, he was found to be in wide complex tachycardia, indicative of VT. An electrocardiogram (ECG) revealed a significantly prolonged QTc interval. Laboratory tests showed mild electrolyte imbalances, which were corrected, but the VT persisted. Methadone was identified as the likely cause of the QTc prolongation. In response, methadone was discontinued, and the patient was treated with intravenous magnesium sulfate and potassium repletion. Despite these measures, the VT continued, necessitating electrical cardioversion, which successfully restored normal sinus rhythm. The patient’s QTc interval normalized over time, and he was monitored in the intensive care unit (ICU). Alternative pain management strategies were implemented before his discharge.

Discussion

This case illustrates the significant risk of methadone-induced QTc prolongation in patients with underlying cardiac conditions, such as AF. Methadone's ability to block cardiac potassium channels can delay ventricular repolarization, increasing the risk of arrhythmias like VT. The presence of AF and the use of Eliquis further complicated the patient's condition, emphasizing the need for close monitoring. Clinicians should be aware of the potential for serious cardiac events in patients on methadone, particularly those with additional risk factors. Regular ECG monitoring and electrolyte management are crucial in such patients to prevent the development of life-threatening arrhythmias.

Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
620	590	580	470	465	420

Conclusion

Methadone-induced QTc prolongation is a serious risk that can lead to severe cardiac arrhythmias, especially in patients with pre-existing cardiac conditions. This case underscores the importance of vigilant cardiac monitoring and careful consideration of methadone therapy in patients with AF or other risk factors for QTc prolongation. Early recognition and prompt intervention are essential to prevent adverse outcomes. The patient in this case responded well to the discontinuation of methadone and appropriate management of his arrhythmia, highlighting the need for individualized care in managing complex cases involving methadone therapy.

References

Walker PW, Klein D, Kasza L. High dose methadone and ventricular arrhythmias: a report of three cases. *Pain*. 2003 Jun;103(3):321-324. doi: 10.1016/S0304-3959(02)00461-X. PMID: 12791438.

Alinejad S, Kazemi T, Zamani N, Hoffman RS, Mehrpour O. A systematic review of the cardiotoxicity of methadone. *EXCLI J*. 2015 May 5;14:577-600. doi: 10.17179/excli2015-553. PMID: 26869865; PMCID: PMC4747000.

Disclosures

None