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Case Report

Dexmedetomidine Withdrawal Mimicking ST-Elevation Myocardial Infarction

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Dexmedetomidine is an alpha-2 adrenoceptor agonist typically used in the intensive care unit (ICU) for sedation without respiratory compromise. Although it can be used safely for a prolonged period of time, herein we highlight a case of abrupt discontinuation of dexmedetomidine that resulted in coronary vasospasm mimicking an ST-elevation myocardial infarction. This adverse event necessitates prompt recognition and management.

Introduction

The sympatholytic effect of dexmedetomidine, an alpha-2 adrenoceptor agonist, makes it an excellent choice for managing anxiety and agitation among ICU patients, as it helps achieve sedation without respiratory compromise.[1] It can be used safely for a prolonged period of time. However, its abrupt discontinuation carries a risk of withdrawal. We describe a complex clinical case of an ischemic cardiac event coincident with dexmedetomidine withdrawal.

Case Presentation

A 38-year-old man was found unresponsive and covered in vomitus in front of a detoxification centre. Upon arrival at the hospital, he required endotracheal intubation for airway protection, with a Glasgow Coma Scale (GCS) score of 8. His heart rate was 114 beats per minute, and his blood pressure was 148/93 mm Hg. Initial laboratory and diagnostic imaging investigations were negative, including the chest radiograph, computed tomography of the head, magnetic resonance imaging of the brain, electroencephalogram, complete blood count, metabolic panel, ammonia levels, and blood cultures, ruling out major structural, ischemic, seizure-like, metabolic, and infectious etiologies for his altered mentation. Although his past medical history was significant for polysubstance use (with cocaine, methamphetamine, and marijuana), human immunodeficiency virus (HIV; nonadherent to antiretroviral therapy), and bipolar disease (not on prescription medications), multiple urine and serum toxicology screens were negative. He was admitted to the medical ICU and remained intubated for 8 days, due to agitation requiring sedation with high doses of dexmedetomidine (as high as 1.3 mcg/kg per hour) and propofol (as high as 80 mcg/kg per minute) infusions, as well as midazolam or lorazepam boluses as needed. Propofol was tapered off over the next 3 days. Upon extubation, on day 8, dexmedetomidine was the only medication he was receiving, at 1 mcg/kg per hour, which was turned off abruptly without tapering. Four hours later, he developed severe, midsternal, angina-like chest pain, and became diaphoretic, tachycardic (120-135 beats per minute), tachypneic (25-35 breaths per minute), and hypoxic (saturating 70% on room air). A 12-lead electrocardiogram (ECG) showed ST elevations in leads V1 to V3 (Fig. 1A), consistent with anterior ST-elevation myocardial infarction (STEMI). Serum troponin-I level was initially 0.7 ng/mL, and it peaked at 16.4 ng/mL, approximately 8 hours after the onset of chest pain. The patient was loaded with aspirin and ticagrelor and was initiated on a continuous infusion of heparin. Cardiac catheterization was recommended by the consultant cardiologist, but the patient refused this intervention, and medical management was continued. Serial ECGs showed improvement in ST elevations with resolution of symptoms, including diaphoresis, chest pain, and hypoxia (Fig. 1B). A 2-dimensional transthoracic echocardiogram did not show any evidence of wall motion abnormalities or apical ballooning. The patient eventually agreed to undergo cardiac catheterization the following day, which showed luminal irregularities without evidence of obstructive coronary artery disease (Fig. 1, C and D). Following this, he was managed medically with no further ECG changes or symptoms of chest pain. Ultimately, he was discharged from the hospital, with planned outpatient follow-up with primary care and cardiology.

Discussion

This case illustrates that the response to the sudden withdrawal of dexmedetomidine can be associated with

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Novel Teaching Points

- Withdrawal is more likely to be associated with discontinuation of dexmedetomidine after prolonged use.
- Although the potential for ischemia seen in the setting of dexmedetomidine withdrawal may be temporary, recognition of this phenomenon is important, to help in instituting appropriate care and preventing fatal cardiac events.

symptoms of severe coronary vasospasm and can mimic STEMI. The commonly reported dexmedetomidine withdrawal signs are tachycardia, hypertension, anxiety, altered mental status, and diaphoresis.[1] Multiple factors, such as dosage, duration, infusion rate, and patient's age, contribute to the withdrawal. Symptoms of dexmedetomidine withdrawal may be due to the sudden withdrawal of antagonistic action presynaptic alpha-2 agonism,[2] which might explain the onset of symptoms of sympathetic stimulation in our patient. Given the similar adrenergic agonist activity, we hypothesized that withdrawal symptoms would be similar to those of clonidine withdrawal.[2] Abrupt cessation of clonidine causes a sympathetic surge, due to increased catecholamine levels and alpha-adrenergic receptor sensitivity. This same mechanism likely is responsible for similar withdrawal symptoms seen with abrupt dexmedetomidine cessation.[2] A similar phenomenon is observed clinically more commonly with beta-blocker withdrawal causing sympathetic hyperactivity. Additionally, the half-life of dexmedetomidine can range from 4 minutes (following a 10-minute infusion) up to 250 minutes (following an 8-hour infusion).[3] Consequently, based on the concept of context-sensitive decrement time, the duration of action after a continuous infusion is time-dependent, which can explain the onset of symptoms in our patient. Prolonged use of dexmedetomidine for greater than 5-7 days is also more likely to be associated with withdrawal symptoms.[4,5] We observed the more commonly seen side effects of sudden sympathetic surge due to cessation of dexmedetomidine, including tachycardia, diaphoresis, and agitation. These effects may have led to increased myocardial oxygen demand and

