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CASE REPORT

Successful transition to buprenorphine in a patient with methadone-induced torsades de pointes

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Abstract A 56-year-old-man presented with syncope and torsades de pointes secondary to methadone-induced *QT* prolongation. After transition from methadone to buprenorphine, a partial μ-opiate-receptor agonist and a κ-opiate-receptor antagonist, the *QT* normalized and ventricular arrhythmias resolved. Buprenorphine should be used for opiate dependence and chronic pain in patients with methadone-induced *QT* prolongation and as first line therapy in patients with risk factors for torsades de pointes.

Keywords Prolonged *QT* · Torsades de pointes · Methadone · Buprenorphine

1 Case report

A 56-year-old man with a remote history of opiate abuse, on methadone maintenance (methadone 100 mg once a day), presented to the Beth Israel Medical Center, with recurrent syncope. Over the previous 1 month, he had experienced three episodes of syncope. All were preceded by palpitations and chest discomfort lasting several seconds. Symptoms progressed rapidly followed by loss of consciousness.

On the day of admission, he collapsed on his way to his physician's office. He regained consciousness in the ambulance and was transferred to the hospital. Telemetry in the emergency room demonstrated frequent, short runs of torsades de pointes (TdP; Fig. 1). His electrocardiogram (ECG) revealed sinus bradycardia with a markedly prolonged *QT* interval (Fig. 2). In addition, he had frequent polymorphic ventricular premature beats on telemetry. Serum electrolytes were significant for potassium 3.5 mmol/L and magnesium of 1.7 mg/dL. Treatment in the ED included potassium and magnesium repletion and transvenous pacing, which successfully suppressed the ventricular arrhythmias.

Methadone was discontinued, and morphine was used as needed for signs and symptoms of opiate withdrawal. The *QT* interval normalized over 3 days, and transvenous pacing was stopped. On hospital day 5, buprenorphine was initiated for methadone withdrawal and chronic opiate dependence. The *QT* interval on buprenorphine remained normal, and no ventricular arrhythmias were noted (Fig. 3). At 12-month follow-up, the *QT* interval remains normal, and there have been no recurrent symptoms or ventricular arrhythmias.

2 Discussion

Methadone, a long-acting synthetic opioid, is an effective treatment for opiate dependence and chronic pain [1]. However, in addition to its opioid effects, methadone blocks Ikr and prolongs the *QT* interval in a dose-dependant fashion. TdP and sudden death have been well documented in patients on methadone [2–5]. This presents a challenge for patients maintained on long-term methadone therapy.

Buprenorphine, a partial μ-opiate-receptor agonist and a κ-opiate-receptor antagonist, is effective at reducing both the use and craving of opiates among opiate-addicted persons [1, 5–7]. Furthermore, there has been no report of

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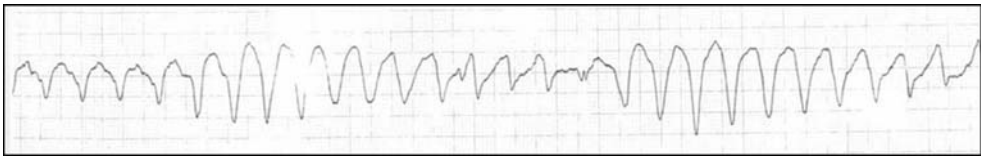

