Cardiac arrest after tramadol injection in a polytrauma patient

Sir,

Serious adverse reactions like cardiac arrest have never been reported with isolated tramadol injection. Patient's consent was obtained for reporting this case.

A 25-year-old male (65 kg) was admitted in an emergency department with right tibia and fibula fractures including ipsilateral 3-7th ribs fractures with flail chest and lung contusion. On examination, he was conscious, oriented, and hemodynamically stable but mildly tachypnic. His investigations showed normal hemogram, coagulation profile, and renal function. Urine for fat droplets and ophthalmoscopic examination did not suggest any fat globules. Focused assessed sonography for trauma was negative. Electrocardiography (ECG) was normal (QT_c of 320 ms). Chest X-ray suggestive of trauma mentioned above showed no visible pneumothorax. On admission to intensive care unit, his oxygen saturation dropped to 90% with PaO_2 81 at 40%. Failure to insert epidural catheter directed us to add tramadol 100 mg 8 hourly with paracetamol. Tramadol (Urgendol, Win Medicare Pvt. Ltd., Modipuram, India) was given 4 h after paracetamol by slow intravenous injection. Ten minutes after tramadol injection the patient developed ventricular tachycardia (VT) with hypotension [Figure 1]. Immediate cardiopulmonary resuscitation (CPR) was initiated. Patient reverted back into sinus rhythm transiently with direct current shock of 200 J but again went into VT. He was intubated, and mechanical ventilation was continued. CPR was continued and repeat shock after magnesium correction resulted into sinus rhythm. Return of spontaneous circulation took 6 min. Adrenaline infusion was initiated and titrated to treat hypotension at the rate of 0.1-0.5 mcg/kg/min. Electrolytes were corrected further. ECG after sinus rhythm showed prolonged QT_c interval of 480 ms [Figure 2]. Echocardiography finding was not significant. Tramadol was discontinued and magnesium sulfate 6 g/day was added. His blood tramadol level was 11 mg/L (plasma therapeutic level 0.28-0.61 mg/L). Over next 24 h adrenaline was tapered. He was extubated on 3rd day.

Tramadol is mainly metabolized to O-desmethyltramadol by the cytochrome P450 CYP2D6.[1] Two voltage-gated K^+ channels such as rapid repolarizing current (I_{Kr}) and slow repolarizing current (I_{Ks}) are responsible for rapid and slow repolarization of cardiac muscle action potential and QT interval in the ECG paper. Rapid repolarizing current is encoded by human ether-a-go-go-related gene (HERG). A 5-hydroxytryptamine 3 (5-HT_3) receptor antagonists cause cardiac arrhythmias by several mechanisms.[2] Recently, it is reported that the molecular level affinity of 5 HT_3 receptor antagonist to the HERG encoded K^+ channel causes prolongation of cardiac repolarization and QT prologation.[3,4] Tramadol inhibits 5-HT_{2C} receptor which may be responsible for K channel related prolonged repolarisation. Blood levels of tramadol and its metabolites can be measured in any serious event. Patient with CYP2D6 genotyping is at risk of tramadol-related cardiotoxicity.[1] De Decker *et al.* reported tramadol intoxication patient presented with asystole and multi-organ dysfunctions.[5] In our patient, secondary survey excluded the other possibilities of pneumothorax, cardiac tamponade, fat embolism, anaphylaxis with the help of several investigations. We presumed the culprit to be tramadol with electrolytes disorder as precipitating factor. In conclusion, caution must be kept in mind using tramadol in high-risk group like patients with electrolytes disturbance and co-administration of drugs causing prolonged QT_c interval in patients with CYP2D6 genotyping.

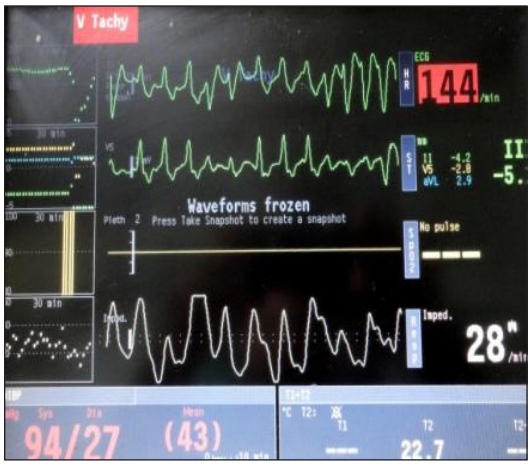

Figure 1: Monitor with electrocardiography showed ventricular tachycardia with hypotension

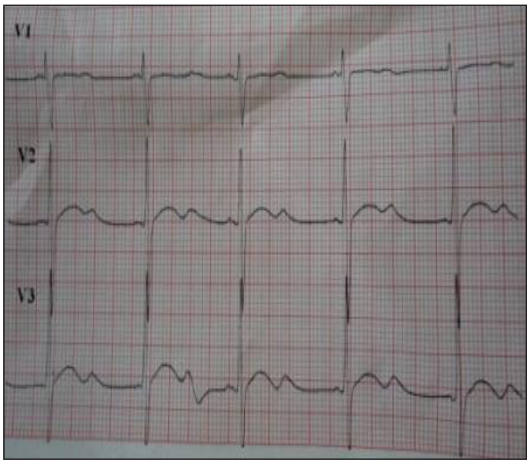

Figure 2: Electrocardiography demonstrated QT_c prolongation

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