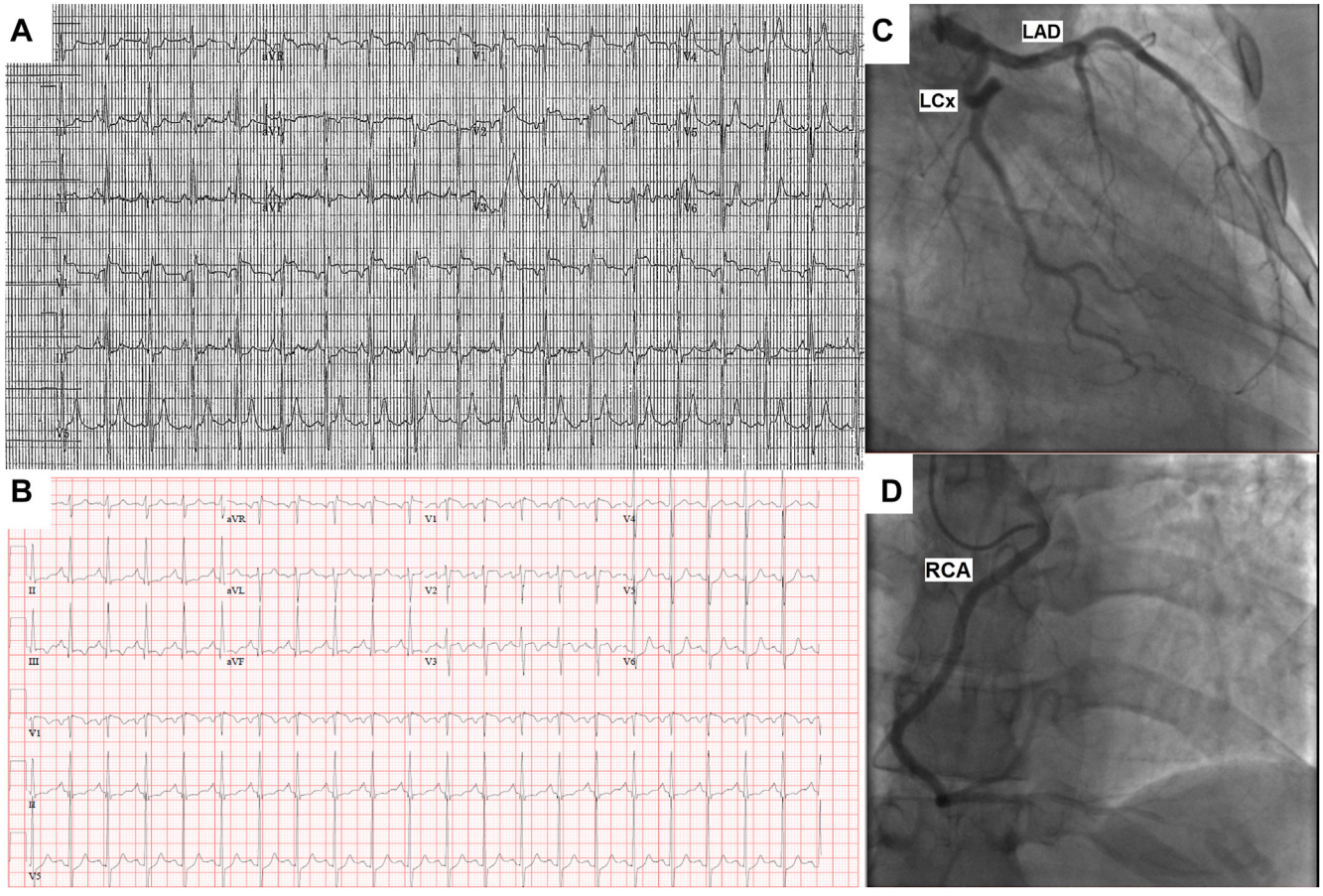

Figure 1. (**A**) Initial 12-lead electrocardiogram showing ST-segment elevations in leads V1 to V3. (**B**) Interval 12-lead electrocardiogram, 4 hours later, showing ST-segment depressions in the inferior and lateral leads, as well as ST and T wave abnormalities in the inferior and anterior leads. (**C, D**) Coronary angiography revealing luminal irregularities, but no obstructive coronary artery disease, in the left anterior descending (LAD) artery, left circumflex (LCx) artery, and right coronary artery (RCA) distributions.

severe coronary vasospasm, mimicking STEMI, given that no obstructive coronary artery disease was noted on coronary angiography. Few prior case reports have been made of similar findings in the setting of clonidine withdrawal with similar clinical presentation.[6] No standard treatment is recommended for dexmedetomidine withdrawal.[2,7] However, given the similarity of the mechanism, clonidine has been used to prevent this phenomenon. Clonidine has a longer half-life, can be given orally or as a patch, and is less expensive. This approach has been reported to help patients be weaned off dexmedetomidine in the ICU.[2,6,7] Unfortunately, no clear dosing of clonidine is recommended to prevent dexmedetomidine withdrawal. The case reports and limited studies suggest that preemptive use of total daily doses of 0.1-0.3 mg tapered down over 4-7 days in patients who received dexmedetomidine for $>$ 72 hours had favorable outcomes.[2,7]

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Ethics Statement

The research reported has adhered to the relevant ethical guidelines.

Patient Consent

Informed consent was obtained from the patient. The authors certify that all possible efforts have been made to protect the identity of the patient mentioned in this manuscript.

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Disclosures

The authors have no conflicts of interest to disclose.

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