Fig. 1 Rhythm strip demonstrating torsades de pointes

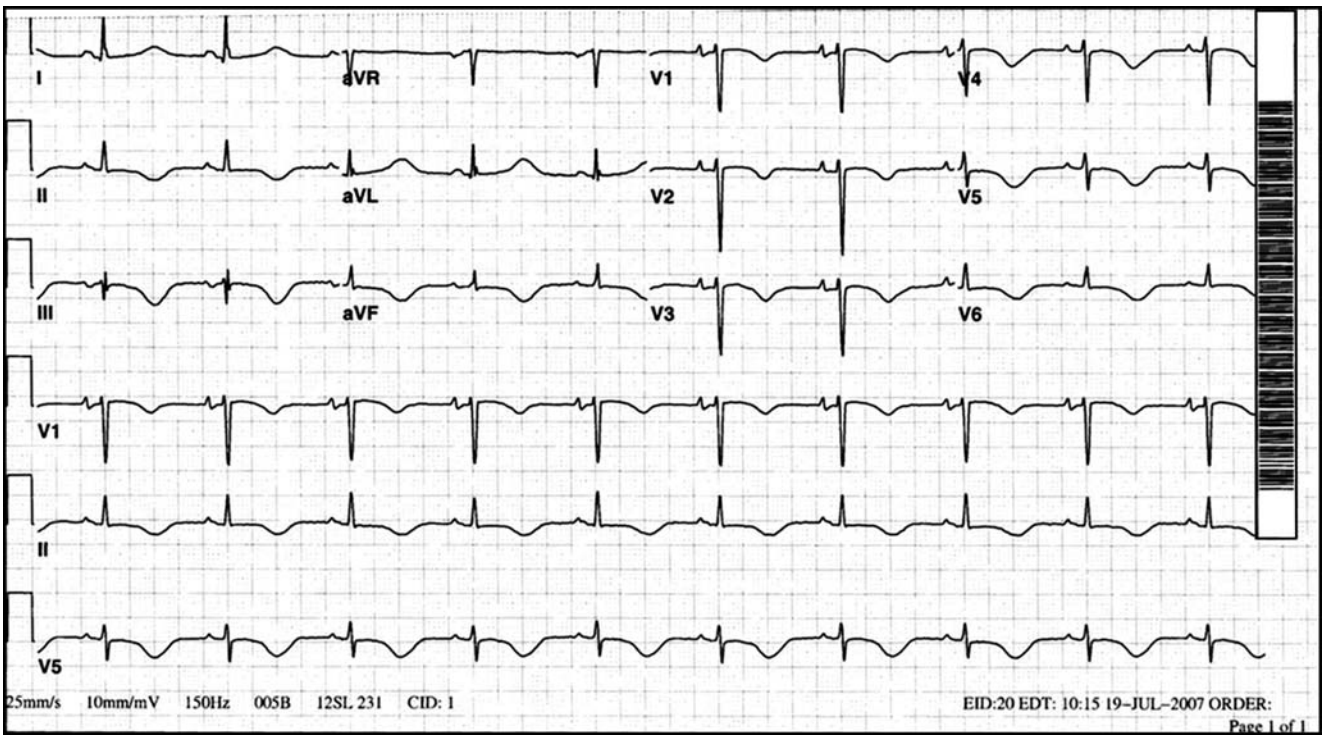

Fig. 2 Admission ECG showing a prolonged *QT* (*QT* corrected 580 ms)

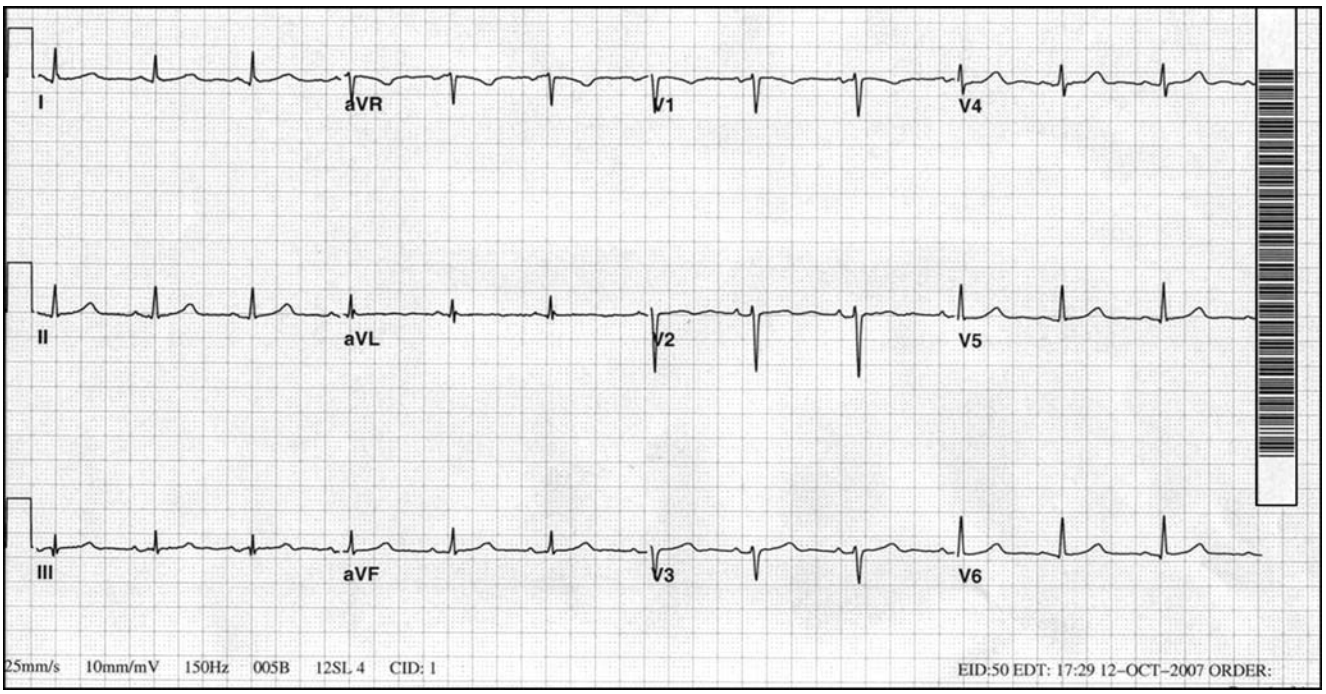

Fig. 3 Follow-up ECG off methadone revealing a normal *QT* interval

QT prolongation caused by buprenorphine [5–7]. Thus, buprenorphine is an important alternative to methadone for opiate dependence and chronic pain.

We have described a case of successful transition from methadone to buprenorphine in a patient with methadone-induced TdP. Buprenorphine is indicated in patients who have had sudden death or TdP secondary to methadone-induced long *QT*. In addition, buprenorphine should be used as a first line agent in patients who are at high risk for TdP or sudden death and require medication for opiate dependence or chronic pain.

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