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Case Report

Cardiac Arrest during Dexmedetomidine Infusion in a Critically Ill Patient

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Abstract

Dexmedetomidine has increasingly been used in ICU for management of pain, agitation and delirium. Use of it in critically ill patients may be associated with improved outcome. However, dexmedetomidine therapy has also been associated with adverse cardiovascular events like hypotension, bradycardia and asystole. Diabetic patients are at risk of cardiovascular morbidity including sudden cardiac death. Here we present a case of sudden cardiac arrest during dexmedetomidine infusion in a critically ill patient with long standing diabetes mellitus and possible diabetic cardiac autonomic neuropathy.

Keywords: Cardiac Arrest; Critically Ill; Dexmedetomidine; Diabetes Mellitus

Introduction

Dexmedetomidine is a selective α2 – receptor agonist with sedative, analgesic and sympatholytic properties with minimal respiratory depression [1]. Use of dexmedetomidine can reduce delirium in ICU and is associated with preserved cognitive function [2,3]. The Federal Drug Administration approved a maximum dose of 0.7 μg/kg/hour, but doses upto1.4 μg/kg/hour are frequently used in clinical practice [1,4,5]. It has been approved for short term sedation of ICU patients (less than 24 hours) at a maximal dose of 0.7 μg/kg/hour. [1]However, studies demonstrate safety and efficacy of dexmedetomidine infusion administered for greater than 24 hours and at higher doses [6,7]. Bradycardia is a common side effect and the risk is significantly higher when both a loading dose and high maintenance dose is used [8].

Diabetic patients are at risk of sudden cardiac death, in part due to cardiac autonomic neuropathy (CAN) [9]. Autonomic imbalance between sympathetic and parasympathetic nervous system regulation of cardiovascular function can contribute to significant morbidity and mortality in diabetic patients [10]. Here we present a case of cardiac arrest in a critically ill patient with longstanding diabetes mellitus during dexmedetomidine infusion. The patient had previous episodes of unexplained bradycardia and cardiac arrest.

Case Description

A 40 years old male patient with the history of long standing diabetes mellitus presented with infected ulcer in foot. He underwent debridement of ulcer under general anesthesia which was uneventful. His baseline ECG was normal and he had no history of chest pain or exertional shortness of breath. On fourth post-operative day, he had an episode of bradycardia (heart rate upto 35/minute) managed with atropine 0.6 mg. No obvious reasons for bradycardia were elicited. He was not on beta blockers or calcium channel blockers and 12 lead ECG showed no evidence of inferior wall myocardial infarction. On sixth post-operative day, he had sudden bradycardia progressing to cardiac arrest. Patient had return of spontaneous circulation (ROSC) after 5 minutes of cardio-pulmonary resuscitation (CPR). No obvious causes were identified. Patient was intubated during CPR. Therapeutic hypothermia was not commenced as the patient was responsive at the end of CPR. However, patient remained intubated due to poor oxygenation probably due to aspiration of gastric contents. Patient was successfully weaned off the ventilator after 5 days. After extubation, patient was delirious with the Richmond Agitation Sedation Scale (RASS) of +4, which decreased

to +3 after Haloperidol loading dose titrated upto 40 mg. His ECG was normal with no prolongation of QTc interval. He was haemodynamically stable with blood pressure of 140/80 mm Hg and heart rate of 94/minute. His arterial blood gas analysis revealed normal gas exchange and normal biochemical findings.

He was started on Dexmedetomidine with the loading dose of 1μg/kg over 10 minutes and then put on maintenance infusion of 0.4μg/kg/hour. After 9 hours of continuous infusion, patient developed sudden bardycardia followed by asystole. Patient had ROSC after 15 minutes of CPR. Dexmedetomidine was stopped. Patient was intubated and kept on mechanical ventilation. His haemodynamics and metabolic parameters were normal after ROSC. After 3 hours he had another episode of bradycardia and cardiac arrest. This time, despite 30 minutes of CPR, patient could not be revived. No obvious reversible causes were identified.

Discussion

Dexmedetomidine has increasingly been used in anaesthesia and intensive care. It provides a unique conscious sedation and analgesia without respiratory depression. There is increasing evidence of its organ protective effects and its beneficial effects in critically ill patients. However, it causes dose-dependent decrease in heart rate and blood pressure, primarily mediated by decrease in sympathetic tone and partly by baroreceptor reflex and enhanced vagal activity [1]. Bradycardia is significantly higher when both a loading dose and high maintenance dose (more than 0.7 μg/kg/hour) is used [8]. Both the prolonged infusion (more than 24 hours) and higher dose of dexmedetomidine (more than 0.7 μg/kg/hour) are reported to be safe [6,7]. However, there are reports of cardiac arrest during its infusion, especially in patients with significant cardiac disease, which were potentially preventable [11].

Our patient remained agitated with RASS of +3 even after administration of Haloperidol 40 mg. The patient was hemodynamically stable with no bradycardia. As the patient had no obvious contraindications for dexmedetomidine (like bradycardia, hypotension or concomitant drug use contributing to hemodynamic instability), except for the possibility of CAN (which at that time was not proven, but suspected), he was started on dexmedetomidine which has minimal respiratory depressant property [1] and bradycardia is less likely at dose of less than 0.7 μg/kg/hour [8]. So, a lower dose of 0.4μg/kg/hour was chosen. We chose to administer dexmedetomidine over intubation the patient as mechanical ventilation is associated multitude of complications [12].

The reversal Naloxone was not used as its utility is seen only in some animal studies with no larger studies proving it [13]. Atipamezole was not used as it was not available.

In patients with diabetes mellitus, CAN has been reported to be a poor prognostic factor associated with an increased incidence of sudden cardiac deaths [9]. CAN is one of the most overlooked of all serious complications of diabetes found in one fourth of type 1 and one third of type 2 diabetic patients. These patients experience greater perioperative cardiovascular instability. Intracardiac sympathetic imbalance predispose to arrhythmias and sudden unexpected deaths [14].

In our patient, unexplained recurrent episodes of bradycardia and cardiac arrest may be the manifestation of CAN, which was not certain as the formal testings were not performed due to critical illness. Even the recommended dose of dexmedetomidine precipitated sudden bradycardia followed by asystole in our patient, in whom, CAN may have been overlooked. High index of suspicion is needed for diagnosis of CAN. Testing for heart rate variability can be helpful [14].

To conclude, dexmedetomidine infusion should be avoided in critically ill patient with possible CAN.

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